



CIGNA MEDICAL COVERAGE POLICY

The following Coverage Policy applies to all health benefit plans administered by CIGNA Companies including plans formerly administered by Great-West Healthcare, which is now a part of CIGNA.

**Subject Implantable Cardioverter
Defibrillator (ICD)**

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Coverage Policy

CIGNA covers an implantable cardioverter defibrillator (ICD) as medically necessary for individuals who are receiving ongoing optimal medical therapy and ANY of the following criteria are met:

- cardiac arrest due to ventricular fibrillation (VF) or hemodynamically unstable sustained ventricular tachycardia (VT) after evaluation to define the cause of the event and to exclude any completely reversible causes
- structural heart disease and spontaneous sustained VT, whether hemodynamically stable or unstable.
- syncope of undetermined origin with clinically relevant, hemodynamically significant sustained VT or VF induced at electrophysiological study
- left ventricular ejection fraction (LVEF) less than 35% due to prior myocardial infarction (MI), at least 40 days post-MI, in New York Heart Association (NYHA) functional Class II or III
- nonischemic dilated cardiomyopathy (DCM), LVEF less than or equal to 35%, in NYHA functional Class II or III.
- LV dysfunction due to prior MI, at least 40 days post-MI, LVEF less than 30%, in NYHA functional Class I
- nonsustained VT due to prior MI, LVEF less than 40%, and inducible VF or sustained VT at electrophysiological study
- unexplained syncope, significant LV dysfunction, and nonischemic DCM.
- sustained VT, with normal or near-normal ventricular function
- hypertrophic cardiomyopathy (HCM) with one or more major risk factors for sudden cardiac death (SCD)

- arrhythmogenic right ventricular dysplasia/cardiomyopathy (ARVD/C), with one or more risk factors for SCD.
- long-QT syndrome, experiencing syncope and/or VT while receiving beta blockers
- non-hospitalized individuals awaiting transplantation
- Brugada syndrome with syncope
- Brugada syndrome with documented VT that has not resulted in cardiac arrest
- catecholaminergic polymorphic VT with syncope and/or documented sustained VT while receiving beta blockers
- cardiac sarcoidosis, giant cell myocarditis, or Chagas disease
- symptomatic sustained VT in a child or adult with congenital heart disease who has undergone hemodynamic and electrophysiological evaluation.
- recurrent syncope of undetermined origin in a child or adult with congenital heart disease in the presence of either ventricular dysfunction or inducible ventricular arrhythmias at electrophysiological study

CIGNA covers an ICD in a child who is receiving optimal medical therapy and has survived cardiac arrest as medically necessary when evaluation fails to identify a reversible cause.

General Background

There is a high incidence of sudden cardiac death (SCD) in patients with heart failure and diminished left ventricular ejection fraction (LVEF) and in patients who are recovering from acute myocardial infarction (MI). Although significant effort has been directed to the identification and treatment of high-risk patients, this group actually accounts for a small proportion of preventable SCD. Although the risk of SCD increases in proportion to the severity of cardiac disease in an individual patient, most events occur in patients with no known cardiac history and with few or no risk factors. There is no single test capable of accurately predicting SCD risk in various clinical settings and patient populations. Although available tests can provide valuable information, they are hampered by limited positive predictive value and are not sufficiently investigated in many categories of patients with structural heart disease (Zipes et al., 2006; Kusmirek and Gold, 2007).

Ventricular fibrillation is the rhythm most frequently recorded at the time of sudden cardiac arrest. Although a number of studies have investigated the electrophysiologic (EP) mechanisms responsible for the onset of ventricular tachycardia and ventricular fibrillation, antiarrhythmic agents have not been shown to be effective in preventing SCD. Rather, it is the drugs that have no direct EP actions on cardiac muscle or specialized conducting tissue that have been demonstrated to be effective in preventing SCD. Such drugs include beta blockers, ACE inhibitors, angiotensin receptor-blocking agents, lipid-lowering agents, spironolactone, and fibrinolytic and anti-thrombotic agents (Zipes, et al., 2006).

SCD, a direct result of cardiac arrest, may be reversible if responded to promptly. The implantable cardioverter defibrillator (ICD) is a surgically implanted device designed to constantly monitor an individual's heart rate, recognize VF or VT and deliver an electric shock to terminate these arrhythmias in order to reduce the risk of sudden death. The device is connected to leads positioned inside the heart or on its surface. These leads sense the cardiac rhythm, deliver electrical shocks, and sometimes pace the heart, as needed. The leads are tunneled to a pulse generator, which is implanted in a pouch beneath the skin of the chest or abdomen. Progressive improvements in design and miniaturization have allowed transvenous placement of ICDs to become routine. Problems associated with ICDs include inappropriate shock discharge, defibrillator storm with appropriate recurrent ICD discharge for recurrent ventricular tachyarrhythmias, inappropriate discharge for multiple reasons, infections related to implantation, and exacerbation of heart failure when a high percentage of the heartbeats are paced from the right ventricle apex and ventricular function is already compromised.

Two categories of trials have investigated the use of ICDs for prevention of SCD. ICDs have been evaluated for primary (i.e., prophylactic) prevention of SCD in patients who have not experienced a life-threatening ventricular arrhythmia (or a symptomatic equivalent). Secondary prevention trials have evaluated the use of ICDs in patients who have had an abortive cardiac arrest, a life-threatening VT, or unexplained syncope with high probability that a ventricular tachyarrhythmia was the cause (Zipes, et al., 2006).

U.S. Food and Drug Administration (FDA)

Multiple ICD devices have been approved by the U. S. Food and Drug Administration (FDA) through the Premarket Approval (PMA) process. Manufacturers of ICD devices include Biotronik (Lake Oswego, OR), Boston Scientific (Natick, MA), Sorin Group (Arvada, CO), Medtronic (Minneapolis, MN), and St. Jude Medical (St. Paul, MN),

ICD Use in Patients with Reduced LVEF With/Without Prior MI

Patients with Reduced LVEF and Prior MI: Several randomized controlled trials have evaluated the use of ICDs in patients with reduced LVEF and prior MI (i.e., chronic ischemic cardiomyopathy). The Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT) (n=2521) was designed to determine whether amiodarone or a conservatively programmed shock-only, single-lead ICD would decrease the risk of death from any cause in a broad population of patients with mild-to-moderate heart failure (Bardy, et al., for the SCD-HeFT Investigators, 2005). Patients with NYHA class II or III and LVEF \leq 035% were randomly assigned to conventional therapy for congestive heart failure (CHF) plus placebo (n=847) conventional therapy plus amiodarone (n=845), or conventional therapy plus a conservatively programmed, shock-only, single-lead ICD (n=829). The type of ICD therapy was deliberately selected to treat only rapid, sustained VT or VF. Approximately 52% of patients had ischemic cardiomyopathy. Median follow-up was 45.5 months; there were 244 deaths (29%) in the placebo group, 240 (28%) in the amiodarone group, and 182 (22%) in the ICD group. The authors concluded that amiodarone had no effect on survival, while single-lead, shock-only ICD therapy reduces mortality in this patient population by 23%. The SCD-HeFT trial included patients with and without a history of MI. Subgroup analysis confirmed the benefit of ICD therapy in patients with prior MI and reduced LVEF.

