

## Automated Implantable Cardioverter Defibrillators

BY SUSAN KAPRELIAN, MD; FRANCOIS HADDAD, MD; ANDRÉ DENAULT, MD;  
MARC DUBUC, MD; AND PIERRE COUTURE, MD.

Automated implantable cardioverter defibrillators (ICDs) were introduced in the early 1980s after a report by Mirowski et al described the first successful defibrillation with an ICD in humans.<sup>1</sup> Since then, multiple randomized clinical trials have proven the efficacy and superiority of ICD therapy for both the secondary and primary prevention of sudden cardiac death.<sup>2-6</sup> Because the clinical indications for these devices are expanding, more patients with an ICD are likely to undergo surgical or obstetrical procedures. This issue of *Anesthesiology Rounds* reviews the history, structure, function, and clinical indications for ICD technology, including a discussion of anesthetic management strategies.

### HISTORY

ICDs were originally developed to prevent sudden cardiac death (SCD) in patients who had already experienced life-threatening ventricular arrhythmias, such as ventricular tachycardia/ventricular fibrillation (VT/VF). The early model generators were large (160-190 cm<sup>3</sup>) and heavy (>200 g), necessitating abdominal wall implantation under general anesthesia. The energy output was low and often nonprogrammable; in addition, the batteries had limited energy (~150 shocks). A sternotomy or lateral thoracotomy was needed to expose the heart for electrode placement (epicardial leads).<sup>6</sup> Moreover, the first models did not have the capacity to pace. Now, ICDs are much smaller, multiprogrammable anti-arrhythmia devices that are capable of treating bradycardia, VT/VF and, in some models, atrial tachycardia. The new devices can even incorporate biventricular pacing with the objective of treating cardiac dyssynchrony.

### COMPONENTS OF AN ICD SYSTEM

The implanted system consists of an ICD generator, pacing and sensing leads (or electrodes), and  $\geq 1$  high energy leads for defibrillation.<sup>7</sup>

**The generator:** The size of the new generators is only one-third that of older models. They can be implanted subcutaneously in the prepectoral region. They have transtelephonic programmability, improved tachycardia discrimination algorithms, anti-tachycardia and bradycardia pacing, and biphasic waveform shocks. The ICD generator casing is made of titanium, which serves to protect the circuitry and often acts as an active shocking electrode. The batteries and capacitors are contained inside the casing.

The most commonly used **battery** is a lithium silver vanadium oxide cell that contains ~18,000 joules (J) of energy. Each ICD generator uses two 3.2 Volt (V)-batteries, configured in series, for an initial full voltage of 6.4 V. Some models utilize a third battery for all non-shock activities in order to prolong the functional life of the device, particularly, when a significant amount of bradycardia pacing is provided. The measured voltage generally determines the battery status. Elective replacement is normally at 2.6 V, while a value of 2.2 V indicates that urgent replacement is needed; however, actual voltage values may vary with the model.

The **capacitors** are used to store energy (up to 30-40 J) from the batteries (6.4 V) and deliver it at a high voltage (up to 750 V) within 10-20 milliseconds (msec), because the battery itself cannot deliver high enough energy for defibrillation. Aluminium electrolytic capacitors have been used because of their ability to charge within the prescribed time limitation (20-30 seconds).

**Electrodes or leads:** The ICD detects ventricular rhythm via sensing electrodes and delivers therapy to the heart via defibrillation electrodes. Detection is accomplished with bipolar electrodes that are also used for pacing. These electrodes are usually positioned transvenously at the right ventricular apex in the endocardium. They are rarely placed on the epicardium, except in open-heart surgery. In the case of dual-chamber ICDs, the pacemaker has

Committee for Continuing  
Medical Education  
Department of Anesthesiology  
University of Montreal

Pierre Drolet, MD  
Chairman and co-editor  
Maisonneuve-Rosemont Hospital

Jean-François Hardy, MD  
Co-editor and Chairman of the  
Department of Anesthesiology,  
University of Montreal

François Donati, MD, Co-editor  
Maisonneuve-Rosemont Hospital

Edith Villeneuve, MD  
Ste-Justine Hospital

Robert Blain, MD  
Montreal Heart Institute

Anna Fabrizi, MD  
CHUM

Robert Thivierge, MD  
Vice-Dean  
Continuing Education  
University of Montreal

University of Montreal  
Department of Anesthesiology  
Faculty of Medicine

Université   
de Montréal  
Faculty of Medicine  
Department of Anesthesiology

The editorial content of *Anesthesiology Rounds* is determined solely by the Department of Anesthesiology of the University of Montreal Faculty of Medicine

This issue and CME questionnaire  
are available on the Internet  
[www.anesthesiologyrounds.ca](http://www.anesthesiologyrounds.ca)

an additional sensing and pacing electrode placed in the right atrium. Defibrillation electrodes previously required a thoracotomy to implant epicardial patches. In modern ICDs, the generator casing itself serves as the shocking electrodes. This system is called an “active can” and requires the generator to be implanted in the pectoral region. Current flows from the distal defibrillation coil electrode, positioned in the right ventricular apex, to the generator itself. In the case of a double coil electrode, current flows from the distal coil to a more proximal coil in the superior vena cava. The advantage of an “active can” is a reduced defibrillation threshold.

