CONTINUING MEDICAL EDUCATION

Current Pacemaker and Defibrillator Therapy

Hans-Joachim Trappe and Jan Gummert

SUMMARY

Background: Pacemakers have been available for 50 years, and implantable cardiac defibrillators for 25. Clear indications for each have been established on the basis of data from randomized clinical trials (RCTs).

Methods: This review article is the product of a collaborative effort by a cardiologist and a cardiac surgeon. The authors arrived at a consensus through a selective review of the literature, with special attention to randomized controlled trials and registry data.

Results: Atrioventricular (AV) block only rarely necessitates permanent pacemaker stimulation after inferior myocardial infarction, of which it is a rare (12% to 20%) and often transient accompaniment. AV block is more common, however, in anterior wall infarction (frequency ca. 5%), and often necessitates permanent pacemaker therapy in such cases. Pacemaker complications are rare; they include oversensing (the detection of impulse noise) (0.7%), undersensing (the failure to detect impulses) (3.8%), electrode fractures (3.8%), isolation defects (3.4%), perforation (<1%), dislocation (<1%), and infection (<1% to 12%). Many RCTs have confirmed that defibrillators are effective in the prevention of sudden cardiac death (SCD): they lower the risk of SCD by 20% to 30% in primary prevention and by 20% to 40% in secondary prevention. Cardiac resynchronization therapy improves the clinical manifestations and outcome of patients with congestive heart failure, with a relative risk reduction (RR) of 20% to 40%, even among patients in NYHA classes I and II (RR ca. 40%). Implantable defibrillators only rarely cause problems or complications in either the short or the long term. Emotional disturbances, including anxiety, are a rare side effect, occurring in less than 1% of cases.

Conclusion: Pacemakers and implantable defibrillators are well-established electrotherapeutic devices that are highly effective and have only rare complications.

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B radyarrhythmias (i.e., cardiac arrhythmias with a ventricular rate below 50/min) and especially tachyarrhythmias (those with a ventricular rate above 100/min) are often life-threatening. In Germany, some 100 000 persons sustain a cardiac arrest each year, caused in 65% to 80% of cases by a ventricular tachyarrhythmia (1). Bradycardia is a less common cause of cardiac arrest, accounting for only 5% to 20% of cases. Pacemakers have been available for 50 years, and implantable defibrillators for 25 years, as electrotherapy for cardiac arrhythmia.

Learning objectives

The learning objectives for readers of this article are

- to become acquainted with the indications for temporary and permanent pacemaker and defibrillator therapy, and to know their differential diagnosis;
- to be able to recognize the early and late pacemaker complications by their clinical features.

Methods

This review article is based on the current guidelines of the German, European, and American medical societies for pacemaker and defibrillator therapy and on recent pertinent literature retrieved by a search in PubMed. The authors derived their consensus views from these publications.

Indications for pacemaker therapy

The indications for pacemaker therapy that are presented here have been confirmed by randomized, controlled studies (2–4). Bradyarrhythmias generally require treatment when an insufficient intrinsic ventricular rate leads to major clinical manifestations such as dizziness, syncope, incipient heart failure, or persistent myocardial ischemia. The indications for temporary electrotherapy differ in patients with inferior and anterior infarcts because of differences in the anatomy and pathophysiology of the AV node.

Incidence

In Germany, some 100 000 persons sustain a cardiac arrest each year, caused in 65% to 80% of cases by a ventricular tachyarrhythmia.