The Multi-Center Automatic Defibrillator Implantation Trial II (MADIT-II) was a randomized controlled trial designed to determine the effect of ICD therapy on survival of post-infarction patients with advanced left ventricular dysfunction (Moss, et al., for the MADIT-II Investigators, 2002). The MADIT-II trial randomized 1232 post-infarction patients with an ejection fraction of \leq 30% to receive ICD (n=742) or conventional therapy (n=490). The primary endpoint was total mortality. During an average follow-up of 20 months, the mortality rates were 19.8% in the conventional-therapy group and 14.2% in the ICD group, representing a 31% reduction in mortality in patients who received an ICD. The trial was stopped early when an analysis revealed that the difference in mortality between the two groups had reached the prespecified efficacy boundary. Goldenberg et al. evaluated the long-term benefit of ICDs as primary prevention in a follow-up of MADIT II published in 2010. At eight years, the cumulative probability of all-cause mortality was 49% among patients treated with an ICD compared to 62% among non-ICD patients (p< 0.001). ICD therapy was associated with a significant reduction in the risk of death during the early phase of the extended follow-up period (0–4 years; p< 0.001) and with continued life-saving benefit during the late phase of follow-up (5–8 years; p=0.02).

A significant association between appropriate ICD shocks and subsequent risk of heart failure was demonstrated in an analysis conducted by Goldenberg et al., for the MADIT-II Investigators (2006). High-risk patients in the MADIT-II trial whose lives were saved by appropriate ICD therapy were also at high risk for developing subsequent heart failure. This resulted in a significant reduction in the risk of sudden death but a concurrent increase in the risk of heart failure events among patients in the defibrillator arm of the trial.

Several additional randomized controlled trials conducted in the 1990's also evaluated the use of ICDs in patients with reduced LVEF and prior MI. The Multi-Center Automatic Defibrillator Implantation Trial (MADIT) (n=196) demonstrated improved survival in high-risk patients treated with ICD therapy, compared to patients treated with conventional medical therapy (Moss, et al., for the MADIT Investigators, 1996). The Multicenter Unsustained Tachycardia Trial (MUSTT) (n=704) evaluated outcomes in patients with sustained ventricular tachyarrhythmias induced by programmed stimulation who were treated with antiarrhythmic therapy, including drugs and ICDs, compared to no antiarrhythmic therapy. Neither the rate of cardiac arrest or death from arrhythmias nor the overall mortality rate was lower in the patients randomized to EP- guided therapy and treated with antiarrhythmic drugs than among the patients assigned to no antiarrhythmic therapy. The risk of cardiac arrest or death from arrhythmia was significantly lower in the patients who received ICDs than among the patients discharged without ICDs ((Buxton et al., 1999).

Patients with Reduced LVEF and No Prior MI: The use of ICDs in patients who have not had an MI but have reduced LVEF (i.e., nonischemic dilated cardiomyopathy [NIDCM]) has been investigated in several small randomized controlled trials. The Defibrillators in Non-Ischemic Cardiomyopathy Treatment Evaluation (DEFINITE) trial included 458 patients with no history of MI who had LVEF \leq 35, asymptomatic nonsustained

supraventricular tachycardia, or ≥ 10 premature ventricular complexes per 24 hours on Holter monitor (Kadish, et al., for the DEFINITE Investigators, 2004). Patients were randomly assigned to receive standard medical therapy (n=229) or standard medical therapy plus a single-chamber ICD (n=229). The mortality rate at two years was 14.1% in the standard-therapy group and 7.9% in the ICD group. There were three sudden deaths from arrhythmias in the ICD group and 14 in the standard-treatment group. The authors concluded that in patients with severe NIDCM treated with angiotensin-converting enzyme (ACE) inhibitors and beta blockers, the implantation of an ICD significantly reduced the risk of sudden death from arrhythmia and was associated with a nonsignificant reduction in the risk of death from any cause.

The Comparison of Medical Therapy, Pacing, and Defibrillation in Heart Failure (COMPANION) trial was designed to determine whether prophylactic cardiac-resynchronization therapy with a pacemaker with or without a defibrillator would reduce the risk of death and hospitalization among patients with advanced chronic heart failure and intraventricular conduction delays (Bristow, et al., for the COMPANION Investigators, 2004). A total of 1520 patients with NYHA class III or IV due to ischemic or nonischemic cardiomyopathy and wide QRS interval were randomly assigned in a 1:2:2 ratio to receive optimal medical management (group 1), optimal medical management plus cardiac resynchronization therapy (group 2), or optimal medical management plus cardiac resynchronization therapy and an ICD (group 3). Mortality at 12-month follow-up was 15% in group 2 and 12% in group 3. The study suggests a small mortality benefit for ICD therapy when compared to cardiac resynchronization therapy, although statistical significance was not achieved.

The Amiodarone Versus Implantable Cardioverter-Defibrillator Randomized Trial in Patients With Nonischemic Dilated Cardiomyopathy and Asymptomatic Nonsustained Ventricular Tachycardia (AMIOVIRT) was a small randomized trial designed to evaluate total morbidity during therapy with amiodarone compared to morbidity using ICD therapy in patients with NIDCM and nonsustained VT (Strickberger, et al., for the AMIOVIRT Investigators, 2003). A total of 103 patients with NIDCM and LVEF $\leq 35\%$ and asymptomatic nonsustained VT were randomized to receive either amiodarone (n=52) or ICD (n=51). Survival at one and three years was similar in both groups, and the trial was stopped early because it was determined that statistical significance could not be demonstrated.

The SCD-HeFT Trial (discussed above) included patients with or without MI with NYHA class II or III and LVEF $\leq 35\%$. As stated, a significant benefit was seen for ICD in patients with prior MI. Although favorable results were also seen in a subgroup analysis comparing ICD with placebo in patients without prior MI, mortality reductions did not achieve statistical significance.