## FUNCTION OF THE ICD

The most important function of an ICD is recognition of ventricular tachyarrhythmias and providing treatment (antitachycardia pacing, cardioversion, or defibrillation) upon recognition. An ICD can prevent ventricular tachycardia using anti-bradycardia pacing in specific conditions (eg, the long-QT syndrome).<sup>7,8</sup>

The detection of ventricular arrhythmia relies on transmission of the sensed electrical activity to the generator, via the ventricular electrode. The quality of the signal is determined during lead placement in the electrophysiology (EPS) laboratory and can be reassessed via the programmer. The next step involves detection algorithms. The most important determinant of ventricular arrhythmia detection algorithms is the heart rate or the interval between each ventricular depolarization. Once an electrocardiogram (ECG) event is detected and transmitted to the generator, different modalities of therapy are selected, depending on the arrhythmia detection zone.

**Antiarrhythmia therapy:** In order to derive the maximum efficacy and safety from an ICD, the electrophysiologist has to program appropriate therapies for each arrhythmia detection zone and establish a hierarchy of therapies, choosing from anti-tachycardia pacing, cardioversion, defibrillation, and anti-bradycardia pacing. Table 1 outlines an example of the hierarchy of tiered therapy based on beats per minute (BPM).<sup>9</sup>

**Anti-tachycardia pacing (ATP)** is available in all recent ICDs and is usually the indication for ICD implantation. The goal of this therapy is to stimulate the ventricle to work at a faster rate than the frequency of the detected ventricular rate, thus abolishing the re-entry mechanism and terminating the arrhythmia. It is effective in terminating sustained ventricular tachycardia in approximately 90% of cases.<sup>10</sup> The disadvantage of ATP therapy is that the accelerated rate of the ventricle may not be well-tolerated hemodynamically in some patients.

**Low-energy cardioversion** protocols are usually programmed before defibrillation protocols and after ATP protocols. Compared to defibrillation, cardioversion uses low-energy shocks – usually 1 to 2 J initially – that reduce battery consumption and are less painful, although some patients still complain of significant pain.

**Defibrillation** can be performed safely in most patients using 26 to 36 J of energy. Only a small percentage require a second defibrillation. The minimum energy needed to defibrillate is called the “defibrillation threshold (DFT),” which is determined in the EPS laboratory. To determine the DFT, the electrophysiologist first induces VF, confirms adequate sensing, and delivers an internal shock. If defibrillation is successful, the same

**TABLE 1: Hierarchy of tiered therapy.**

Zone	Brady-cardia	Normal	VT	FVT	VF
Rate (BPM)	<50	50-150	150-169	170-199	>200
First device therapy	Anti-bradycardia pacing	None	ATP or LECV	ATP	DF

VT = ventricular tachycardia, FVT = fast ventricular tachycardia, VF = ventricular fibrillation, ATP = antitachycardia pacing, LECV = low energy cardioversion, DF = defibrillation.

procedure is repeated every 5 minutes, with decreasing energy until no defibrillation is occurring. The lowest energy required to defibrillate is the DFT, with an additional 10 J added as a safety measure. The DFT depends on several factors, including the integrity and position of the ICD system; metabolic factors (eg, hyperkalemia, severe acidosis or alkalosis, hypercapnia, hypoxia, myxedema, or severe hyperglycemia can increase thresholds); structural cardiac factors (eg, inflammation, myocardial infarction [MI]); and drug therapy (eg, class Ic anti-arrhythmic agents can increase defibrillation thresholds).<sup>11</sup> An increasing number of centres no longer use DFT and have replaced this method with the concept of a 10 J safety margin. Using this technique, a baseline test of defibrillation at an output at least 10 J less than the maximum is performed. If successful, the ICD is then programmed to defibrillate at the maximum level.

**Anti-bradycardia** pacing has been incorporated into the most recent ICDs that have the pacemaker function. Assistance during the post-shock period is also provided to help hemodynamic recovery. The more recent ICDs have dual-chamber devices incorporated into them (DDD/DDDR). The advantage of DDD/DDDR pacing is a potential hemodynamic improvement by synchronous auriculo-ventricular (AV) pacing. Dual-chamber sensing systems can also improve discrimination of atrial tachyarrhythmias from ventricular arrhythmias.

## INDICATIONS FOR ICD THERAPY

The indications for ICD placement are continuously evolving and modifications of current recommendations can be expected in the future. In 2002, the American College of Cardiology/American Heart Association/North American Society of Pacing and Electrophysiology (ACC/AHA/NASPE) published their clinical guidelines.<sup>12</sup> Their recommendations are divided into classes depending on the weight of supporting evidence:

**Class I:** Conditions for which there is evidence and/or general agreement that a given procedure or treatment is beneficial, useful, and effective.

**Class II:** Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment.

- **Class IIa:** Weight of evidence/opinion is in favour of usefulness/efficacy.