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TABLE 1

Indications for temporary and permanent pacemaker stimulation^{*1}

	Temporary stimulation	Permanent stimulation
Sinus node dysfunction	Symptomatic patient without response to atropine	Symptomatic patient with sinuatrial blockades
AV block	3rd-degree AV block without adequate escape rhythm: – symptomatic patient – ventricular irritability – hemodynamic worsening Symptomatic patient with 2nd-degree AV block	Persistent 3rd-degree AV block in posterior wall infarction (more than 10 days after infarction) Persistent 3rd-degree AV block in anterior wall infarction Symptomatic patient with persistent 2nd-degree AV block
Bundle branch block	– Anterior wall infarct with new left bundle branch block and hemodynamic instability – Alternating left and/or right bundle branch block – Right bundle branch block and left anterior hemiblock and/or left posterior hemiblock – Left bundle branch block and 1st degree AV block	– Alternating left and/or right bundle branch block – Symptomatic patient with right bundle branch block and left anterior hemiblock and/or left posterior hemiblock

^{*1} modified from Wellens (5)

Patients with sinus node dysfunction should be treated with a temporary pacemaker if a satisfactory hemodynamic status cannot be achieved with medical treatment (parasympatholytics) alone, or if the patient is still suffering from symptoms of bradycardia after such treatment (*Table 1*). Sinus bradycardia arises in about 12% of patients with acute myocardial infarction (5). Atrioventricular (AV) block is common in acute coronary syndromes; in the early stage of an acute infarction, AV block is promoted not just by myocardial ischemia and necrosis, but also by heightened vagal tone. Nonetheless, AV block generally does not necessitate temporary pacemaker stimulation. In the pre-hospital phase, temporary stimulation is needed if bradycardia remains symptomatic despite parasympatholytic treatment, if it triggers a ventricular arrhythmia, or if it worsens the patient's hemodynamic status (5).

AV block is a more serious matter in patients with anterior wall infarction, who should undergo temporary pacemaker stimulation as soon as pump dysfunction becomes clinically evident (*Table 2*). The

frequency of AV block in patients with inferoposterior infarction ranges from 12% to 20%; in patients with anterior wall infarction, it is roughly 5% (5).

Bundle branch block is not, in itself, a reason for temporary pacemaker stimulation in a patient suffering from an acute coronary syndrome. Nonetheless, patients with anterior wall infarction, newly manifest left bundle branch block, and hemodynamic instability should undergo placement of a temporary pacemaker. Pacemaker therapy is also indicated for patients with alternating bundle branch block (frequency <1%) (5).

Problem areas

As soon as a faulty pacemaker system (frequency, 1.1%) is suspected, the pacemaker and the cardiac electrode(s) should be meticulously checked and, if necessary, corrected (6). Faulty impulse detection—either oversensing (0.7%) or undersensing (3.8%)—and faulty stimulation because of electrode fracture (3.8%) are among the potentially dangerous complications (2). Oversensing is defined as the detection of extrinsic electrical interference in addition to cardiac impulses; undersensing is the non-detection of cardiac impulses.

Pathological stimulation leading to abnormal twitching of the diaphragm or other muscles (frequency: 0.4%) is usually reported spontaneously by the patient and is generally easy to diagnose. Electrode dislocation or defective insulation (3.4%) can often be diagnosed from the stimulation artefacts or pacemaker inhibition that may be induced by arm movements or postural changes. In 2008, electrode dysfunction was seen in only 0.6% of patients (6).

Early complications

The most serious complication arising very early after pacemaker implantation is ventricular perforation (frequency, ca. 1%) with pericardial tamponade. This is generally due to forcible advancement of a pacemaker electrode stiffened by a guidewire. A pericardial effusion requiring treatment arises after 0.1% of all pacemaker implantations (6). Some patients with myocardial perforation have an uneventful course and are discharged home a few days after implantation; perforation is only suspected some time afterward because of diaphragmatic twitching, a rise in the pacemaker threshold, and/or abnormal ECG or X-ray findings (*Figure 1*).

Problem areas

As soon as a faulty pacemaker system is suspected, the pacemaker and the cardiac electrode(s) should be meticulously checked and, if necessary, corrected.

Early complications

The most serious complication arising very early after pacemaker implantation is ventricular perforation with pericardial tamponade.