Meta-Analyses: Desai et al. (2004) conducted a meta-analysis to determine whether ICDs reduce all-cause mortality in patients with nonischemic cardiomyopathy. The meta-analysis included four randomized controlled trials discussed above (AMIOVIRT, DEFINITE, COMPANION, and SCD-HeFT) and three additional trials. The studies were reviewed to determine the number of patients randomized, mean duration of follow-up, primary endpoint, mortality of ICD cohort and mortality of control cohort. Pooled analysis of the five primary prevention trials enrolling 1854 patients with nonischemic cardiomyopathy suggested a significant reduction in total mortality among patients randomized to ICD or cardiac resynchronization therapy plus defibrillator vs. medical therapy. Two of the three secondary prevention trials provided subgroup estimates for ICD efficacy in nonischemic cardiomyopathy. Polled analysis of these secondary prevention trials (n=256 patients with NIDCM) indicated an equivalent, although not statistically significant, mortality reduction with ICD therapy. Combined analysis of all seven trials demonstrated a statistically significant 31% overall reduction in mortality with ICD therapy.

Lee et al. (2003) conducted a meta-analysis of nine randomized controlled trials including over 5000 patients to compare the effectiveness of ICDs and medical strategies for the prevention of arrhythmic events and death. The primary and secondary prevention trials showed a significant benefit of the ICD with respect to arrhythmic death. The mortality benefit of the ICD was entirely attributable to a reduction in arrhythmic death in all trials. While the secondary prevention trials demonstrated a robust decrease in all-cause ICD mortality, the pooled primary prevention trials demonstrated decreased all-cause ICD mortality that was dependent on selected individual trials. The authors stated that the disparity in ICD-related mortality reductions in the primary prevention trials was related to variability in the incidence of arrhythmic death between the individual trials. The authors concluded that the ICD decreases the risk of arrhythmic death, although its impact on all-cause mortality is related to the underlying risk of arrhythmia-related death relative to competing causes.

Multiple clinical trials have demonstrated that ICD use results in improved survival compared to antiarrhythmic drug therapy in patients who have had an abortive cardiac arrest, life-threatening ventricular tachycardia, or unexplained syncope with high probability that a ventricular tachyarrhythmia was the cause (i.e. secondary prevention). There is also adequate evidence that ICD use is effective for primary prevention of sudden death and improves survival in selected patients who have not had a cardiac arrest or sustained ventricular tachycardia

ICD Use in Patients with Reduced LVEF and Recent MI

The risk of sudden death following acute myocardial infarction (MI) is highest early after the event, and declines progressively over the next six to twelve months. Evidence published to date from several randomized controlled trials, however, has failed to show a survival benefit for ICD implantation early after MI. The reasons for this acute MI-sudden cardiac death paradox are not yet clear. The pathophysiology of sudden cardiac death in the early post-MI period may differ from that which occurs in the later post-MI period. Since sudden cardiac death is not synonymous with an arrhythmic event, it is possible that the increased incidence of sudden death after acute MI is largely not caused by a lethal ventricular arrhythmia. An ICD, therefore, would not be expected to have an impact on this type of sudden death. Researchers have also suggested that different risk stratifiers are needed to identify patients at high risk for reversible ventricular arrhythmias who may benefit from ICD use. It is also possible that the device implantation and testing itself may be deleterious and negate any potential benefits the device may offer. Poole et al. (2008) evaluated the prognostic importance of defibrillator shocks in patients with heart failure and found that an appropriate ICD shock, as compared to no appropriate shock, was associated with a significant increase in the subsequent risk of death from all causes. High-voltage ICD shocks have been associated with several deleterious effects, including transient myocardial dysfunction and troponin release/elevation, and whether these effects occur more frequently in the setting of a healing vs. healed MI requires further study (Goldberger and Passman, 2009).

In order to better understand the pathophysiological events that lead to sudden death after MI, Pouleur et al. (2010) assessed autopsy results in a series of cases classified as sudden death events in patients enrolled in the VALsartan In Acute myocardial infarctioN Trial (VALIANT). A total of 398 autopsy records were available (14% of deaths), and 105 of these patients had clinical circumstances consistent with sudden death. On the basis of the autopsy findings, the authors assessed the probable cause of sudden death, and how these causes varied with time after MI. Of the 105 deaths considered to be sudden, autopsy results suggested the following causes: three index MIs in the first seven days (2.9%); 28 recurrent MIs (26.6%); 13 cardiac ruptures (12.4%); four pump failures (3.8%); two other cardiovascular causes (stroke or pulmonary embolism) (1.9%); and one non-cardiovascular cause (1%). A total of 54 cases had no acute specific autopsy evidence other than the index MI and were therefore presumed to be arrhythmic. The percentage of sudden death due to recurrent MI or rupture was highest in the first month after the index MI. Conversely, after three months, the percentage of presumed arrhythmic death was higher than recurrent MI or rupture ($p < 0.0001$). The authors stated that these findings may help explain the lack of benefit of early ICD therapy.

The Immediate Risk Stratification Improves Survival (IRIS) trial tested the hypothesis that patients at increased risk who are treated early with an ICD will live longer than those who receive optimal medical therapy alone (Steinbeck, et al., for the IRIS Investigators, 2009). This randomized, prospective, multicenter trial registered 62,944 unselected patients with MI. Of these patients, 898 were enrolled 5 to 31 days after the event if they met the following criteria: LVEF $\leq 40\%$ and heart rate of 90 or more on the first available electrocardiogram (criterion 1, $n=602$); non-sustained ventricular tachycardia ≥ 150 during Holter monitoring (criterion 2, $n=208$), or both criteria ($n=88$). Patients were randomized to ICD treatment ($n=445$) or medical therapy alone ($n=453$). During a mean follow-up of 37 months, 233 patients died; 116 in the ICD group and 117 in the control group. Overall mortality was not reduced in the ICD group ($p=0.78$). There were fewer sudden cardiac deaths in the ICD group than in the control group (27 vs. 60, $p=0.049$), but the number of non-cardiac deaths was higher in the ICD group than in the control group (68 vs. 39, $p=0.001$).