TABLE 2

Types of AV block in anterior and posterior wall infarction and their prognostic significance^{*1}

	Anterior wall infarction	Posterior wall infarction
Frequency	5%	12–20%
Site of block	Bundle branches	AV nodes
Affected vessel	Anterior interventricular branch	Right coronary artery
Escape rhythm	Wide QRS complex, heart rate <40/min	Narrow or wide QRS complex; heart rate 40–60/min
Duration of block	Temporary	Temporary
Mortality compared to same infarct without block	4-fold	2 1/2-fold

^{*1} modified from Wellens (5)

Other early complications that arise from electrode placement and require emergency treatment include arterial injury with hemothorax and pleural injury with pneumothorax (frequency, 0.4%). Electrode dislocation is one of the more common early complications of pacemaker implantation. It cannot be avoided in some cases and is seen particularly often in patients with a rarefied right ventricular trabecular system or with dilated ventricles. In experienced hands, the dislocation rate is less than 1% (6). Dislocation of the atrial electrode occurs more often in patients with implanted sequential pacemaker systems (2). It manifests itself clinically in pacemaker dysfunction, including faulty recognition and stimulation as well as elevation of the stimulation threshold.

Finally, infection of the pacemaker pouch and/or electrode system is a dreaded early complication, occurring at frequencies ranging from 1% to 12%. According to a recent annual report of the German Pacemaker Registry, infections were seen in 0.1% of patients perioperatively and led to operative revision in 5.8% (6). Early pacemaker infection, defined as an infection occurring within the first six months of implantation, manifests itself in overt signs of infection such as redness, swelling, and/or abscess formation at the pacemaker pouch site, along with fever, leukocytosis, and an elevated serum concentration of C-reactive protein. Florid infection should be treated by immediate surgical removal of the impulse generator and the electrode system.

Potential infections after pacemaker implantation

Infection after pacemaker implantation is often due to poor wound healing or pressure necrosis.

Late complications

Late pacemaker complications mainly involve dysfunction of the impulse generator and the electrode system. The hallmarks of such complications are absent stimulation (no stimulus artefact on ECG), lack of stimulation despite presence of a stimulus artefact, and/or faulty impulse recognition (*Figure 2*) (2). Late infection is also a possibility and is associated with elevated morbidity and mortality (6); it is often the result of impaired wound healing or pressure necrosis. Complications such as the pacemaker syndrome and pacemaker-induced tachycardia are relatively rare. They are due to interactions between the pacemaker and the patient's cardiac conducting system (5).

Recent developments

Pacemaker therapy has advanced remarkably over the past 50 years. One million pacemakers are now implanted all over the world every year, including about 70 000 new pacemakers in Germany alone (2006, 66 059; 2007, 67 105; 2008, 68 714). Multi-purpose devices with a wide range of diagnostic and therapeutic stimulating functions are now available. Preventive stimulation and preventive algorithms can now suppress atrial fibrillation: in the ADOPT trial, atrial overstimulation with an atrial fibrillation suppression algorithm reduced the time spent in atrial fibrillation (the "AF burden") by 25% (92.9% in the stimulation group compared to 67.9% in the control group, $p < 0.0001$) (7). The ability to store intracardiac ECGs of high quality has markedly expanded the diagnostic capabilities of pacemakers and now enables more individualized therapy, e.g., in patients with atrial fibrillation, extrasystoles, or anticoagulation. The adaptation of AV transmission is important from the hemodynamic and clinical point of view. Contemporary pacemakers are designed to enhance the dynamics of AV transmission; this is particularly important in patients who undertake vigorous physical activity, such as sports, who need AV time optimization for hemodynamic reasons (2). Biventricular stimulation algorithms for patients with heart failure are a further important development. MRI-compatible electrodes and impulse generators were recently introduced.

Indications for defibrillator therapy

In 2005, the German Cardiac Society (*Deutsche Gesellschaft für Kardiologie-, Herz- und Kreislaufforschung*) issued its guidelines on the implantation of automatic cardiac defibrillators (8). There is a class IA recommendation to implant an implantable

Late complications

Late pacemaker complications mainly involve dysfunction of the impulse generator and the electrode system.

cardioverter-defibrillator (ICD) for patients with the following conditions (Table 3):

- cardiac arrest due to ventricular tachycardia
- cardiac arrest due to ventricular fibrillation
- ventricular tachycardia with hemodynamic effects
- syncope without ECG documentation and with a left ventricular ejection fraction (LVEF) $\leq 40\%$.

Secondary prevention

The superiority of defibrillator therapy to medical treatment for secondary prevention was clearly demonstrated in the CIDS, CASH, and AVID trials (9–11).

In the CIDS trial, the annual overall cardiac mortality was 10.2% with amiodarone and 8.3% with an ICD, corresponding to a relative risk reduction of 20%, while the annual incidence of sudden cardiac death in the same two patient groups was 4.5% and 3.0%, respectively, corresponding to a relative risk reduction of 33% (9).

The overall mortality in the ICD arm of the CASH trial over a mean follow-up interval of 57 months (standard deviation, 34 months) was 36.4%, compared to 44.4% in the amiodarone/metoprolol arm (relative risk reduction, 23%) (10).

In the AVID trial, 89.3% of the patients in the ICD group survived 1 year, compared with 82.3% in the medical antiarrhythmic treatment group (relative risk reduction for overall mortality, 39.5%); at two years, the survival rates were 81.6% and 74.7% (relative risk reduction for overall mortality, 27%) (11).

Primary prevention

Patients with coronary heart disease

Prospective trials such as MADIT-I and MADIT-II have documented not just a lower frequency of sudden cardiac death, but also lower overall mortality after defibrillator implantation (12, 13). For patients who have sustained a myocardial infarction, the benefit of preventive ICD therapy is greater the longer the device has been implanted. In the MADIT-II trial, ICD therapy was associated with significantly higher survival over a mean follow-up duration of 20 months (hazard ratio [HR] 0.69, $p = 0.016$). The overall mortality was 14.2% in the ICD group and 19.8% in the control group (3). In the SCD-HeFT trial, involving patients with non-ischemic and ischemic cardiomyopathy who had an ejection fraction under 35%, amiodarone was compared to

Figure 1: This anteroposterior chest X-ray of a man with a double-chamber pacemaker reveals that an electrode has been damaged by “crush syndrome” at the insertion site near the clavicle, explaining the clinical signs of inadequate pacemaker stimulation

TABLE 3

Indications for ICD implantation^{*1}

Clinical manifestations	Recommendations	Evidence level
Cardiac arrest because of ventricular tachycardia or ventricular fibrillation	I	A
Ventricular tachycardia with hemodynamic effect	I	A
Syncope and EF $\leq 40\%$ without other causes	I	A
Myocardial infarction (>4 wks ago) and EF $\leq 30\%$	I	B
Heart failure (NYHA II/III) and EF $\leq 35\%$	I	B
Brugada syndrome, syncope of uncertain origin	I	C
Brugada syndrome, asymptomatic, with markers of elevated risk	IIa	C
Long QT syndrome with syncope on beta-blockers	IIa	C
Hypertrophic cardiomyopathy, ARVCN with risk markers	IIa	C
Short QT syndrome	IIa	C
Brugada syndrome, asymptomatic, without markers of elevated risk	IIb	C
DCM and EF $\leq 35\%$, duration >9 months	IIb	A

ARVCN, arrhythmogenic right-ventricular cardiomyopathy; NYHA, New York Heart Association; DCM, dilated cardiomyopathy; EF, left-ventricular ejection fraction. ^{*1} Modified from (3)

Secondary prevention

The superiority of defibrillator therapy to medical treatment for secondary prevention was clearly demonstrated in the CIDS, CASH, and AVID trials.