The Beta-Blocker Strategy plus Implantable Cardioverter Defibrillator (BEST + ICD) Trial ($n=143$) (Raviele, et al., 2005) was a randomized double-arm observational study to determine whether survival is improved in high-risk post-MI patients treated with beta blockers and ICD therapy compared to patients treated with conventional therapy. Enrolled patients were survivors of MI (< 1 month) with LVEF $\leq 35\%$ and either frequent (≥ 10 /hour) premature ventricular contractions (PVCs), depressed heart rate variability, or abnormal signal-averaged ECG, who were able to tolerate optimum beta blocker therapy (68 ± 40 mg/day of metoprolol). Patients were randomized in a 2:3 ratio to conventional strategy ($n=59$) or EPS-guided ICD strategy ($n=79$). ICD implantation

took place in 24 patients in the latter group, with the remaining 55 patients treated with conventional therapy. During a mean follow-up of 540 ± 378 days, nine patients died (6.5%) due to sudden death and nine (6.5%) due to nonsudden death, and four (3%) due to noncardiac causes. The overall mortality for the conventional and EPS-guided ICD arms was 18% vs. 14% after one year and 29.5% vs. 20% after two years, respectively. The authors stated that, despite optimal therapy, mortality remained significant in high-risk patients following MI, and although there was a trend in favor of EPS-guided ICD therapy, the data are insufficient to demonstrate a survival benefit of this strategy early after MI. The authors stated that the study was inadequately powered to determine whether the EPS + ICD strategy should be implemented soon after MI or delayed until a later phase. Although over 15,000 patients were screened, difficulties in enrollment caused the study to end with only 12% of the target population randomized.

The Defibrillator in Acute Myocardial Infarction Trial (DINAMIT) was a randomized open-label comparison of ICD therapy and no ICD therapy in patients 6–40 days following an MI (Hohnloser, et al., on behalf of the DINAMIT Investigators, 2004). Patients with reduced LVEF of $\leq 35\%$ and impaired cardiac autonomic function (i.e., depressed heart rate variability or elevated average 24-hour heart rate on Holter monitoring) were randomized to ICD therapy (n=332) or no ICD therapy (n=342). The primary outcome was mortality from any cause, and the secondary outcome was death from arrhythmia. During a mean follow-up of 30 ± 13 months, there was no difference in overall mortality between the two groups. Of 120 patients who died, 62 were in the ICD group, and 58 were in the control group. There were 12 deaths due to arrhythmia in the ICD group and 29 in the control group. There were 50 deaths from nonarrhythmic causes in the ICD group, however, and 29 in the control group. The authors concluded that ICD therapy does not reduce overall mortality in high-risk patients who have recently had an MI. Although ICD therapy was associated with a reduction in arrhythmia-related death, this was offset by an increase in nonarrhythmic-related death.

Wilber et al. (2004) conducted a retrospective analysis of MADIT-II data to examine whether the mortality risk and survival benefit of ICD therapy is dependent on elapsed time from MI. MADIT-II randomized patients with ejection fractions of $\leq 30\%$ to either ICD or conventional care. A total of 1159 of these patients were enrolled more than one month following MI (mean 81 ± 78 months). Time dependence of mortality and ICD benefit were examined by dividing times from most recent MI to enrollment into approximate quartiles of < 18 months, 18–59 months, 60–119 months, and ≥ 120 months. Mortality rate in the conventionally treated group increased as a function of time from most recent MI to enrollment in MADIT-II from 7.8 per hundred-person years in the recent MI group to 14.0 in the most remote MI group. Mortality in the ICD group was consistently lower in each quartile and demonstrated minimal time-dependent changes in mortality from 7.2 to 9.0. There appeared to be a trend toward increasing survival benefit associated with ICD therapy as time from MI increased. The authors concluded that mortality risk increases as a function of time from MI, and that the survival benefit of ICD treatment appears to be greater for remote MI and remains substantial for at least 15 years after MI.

Data from well designed randomized controlled trials have failed to demonstrate that prophylactic ICD therapy improves overall mortality in patients with acute MI who are considered to be at increased risk. Additional research is needed to determine the actual causes of death in this patient population, and which interventions may be needed to reduce the incidence of sudden death

National Institute for Clinical Excellence (NICE) Guidance (United Kingdom)

A NICE Technology Appraisal on the use of ICDs for arrhythmias updated in 2006 states that ICDs are recommended for patients in the following categories:

Secondary prevention, i.e., for patients who present in the absence of a treatable cause with one of the following:

- having survived a cardiac arrest due to either VT or VF
- spontaneous sustained VT causing syncope or significant hemodynamic compromise
- sustained VT without syncope or cardiac arrest, and who have an associated reduction in ejection fraction of less than 35% but are no worse than NYHA class 3

Primary prevention for patients with

- a history of previous (more than four weeks) MI and either:
 - left ventricular dysfunction with LVEF less than 35% and no worse than NYHA class III, non-sustained VT on Holter 24-hour ECG monitoring, and inducible VT on EP testing

or

- left ventricular dysfunction with an LVEF of less than 30% and no worse than NYHA class III, and QRS duration of ≥ 120 milliseconds
- a familial cardiac condition with a high risk of sudden death, including long QT syndrome, hypertrophic cardiomyopathy, Brugada syndrome, an arrhythmogenic right ventricular dysplasia (ARVD), or have undergone surgical repair of congenital heart disease

Agency for Healthcare Research and Quality (AHRQ)

An AHRQ evidence report/technology assessment prepared by the University of Alberta Evidence-based Practice Center (2007) evaluated the safety, efficacy, and effectiveness of cardiac resynchronization therapy and ICDs in patients with LV systolic dysfunction. A total of 12 randomized controlled trials (8516 patients) were identified for the efficacy review, 48 studies (15,097 patients) were identified for the safety review, and all studies enrolled only patients with LV systolic dysfunction. An additional 12 studies were included (68,848 patients) for analysis of peri-implant outcomes for all patients with ICD (i.e., not limited to patients with LV systolic dysfunction). In patients with LV systolic dysfunction, ICD reduced all-cause mortality by 20%; ICD implant success rate was 99%, and peri-implant deaths occurred in 1.2% of LV systolic dysfunction patients and 1.3% of all implants. The frequency of post-implantation complications in LV systolic dysfunction patients per 100 patient years included 1.4 device malfunctions, 1.5 lead problems, 0.6 implant site infections, and 19.1 inappropriate discharges in randomized controlled trial participants and 4.7 inappropriate discharges in patients enrolled in observational studies.

The authors concluded that there is high-quality evidence that ICD reduces all-cause mortality in patients with LVD $\leq 35\%$ and NYHA class II and III symptoms. The relative reduction in all-cause mortality of 20% equates to preventing one death over 35 months for every 20 patients who receive an ICD, although there is no improvement in functional status or morbidity. The authors stated that identifying patients at increased risk for sudden cardiac death and therefore most likely to benefit from ICD placement is vitally important. Although trial eligibility criteria are often cited as a method to identify appropriate patients for ICD placement, two-thirds to three-quarters of ICD recipients in the reviewed trials never received any therapeutic ICD discharges, and in patients who received an appropriate discharge, the benefits were offset over time by deaths due to progressive heart failure. Less than a quarter of cardiac arrest victims have an LVEF $< 30\%$. The meta-regression analysis did not reveal any statistically significant differences in the examined subgroups, but these analyses were post hoc and underpowered because of the small number of trials.