The German Cardiac Society gives a class IA recommendation to implant an ICD for patients with:

- Cardiac arrest due to ventricular tachycardia
- Cardiac arrest due to ventricular fibrillation
- Ventricular tachycardia with hemodynamic effects
- Syncope without ECG documentation and LVEF $\leq 40\%$

Figure 2:

Defective insulation of a pacemaker electrode: the electrode coating is totally destroyed

placebo and to defibrillator therapy over a mean follow-up duration of 46 months. There were far fewer deaths in the ICD group (22%) than in either the amiodarone group (28%) or the placebo group (29%) (ICD-associated relative risk reduction for death = 23%) (14).

The DINAMIT trial, in contrast, failed to reveal any improvement in outcome with ICD therapy (15). This trial included 674 patients who underwent ICD implantation 6 to 40 days after an acute myocardial infarction and who had a left ventricular ejection fraction under 35%. They were randomized into an ICD arm (332 patients) and an arm with best medical treatment (342 patients). After a mean follow-up duration of 30 months, there was no significant difference in overall mortality between the ICD patients (7.5% per year) and the medically treated patients (6.9% per year) (HR 1.08, $p = 0.66$). With respect to arrhythmia-related deaths, however, ICD was, in fact, associated with a significantly better outcome: 1.5% per year in the ICD group, compared to 3.5% per year in the medically treated group ($p = 0.009$) (15).

Patients with non-ischemic heart disease

There have been three large-scale, randomized, prospective trials of ICD therapy for primary prevention in patients with dilated cardiomyopathy: the CAT, AMIOVIRT, and DEFINITE trials (16–18). All three

included only patients with cardiomyopathy of non-ischemic origin.

The 104 patients with dilated cardiomyopathy who were included in the CAT trial had an ejection fraction under 30%, had been in cardiac failure for less than 9 months, and did not have any symptomatic ventricular arrhythmias. Although ICD therapy was found to be associated with a lower overall mortality than conventional medical treatment (26.0% versus 31.5%), this difference was not statistically significant.

The AMIOVIRT study included 103 patients with dilated cardiomyopathy, asymptomatic non-sustained ventricular tachycardia, and a left ventricular ejection fraction below 35%. There was no significant difference in survival between the ICD and amiodarone groups at one year (96% vs. 90%) or at three years (87% vs. 88%; $p = 0.8$).

The DEFINITE study included 458 patients who had dilated cardiomyopathy and an ejection fraction below 36% with either asymptomatic ventricular extrasystoles or non-sustained ventricular tachycardia. The overall mortality did not differ significantly in the ICD and control groups after a mean follow-up interval of 29.0 months (standard deviation, 14.4 months), even though the mortality at two years was almost twice as high in the control group (14.1% vs. 7.9%, $p = 0.08$). The trial did, however, show a significantly lower arrhythmia-related mortality among the ICD patients: the figure of

Primary prevention in coronary heart disease

Most of the prospective trials have shown lower overall mortality after defibrillator implantation.

Preventive ICD therapy

For patients who have had a myocardial infarction, the benefit of preventive ICD therapy is greater the longer the ICD has been implanted.

1.3% in the ICD group compares very favorably to 6.1% in the medically treated group (hazard ratio, 0.2; $p = 0.006$) (17).

Defibrillator therapy with cardiac resynchronization

The MUSTIC, PATH-CHF, and MIRACLE trials showed that cardiac resynchronization therapy (CRT) significantly improves cardiopulmonary reserve and also reduces left-ventricular volume within three to six months (19, 20). In the COMPANION trial, 1520 patients with heart failure of NYHA grade III or IV, a left ventricular ejection fraction below 35%, and a QRS width greater than 0.12 seconds were randomized to one of three treatment arms: optimized medical treatment alone, optimized medical treatment and a pacemaker for CRT, and optimized medical treatment and a CRT defibrillator (21). In the latter two arms, the electrical stimulation was biventricular. After twelve months of follow-up in each arm, the predefined primary endpoint (either death from any cause or hospitalization) was reached by 68% of patients in the medical treatment group and by 56% of patients in each of the CRT groups. Thus, CRT with either a pacemaker or a defibrillator reduced the risk of reaching the primary endpoint by nearly 20%, while the two CRT groups did not differ significantly in outcome with respect to the primary endpoint. The secondary endpoint (death from any cause) was attained by 25.0% of patients in the medical treatment group, 21.2% in the CRT pacemaker group, and 17.6% in the CRT defibrillator group. The difference between the CRT defibrillator group and the medical treatment group corresponded to a 36% lower risk of death and was statistically significant ($p = 0.003$).

In the CARE-HF trial, 813 patients with NYHA grade III or IV heart failure, a QRS width greater than 0.15 seconds, or a QRS width between 0.12 and 0.149 seconds combined with echocardiographic signs of dyssynchrony, were assigned to receive either CRT or medical treatment alone (22). After a mean follow-up duration of 29 months, the combined endpoint of death or hospitalization for heart failure was reached by 39% of patients in the CRT group and by 55% of patients in the medical treatment group, corresponding to a relative risk reduction of 37% ($p < 0.001$). The overall mortality of the CRT group (20%) was also lower than that of the medical treatment group (30%; risk reduction, 36%; $p > 0.002$).

Defibrillators for patients with non-ischemic heart disease

Defibrillator therapy lowered arrhythmia-related mortality in the DEFINITE study, but the difference in overall mortality was not statistically significant.

Figure 3: This anteroposterior chest X-ray of a woman with a triple-chamber pacemaker reveals one electrode each in the right atrium, the right ventricle, and the coronary sinus

In the ALTITUDE trial, the five-year survival rate of 47 032 patients with an ICD was 91.8%, while that of 38 967 patients with ICD-CRT was 75.6% (23). The delivery of a shock was associated with a worse outcome in both patient groups (hazard ratio 1.60, $p < 0.001$); the frequency of shocks in 5 years was 35.5% in the ICD group and 34.5% in the ICD-CRT group.

In the MADIT-CRT trial, which involved 1820 patients, it was found that CRT lowered the frequency of events related to heart failure by 41% in patients with a milder degree of heart failure (NYHA stages I and II) who had poor left ventricular pumping function (ejection fraction $< 30\%$) (24). This finding was statistically significant. Over a mean follow-up duration of 2.4 years, the primary endpoint was reached by 17.2% of patients in the CRT group and by 25.3% in the control group ($p = 0.001$) (Figure 3).

Postoperative complications

Complications such as postoperative hemorrhage, thrombosis, migration and other problems related to

Defibrillator therapy with cardiac resynchronization

Clinical trials have shown that cardiac resynchronization therapy (CRT) significantly improves cardiopulmonary reserve.

the ICD impulse generator (frequency, 6%) and the electrodes, pneumothorax, and infection are well known. Antibiotic prophylaxis is recommended immediately before ICD implantation and for 24 hours thereafter, although the need to do so has not been demonstrated in any study. The particular antibiotic used should be chosen in view of the local spectrum of pathogens and resistances. ICD treatment can also be complicated by electrode-related problems (frequency, 12%), such as insulation defects, cable fracture due to material fatigue, and inadequate shock delivery (frequency, 12%) because of oversensing or tachycardic atrial fibrillation. In addition to these technical complications, there can also be psychosomatic problems—often caused by frequent and/or inadequate shocks—such as depression or even psychosis (frequency less than 1%). Anxiety disorders are rare in patients with implantable defibrillators and are usually brought on by fear of an ICD discharge. To prevent anxiety, the physician should discuss the indications for ICD therapy with the patient and address any possible emotional issues in the outpatient arrhythmia clinic and/or a patient self-help group. Add-on treatment with amiodarone may elevate the defibrillation threshold, leading to ineffective defibrillation; thus, patients who begin treatment with amiodarone while under ICD therapy should have their defibrillation threshold redetermined. The important question whether an ICD patient can be allowed to drive a car was recently addressed in a position paper of the German Cardiac Society (25).