Professional Societies/Organizations

ACC/AHA/Heart Rhythm Society (HRS) Guidelines for Device-Based Therapy of Cardiac Rhythm Abnormalities (Epstein et al.) were published in 2008 to update the 2002 ACC/AHA/NASPE guideline on implantation of cardiac pacemakers and antiarrhythmic devices. Guideline recommendations are classified as Class I, Class IIa, Class IIb, and Class III. The classification system is described as follows:

- Class I: Benefit \ggg Risk; Procedure/Treatment should be performed/administered
- Class IIa: Benefit \gg Risk; Additional studies with focused objectives needed. It is reasonable to perform procedure/administer treatment
- Class IIb: Benefit \geq Risk; Additional studies with broad objectives needed; additional registry data would be helpful. Procedure/treatment may be considered.
- Class III: Risk \geq Benefit; Procedure/treatment should not be performed/administered, since it is not helpful and may be harmful.

The weight of evidence supporting each recommendation is classified as follows:

- Level A: Multiple populations evaluated. Data derived from multiple randomized clinical trials or meta-analyses.
- Level B: Limited populations evaluated. Data derived from a single randomized trial or nonrandomized studies.
- Level C: Very limited populations evaluated. Only consensus opinion of experts, case studies, or standard of care.

The following recommendations for ICD placement are included in the 2008 guideline:

Class I

- ICD therapy is indicated in patients who are survivors of cardiac arrest due to VF or hemodynamically unstable sustained VT after evaluation to define the cause of the event and to exclude any completely reversible causes. *(Level of Evidence: A)*
- ICD therapy is indicated in patients with structural heart disease and spontaneous sustained VT, whether hemodynamically stable or unstable. *(Level of Evidence: B)*
- ICD therapy is indicated in patients with syncope of undetermined origin with clinically relevant, hemodynamically significant sustained VT or VF induced at electrophysiological study. *(Level of Evidence: B)*
- ICD therapy is indicated in patients with LVEF less than 35% due to prior MI who are at least 40 days post-MI and are in NYHA functional Class II or III. *(Level of Evidence: A)*
- ICD therapy is indicated in patients with nonischemic DCM who have an LVEF less than or equal to 35% and who are in NYHA functional Class II or III. *(Level of Evidence: B)*
- ICD therapy is indicated in patients with LV dysfunction due to prior MI who are at least 40 days post-MI, have an LVEF less than 30%, and are in NYHA functional Class I. *(Level of Evidence: A)*
- ICD therapy is indicated in patients with nonsustained VT due to prior MI, LVEF less than 40%, and inducible VF or sustained VT at electrophysiological study. *(Level of Evidence: B)*

Class IIa

- ICD implantation is reasonable for patients with unexplained syncope, significant LV dysfunction, and nonischemic DCM. *(Level of Evidence: C)*
- ICD implantation is reasonable for patients with sustained VT and normal or near-normal ventricular function. *(Level of Evidence: C)*
- ICD implantation is reasonable for patients with HCM who have one or more major risk factors for SCD. *(Level of Evidence: C)*
- ICD implantation is reasonable for the prevention of SCD in patients with ARVD/C who have 1 or more risk factors for SCD. *(Level of Evidence: C)*
- ICD implantation is reasonable to reduce SCD in patients with long-QT syndrome who are experiencing syncope and/or VT while receiving beta blockers. *(Level of Evidence: B)*
- ICD implantation is reasonable for non hospitalized patients awaiting transplantation. *(Level of Evidence: C)*
- ICD implantation is reasonable for patients with Brugada syndrome who have had syncope. *(Level of Evidence: C)*
- ICD implantation is reasonable for patients with Brugada syndrome who have documented VT that has not resulted in cardiac arrest. *(Level of Evidence: C)*
- ICD implantation is reasonable for patients with catecholaminergic polymorphic VT who have syncope and/or documented sustained VT while receiving beta blockers. *(Level of Evidence: C)*
- ICD implantation is reasonable for patients with cardiac sarcoidosis, giant cell myocarditis, or Chagas disease. *(Level of Evidence: C)*

Class IIb

- ICD therapy may be considered in patients with nonischemic heart disease who have an LVEF of less than or equal to 35% and who are in NYHA functional Class I. *(Level of Evidence: C)*
- ICD therapy may be considered for patients with long-QT syndrome and risk factors for SCD. *(Level of Evidence: B)* (16,349–354)
- ICD therapy may be considered in patients with syncope and advanced structural heart disease in whom thorough invasive and noninvasive investigations have failed to define a cause. *(Level of Evidence: C)*
- ICD therapy may be considered in patients with a familial cardiomyopathy associated with sudden death. *(Level of Evidence: C)*
- ICD therapy may be considered in patients with LV noncompaction. *(Level of Evidence: C)*

Class III

- ICD therapy is not indicated for patients who do not have a reasonable expectation of survival with an acceptable functional status for at least 1 year, even if they meet ICD implantation criteria specified in the Class I, IIa, and IIb recommendations above. *(Level of Evidence: C)*

- ICD therapy is not indicated for patients with incessant VT or VF. *(Level of Evidence: C)*
- ICD therapy is not indicated in patients with significant psychiatric illnesses that may be aggravated by device implantation or that may preclude systematic follow-up. *(Level of Evidence: C)*
- ICD therapy is not indicated for NYHA Class IV patients with drug-refractory congestive heart failure who are not candidates for cardiac transplantation or CRT-D. *(Level of Evidence: C)*
- ICD therapy is not indicated for syncope of undetermined cause in a patient without inducible ventricular tachyarrhythmias and without structural heart disease. *(Level of Evidence: C)*
- ICD therapy is not indicated when VF or VT is amenable to surgical or catheter ablation (e.g., atrial arrhythmias associated with the Wolff-Parkinson-White syndrome, RV or LV outflow tract VT, idiopathic VT, or fascicular VT in the absence of structural heart disease). *(Level of Evidence: C)*
- ICD therapy is not indicated for patients with ventricular tachyarrhythmias due to a completely reversible disorder in the absence of structural heart disease (e.g., electrolyte imbalance, drugs, or trauma). *(Level of Evidence: B)*