Conflict of interest statement

The authors declare that they have no conflicts of interest.

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Antibiotics for ICD implantation

Antibiotic prophylaxis is recommended immediately before ICD implantation and for 24 hours thereafter.

Anxiety disorder in patients about to undergo defibrillator implantation

Anxiety disorders are rare in patients with defibrillators and are usually brought on by fear of an ICD discharge.

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Please answer the following questions to participate in our certified Continuing Medical Education program. Only one answer is possible per question. Please select the most appropriate answer.

Question 1

How many persons have a cardiac arrest in Germany each year?

- a) about 40 000
- b) about 60 000
- c) about 80 000
- d) about 100 000
- e) about 120 000

Question 2

Bradycardia with an insufficient intrinsic ventricular rate gives rise to symptoms that require treatment.

Which of the following symptoms are typically seen in this situation?

- a) Night sweats and insomnia
- b) Dizziness and syncope
- c) Fatigue and vomiting
- d) Shortness of breath and ischemia
- e) Chronic dermatitis and hypertension

Question 3

When is a permanent pacemaker indicated for a patient with sick sinus syndrome?

- a) When sinus node dysfunction is present with characteristic clinical manifestations
- b) When the patient does not respond to treatment with atropine
- c) When no adequate escape rhythm is present
- d) When a temporary hemodynamic worsening is diagnosed
- e) When second-degree AV block of Wenckebach type is present

Question 4

What is typically the most serious early complication of pacemaker insertion?

- a) Faulty impulse recognition
- b) Stimulus artefact
- c) Ventricular perforation
- d) Hemodynamic instability
- e) Right bundle branch block

Question 5

Automatic defibrillator implantation is indicated for patients who have sustained a cardiac arrest because of atrial fibrillation.

What is the level of evidence for this indication, according to the guidelines of the German Cardiac Society?

- a) IA
- b) IB
- c) IC
- d) IIaC
- e) IIbA

Question 6

Defibrillation for cardiac resynchronization is associated with a lower risk than medical therapy. According to current studies, how much lower is the relative risk for the combined endpoint of death (from any cause) or hospitalization?

- a) <2 percent lower
- b) 1 to 5 percent lower
- c) 6 to 10 percent lower
- d) 11 to 15 percent lower
- e) >19 percent lower

Question 7

Six weeks after pacemaker implantation, the patient comes to the cardiologist's office complaining of abnormal muscle twitches that arise whenever he changes his position in bed or raises his left arm.

What is the most likely diagnosis?

- a) Myasthenia gravis
- b) Parkinson's dementia
- c) Defective isolation of a pacemaker electrode
- d) Excited state due to hyperthyroidism
- e) Overdose of tricyclic antidepressants

Question 8

What type of medication should be given immediately before a defibrillator is implanted and for up to 24 hours afterwards?

- a) Beta-blockers
- b) Antibiotics
- c) Diuretics
- d) ACE inhibitors
- e) Sedatives

Question 9

What type of problem in a pacemaker system is called oversensing?

- a) Lack of sensing of electrical signals
- b) Transmission of electrical signals
- c) Algorithm-induced signal shifting
- d) Delayed impulses in electrical signals
- e) Interference by external electrical signals

Question 10

A patient is found to have a posterior wall infarct with atrioventricular block. The ECG shows a wide QES complex. The heart rate is 55 beats/min. Which vessel is almost certainly affected?

- a) The circumflex branch
- b) The left coronary artery
- c) The anterior interventricular branch
- d) The atrial branch
- e) The right coronary artery