The 2008 ACC/AHA/HRS guideline provides the following recommendations for ICD placement in children, adolescents, and patients with congenital heart disease:

Class I

- ICD implantation is indicated in the survivor of cardiac arrest after evaluation to define the cause of the event and to exclude any reversible causes. *(Level of Evidence: B)*
- ICD implantation is indicated for patients with symptomatic sustained VT in association with congenital heart disease who have undergone hemodynamic and electrophysiological evaluation. Catheter ablation or surgical repair may offer possible alternatives in carefully selected patients. *(Level of Evidence: C)*

Class IIa

- ICD implantation is reasonable for patients with congenital heart disease with recurrent syncope of undetermined origin in presence of either ventricular dysfunction or inducible ventricular arrhythmias at electrophysiological study. *(Level of Evidence: B)*

Class IIb

- ICD implantation may be considered for patients with recurrent syncope associated with complex congenital heart disease and advanced systemic ventricular dysfunction when thorough invasive and noninvasive investigations have failed to define a cause. *(Level of Evidence: C)*

Class III

- All Class III recommendations listed above apply to pediatric patients and patients with congenital heart disease, and ICD implantation is not indicated in these patient populations. *(Level of Evidence: C)*

Summary

There is sufficient evidence in the published medical literature to demonstrate the safety and efficacy of ICDs for primary and secondary prevention of sudden cardiac death in selected high-risk patients. There is insufficient evidence in the published medical literature, however, to demonstrate the safety and efficacy of prophylactic ICD therapy in patients with recent acute MI who are considered to be at increased risk. Evidence published to date does not demonstrate a reduction in mortality when ICD therapy is used in this patient population. The pathophysiology of sudden cardiac death in the early post-MI period may differ from that which occurs in the later post-MI period. Additional research is needed to determine the actual causes of death in this patient population, and which interventions may reduce the incidence of sudden death

Coding/Billing Information

Note: This list of codes may not be all-inclusive.

Covered when medically necessary:

CPT®*	Description
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Codes	
33216	Insertion of a transvenous electrode; single chamber (one electrode) permanent pacemaker or single chamber pacing cardioverter-defibrillator
33217	Insertion of a transvenous electrode; dual chamber (two electrodes) permanent pacemaker or dual chamber pacing cardioverter-defibrillator
33224	Insertion of pacing electrode, cardiac venous system, for left ventricular pacing, with attachment to previously placed pacemaker or pacing cardioverter-defibrillator pulse generator (including revision of pocket, removal, insertion and/or replacement of generator)
33225	Insertion of pacing electrode, cardiac venous system, for left ventricular pacing, at time of insertion of pacing cardioverter-defibrillator or pacemaker pulse generator (including upgrade to dual chamber system) (List separately in addition to code for primary procedure)
33240	Insertion of single or dual chamber pacing cardioverter-defibrillator pulse generator
33249	Insertion or repositioning of electrode lead(s) for single or dual chamber pacing cardioverter-defibrillator and insertion of pulse generator

HCPCS Codes	Description
C1721	Cardioverter-defibrillator; dual chamber (implantable)
C1722	Cardioverter-defibrillator; single chamber (implantable)
C1882	Cardioverter-defibrillator, other than single or dual chamber (implantable)

ICD-9-CM Diagnosis Codes	Description
086.0	Chagas' disease with heart involvement
422.91	Idiopathic myocarditis
425.1	Hypertrophic obstructive cardiomyopathy
425.4	Other primary cardiomyopathies
425.9	Secondary cardiomyopathy, unspecified
426.82	Long QT syndrome
427.1	Paroxysmal ventricular tachycardia
427.41	Ventricular fibrillation
427.5	Cardiac arrest
428.0-428.9	Heart Failure
780.2	Syncope and collapse
V12.53	Personal history of sudden cardiac arrest

***Current Procedural Terminology (CPT®) © 2010 American Medical Association: Chicago, IL.**

References

1. Almendral J, Josephson ME. All patients with hemodynamically tolerated postinfarction ventricular tachycardia do not require an implantable cardioverter-defibrillator. *Circulation*. 2007 Sep 4;116(10):1204-12.
2. Antman EM, Anber DT, Armstrong, PW, Bates ER, Green LA, Hand M, et al. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1999 Guidelines for the Management of Patients with Acute Myocardial Infarction). 2004. *Am Coll Cardiol*. 2004 Aug 4;44(3):671-719. No abstract available. Erratum in: *J Am Coll Cardiol*. 2005 Apr 19;45(8):1376.

3. Bardy G, Lee KL, Mark DB, Poole JE, Packer DL, Boineau R for the Sudden Cardiac Death in Heart Failure Trial (SCD0HeFT) Investigators. Amiodarone or an implantable cardioverter-defibrillator for congestive heart failure. *N Engl J Med.* 2005 Jan 20;352(3):225-37. Erratum in: *N Engl J Med.* 2005 May 19;352(20):2146.
4. Berul CI, Van Hare G, Kertesz NJ, Dubin AM, Cecchin F, Collins KK, et al. Results of a multicenter retrospective implantable cardioverter-defibrillator registry of pediatric and congenital heart disease patients. *Am Coll Cardiol.* 2008 Apr 29;51(17):1685-91.
5. Bristow MR, Saxon LA, Boehmer J, Krueger S, Kass DA, De Marco T, et al. Cardiac-resynchronization therapy with or without an implantable defibrillator in advanced chronic heart failure. *N Engl J Med.* 2004 May 20;350(21):2140-50.
6. Bunch TJ, Hohnloser SH, Gersh BJ. Mechanisms of sudden cardiac death in myocardial infarction survivors: insights from the randomized trials of implantable cardioverter-defibrillators. *Circulation.* 2007 May 8;115(18):2451-7.
7. Buxton AE, Lee KL, Fisher JD, Josephson ME, Prystowsky EN, Hafley G. A randomized study of the prevention of sudden death in patients with coronary artery disease. Multicenter Unsustained Tachycardia Trial Investigators (MUSTT). *N Engl J Med.* 1999 Dec;341(25):1882-90.
8. Buxton AE, Lee KL, Hafley GE, Pires LA, Fisher JD, Gold MR, et al. for the MUSTT Investigators. Limitations of ejection fraction for prediction of sudden death risk in patients with coronary artery disease: lessons from the MUSTT study. *J Am Coll Cardiol.* 2007 Sep 18;50(12):1150-7.
9. Carlson MD, Wilkoff BL, Maisel WH, Carlson MD, Ellenbogen KA, Saxon LA, et al. Recommendations from the Heart Rhythm Society Task Force on Device Performance Policies and Guidelines Endorsed by the American College of Cardiology Foundation (ACCF) and the American Heart Association (AHA) and the International Coalition of Pacing and Electrophysiology Organizations (COPE). *Heart Rhythm.* 2006 Oct;3(10):1250-73.
10. Cawley PJ, Al-Khatib SM. Amiodarone versus implantable cardioverter defibrillator for asymptomatic nonsustained ventricular tachycardia in nonischemic dilated cardiomyopathy. *Am Heart J.* 2004 May;147(5):790-1.
11. Connolly SJ, Gent M, Roberts RS, Dorian P, Roy D, Sheldon RS for the CIDS Investigators. Canadian implantable defibrillator study (CIDS) : A randomized trial of the implantable cardioverter defibrillator against amiodarone. *Circulation.* 2000 Mar 21;101(11):1297-302.
12. Connolly SJ, Hallstrom AP, Cappato R, Schron EB, Kuck KH, Zipes HL on behalf of the Investigators of the AVID, CASH and CIDS studies. Meta-analysis of the implantable cardioverter defibrillator secondary prevention trials. AVID, CASH and CIDS studies. Antiarrhythmics vs Implantable Defibrillator study. Cardiac Arrest Study Hamburg . Canadian Implantable Defibrillator Study. *Eur Heart J.* 2000 Dec;21(24):2071-8.
13. Desai AS, Fang JC, Maisel, WH, Baughman KL. Implantable defibrillators for the prevention of mortality in patients with nonischemic cardiomyopathy: a meta-analysis of randomized controlled trials. *JAMA.* 2004 Dec 15;292(23):2874-9.
14. Ellenberger K, A, Levine JH, Berger RD, Daubert JP, Winters SL, Greenstein E, et al., for the DEFINITE Investigators. Are implantable cardioverter defibrillator shocks a surrogate for sudden cardiac death in patients with nonischemic cardiomyopathy? *Circulation.* 2006 Feb 14;113(6):776-82. Epub 2006 Feb 6.
15. Epstein AE, DiMarco JP, Ellenbogen KA, Estes NA 3rd, Freedman RA, Gettes LS, et al. American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the ACC/AHA/NASPE 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices); American Association for Thoracic Surgery; Society of Thoracic Surgeons. ACC/AHA/HRS 2008 Guidelines for Device-Based Therapy of Cardiac Rhythm

Abnormalities: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the ACC/AHA/NASPE 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices) developed in collaboration with the American Association for Thoracic Surgery and Society of Thoracic Surgeons. *Am Coll Cardiol.* 2008 May 27;51(21):e1-62.

16. Estes NA III, Weinstock J, Wang PJ, Homoud MK, Link MA. Use of anti-arrhythmics and implantable cardioverter-defibrillators in congestive heart failure. *Am J Cardiol.* 2003 Mar;91(6A):45D-52D.
17. Ezekowitz JA, Rowe BH, Dryden DM, Hooton N, Vandemeer B, Spooner C, McAlister FA. Systematic review: implantable cardioverter defibrillators for adults with left ventricular systolic dysfunction. *Ann Intern Med.* 2007 Aug 21;147(4):251-62.
18. Goldberger JJ, Passman R. Implantable cardioverter-defibrillator therapy after acute myocardial infarction: the results are not shocking. *J Am Coll Cardiol.* 2009 Nov 24;54(22):2001-5.
19. Goldenberg I, Gillespie J, Moss AJ, Hall WJ, Klein H, McNitt S, Brown MW, et al. Executive Committee of the Multicenter Automatic Defibrillator Implantation Trial II. Long-term benefit of primary prevention with an implantable cardioverter-defibrillator: an extended 8-year follow-up study of the Multicenter Automatic Defibrillator Implantation Trial II. *Circulation.* 2010 Sep 28;122(13):1265-71. Epub 2010 Sep 13
20. Goldenberg I, Moss AJ. Long QT syndrome. *J Am Coll Cardiol.* 2008 Jun 17;51(24):2291-300.
21. Goldenberg I, Moss AJ, Hall, J, McNitt S, Zareba W, Andrews ML, et al. Causes and consequences of heart failure after prophylactic implantation of a defibrillator in the multicenter automatic defibrillator implantation trial II. *Circulation.* 2006 Jun 20;113(24):2810-7. Epub 2006 Jun 12.
22. Goldenberg I, Moss AJ, McNitt S, Zareba W, Hall WJ, Andrews ML, et al. Time dependence of defibrillator benefit after coronary revascularization in the Multicenter Automatic Defibrillator Implantation Trial (MADIT)-II. *J Am Coll Cardiol.* 2006 May 2;47(9):1811-7. Epub 2006 Apr 17.
23. Gregoratos G, Abrams J, Epstein AE, Freedman RA, Hayes DL, Hlatky, et al. ACC/AHA/NASPE 2002 guideline update for implantation of cardiac pacemakers and antiarrhythmia devices: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/NASPE Committee to Update the 1998 Pacemaker Guidelines). *Circulation.* 2002 Oct 15;106(16):2145-61.
24. Greenberg H, Case RB, Moss AJ, Brown MW, Carroll ER, Andrews ML and MADIT-II Investigators. Analysis of mortality events in the Multicenter Automatic Defibrillator Implantation Trial (MADIT-II). *J Am Coll Cardiol.* 2004 Apr 21;43(8):1459-65.
25. Hohnloser, Sh, Heinz K, Dorian D, Roberts R, Hampton JR, Hatala, et al. on behalf of the DINAMIT Investigators. Prophylactic use of an implantable cardioverter-defibrillator after acute myocardial infarction. *N Engl J Med.* 2004 Dec 9;351(24):2481-8.
26. Kadish A, Dyer A, Daubert JP, Quigg R, Estes NA, Anderson KP, et al. Prophylactic defibrillator implantation in patients with nonischemic dilated cardiomyopathy. *N Engl J Med.* 2004 May 20;350(21):2151-8.
27. Kadish A, Schaechter A, Subacius H, Thattassery E, Sanders W, Anderson KP, et al. Patients with recently diagnosed nonischemic cardiomyopathy benefit from implantable cardioverter-defibrillators. *J Am Coll Cardiol.* 2006 Jun 20;47(12):2477-82. Epub 2006 May 30.
28. Kusmirek SL, Gold MR. Sudden cardiac death: the role of risk stratification. *Am Heart J.* 2007 Apr;153(4 Suppl):25-33.

29. Lee DS, Green LD, Liu PP, Dorian P, Newman DM, Grant FC, et al. Effectiveness of implantable defibrillators for preventing arrhythmic events and death: a meta-analysis. *J Am Coll Cardiol*. 2003 May 7;41(9):1573-82.
30. Libby: Braunwald's heart disease: a textbook of cardiovascular medicine, 8th ed. Saunders, an imprint of Elsevier; 2007.
31. Lim HS, Lip GYH, Tse H-F. Implantable cardioverter defibrillator following acute myocardial infarction: the '48-hour' and '40-day' rule. *Europace*. 2008 May;10(5):536-9. Epub 2008 Mar 26.
32. Maron BJ, McKenna WJ, Danielson GK, Kappenberger LJ, Kuhn HJ, Seidman CE, et al. ACC/ESC clinical expert consensus document on hypertrophic cardiomyopathy: a report of the American College of Cardiology Task Force on Clinical Expert Consensus Documents and the European Society of Cardiology Committee for Practice Guidelines (Committee to Develop an Expert Consensus Document on Hypertrophic Cardiomyopathy). Accessed Dec 3, 2009. Available at URL address: <http://www.acc.org/qualityandscience/clinical/consensus/cardiomyopathy/index.htm>
33. McAlister FA, Ezekowitz J, Dryden DM, Hooton N, Bandermeer B, Friesen C, et al. Cardiac resynchronization therapy and implantable cardiac defibrillators in left ventricular systolic dysfunction. *Evid Rep Technol Assess (Full Rep)*. 2007 Jun;(152):1-199.
34. Moss AJ, Hall WJ, Cannom DS, Daubert JP, Higgins SL, Klein H, et al. for the Multicenter Automatic Defibrillator Implantation Trial Investigators. Improved survival with an implanted defibrillator in patients with coronary disease at high risk for ventricular arrhythmia.. *N Engl J Med*. 1996 Dec 26;335(26):1933-40.
35. Moss AJ, Wojciech Z, Hall J, Klein H, Wilber DJ, Cannom DS, et al. for the Multicenter Automatic Defibrillator Implantation Trial Investigators. Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. *N Engl J Med*. 2002 Mar 21;346(12):877-83. Epub 2002 Mar 19.
36. Myerburg RJ. Implantable cardioverter-defibrillators after myocardial infarction. *N Engl J Med*. 2008 Nov 20;359(21):2245-53.
37. National Institute for Health and Clinical Excellence (NICE). Technology appraisal 11. Implantable cardioverter defibrillators for arrhythmias. London, UK: NICE; 2006 Jan. Accessed Dec 3, 2009. Available at URL address: <http://www.nice.org.uk/search/guidancesearchresults.jsp?keywords=icd&searchType=Guidance>
38. Passman R, Kadish A, Sudden death prevention with implantable devices. *Circulation*. 2007 Jul 31;116(5):561-71.
39. Pelosi F, Morady F. Sudden cardiac death and implantable cardioverter-defibrillators. In: Topol EJ, editor. *Textbook of cardiovascular medicine*, 3rd ed. Lippincott, Williams & Wilkins; 2007.
40. Poole JE, Johnson GW, Hellkamp AS, Anderson J, Callans DJ, Raitt MH, et al. Prognostic importance of defibrillator shocks in patients with heart failure. *N Engl J Med*. 2008 Sep 4;359(10):1009-17.
41. Pouleur AC, Barkoudah E, Uno H, Skali H, Finn PV, Zelenkofske SL, et al.; VALIANT Investigators. Pathogenesis of sudden unexpected death in a clinical trial of patients with myocardial infarction and left ventricular dysfunction, heart failure, or both. *Circulation*. 2010 Aug 10;122(6):597-602. Epub 2010 Jul 26.
42. Raviele A, Bongiorno MG, Brignole M, Cappato R, Capucci A, Gaita F, for the BEST + ICD Trial Investigators. Early EPS/ICD strategy in survivors of acute myocardial infarction with severe left ventricular dysfunction on optimal beta-blocker treatment. The BEta-blocker STRategy plus ICD trial. *Europace*. 2005 Jul;7(4):327-37.

43. Solomon SD, Zelenkofske S, McMurray JJV, Finn PV, Velazquez E, Ertl G, et al., for the Valsartan in Acute Myocardial Infarction Trial (VALIANT) Investigators. Sudden death in patients with myocardial infarction and left ventricular dysfunction, heart failure, or both. *N Engl J Med.* 2005 Jun 23;352(25):2581-8. Erratum in: *N Engl J Med.* 2005 Aug 18;353(7):744.
44. Steinbeck G, Andresen D, Seidl K, Brachmann J, Hoffmann E, et al., for the IRIS Investigators. Defibrillator implantation early after myocardial infarction. *N Engl J Med.* 2009 Oct 8;361(15):1427-36
45. Tung R, Zimetbaum P, Josephson ME. A critical appraisal of implantable cardioverter-defibrillator therapy for the prevention of sudden cardiac death. *J Am Coll Cardiol.* 2008 Sep 30;52(14):1111-21.
46. Wilber DJ, Wojciech Z, Hall WJ, Brown MW, Lin AC, Andrews ML, et al. Time dependence of mortality risk and defibrillator benefit after myocardial infarction. *Circulation.* 2004 Mar 9;109(9):1082-4. Epub 2004 Mar 1.
47. Yap YG, Duong T, Bland M, Malik M, Torp-Pedersen C, Kober L, et al. Optimising the dichotomy limit for left ventricular ejection fraction in selecting patients for defibrillator therapy after myocardial infarction. *Heart.* 2007 Jul;93(7):832-6. Epub 2007 Jan 19.
48. Yap YG, Duong T, Bland M, Malik M, Torp-Pedersen C, Kober L, et al. Temporal trends on the risk of arrhythmic vs. non-arrhythmic deaths in high-risk patients after myocardial infarction: a combined analysis from multicentre trials. *Eur Heart J.* 2005 Jul;26(14):1385-93. Epub 2005 May 24.
49. Zipes DP, Camm AJ, Borggreffe M, Buxton AE, Chaitman B, Fromer M, et al. ACC/AHA 2006 guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: a report of the American College of Cardiology/American Heart Association Task Force and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Develop Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death). *J Am Coll Cardiol* 2006;48:e247-346.

Policy History

Pre-Merger Organizations	Last Review Date	Policy Number	Title
CIGNA HealthCare	01/15/2007	0181	Implantable Cardioverter Defibrillator (ICD) Implantable Cardioverter Defibrillator (ICD)
Great-West Healthcare	08/28/2006	04.246.02	Cardioverter Defibrillator, Implantable and Wearable

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