- **Class IIb:** Usefulness/efficacy is less well-established by evidence/opinion, some patients can benefit from the therapy.

**Class III:** Conditions for which there is evidence and/or general agreement that a procedure/treatment is not useful/effective and in some cases may be harmful.

## SPECIFIC CONDITIONS

Listed below are common diagnoses that are divided into the ACC/AHA/NASPE classes for use of ICD therapy.

### **Cardiac arrest**

- Cardiac arrest due to VF (including idiopathic VF) or VT not due to a transient or reversible cause (ACC/AHA class I).
- Cardiac arrest is presumed due to VF when electrophysiological testing is precluded by other medical conditions. (ACC/AHA class IIb).

### **Sustained ventricular tachycardia (VT)**

- Spontaneous sustained VT associated with structural heart disease (ACC/AHA class I).
- Spontaneous sustained VT in patients without structural heart disease, not amenable to other treatments (ACC/AHA class I).

### **Syncope**

- Syncope of undetermined origin with clinically relevant, hemodynamically significant sustained VT or VF induced at electrophysiological study when drug therapy is ineffective, not tolerated, or not preferred (ACC/AHA class I).
- Recurrent syncope of undetermined origin in the presence of ventricular dysfunction and inducible ventricular arrhythmias at electrophysiological study when other causes of syncope have been excluded (ACC/AHA class IIb).
- Syncope in patients with advanced structural heart disease, where thorough invasive and non-invasive investigations have failed to define a cause (ACC/AHA class IIb).

**Coronary artery disease (CAD):** ICD implantation led to a reduction in mortality at 4 years in patients with left ventricular ejection fraction (LVEF)  $\leq 30\%$  at least 1 month post-MI and 3 months post-coronary artery revascularization surgery (ACC/AHA class IIa).<sup>5,13</sup> Non-sustained VT in patients with coronary disease, prior MI, left ventricular (LV) dysfunction, and inducible VF or sustained VT at EPS study, not suppressible by class I antiarrhythmic drugs, are classified as ACC/AHA class I, or without reference to inducibility to ACC/AHA class IIb. In practical terms, a patient with non-sustained VT and an ejection fraction (EF)  $>30\%$ - $35\%$  and  $<40\%$ , is examined by EPS to determine inducibility. If it is positive, an ICD is indicated irrespective of the suppressibility by class I anti-arrhythmic agents. In a patient with an EF  $>40\%$ , medical therapy should be optimized and reversible causes corrected.

**Idiopathic dilated cardiomyopathy:** The degree of LV systolic dysfunction is the best “marker” for the risk of sudden cardiac death. With the results of the Sudden Cardiac Death-Heart Trial (SCD-HeFT),<sup>6</sup> an ICD is now considered in patients with New York Heart Association (NYHA) functional class II or III congestive heart failure who are diagnosed at  $\geq 9$  months and with an EF  $\leq 30\%$ .

**Hypertrophic cardiomyopathy** is a genetic disorder of the cardiac sarcomere, which puts the patient at risk of sudden cardiac death. The major risk factors include cardiac arrest (ventricular fibrillation), spontaneous sustained ventricular tachycardia, and a familial history of sudden death (sudden cardiac death in first-degree relatives aged  $<40$  years of age).<sup>14</sup> The minor risk factors include  $\geq 2$  unexplained syncope episodes within a year,

LV hypertrophy with wall thickness  $>30$  mm, abnormal blood pressure on exercise, non-sustained VT, specific mutations, microvascular obstruction, and LV outflow obstruction (a less-important factor).

**Arrhythmogenic right ventricular dysplasia (ARVD)** is an autosomal dominant disorder with variable penetrance of unknown cause. This disorder is characterized by the patchy replacement of RV myocardium by fibrous fatty tissue that provides a substrate for re-entrant ventricular arrhythmias.<sup>15</sup> As a result, these patients are in a high-risk group for ventricular arrhythmias and sudden cardiac death. In patients with drug-refractory arrhythmias, an ICD provides prophylaxis against syncope and sudden cardiac death.

**Brugada syndrome:** On ECG, the findings of right bundle branch block (RBBB) and ST elevation in leads V1-V3 are associated with the Brugada syndrome. When this finding is associated with symptoms (eg, syncope or a family history of sudden death), an ICD is usually recommended (ACC/AHA class IIb) for these patients.<sup>16</sup>

**The long QT syndrome** is a disorder of sodium or potassium channels that can be transmitted in an autosomal dominant or recessive fashion. An ICD is recommended in high-risk patients, such as those with a history of syncope, a familial history of sudden death, QTc  $>600$  ms, T-wave alternans, and the post-partum period.

**Bridge to cardiac transplantation:** An ICD is associated with a decreased risk of sudden death in patients with severe symptoms attributable to ventricular tachyarrhythmias awaiting cardiac transplantation (ACC/AHA class IIb).

**Pediatric patients:** Indications in the pediatric population are the same as in adults. Important considerations about physical growth and the psychological well-being of the child have to be considered.

Various contraindications for ICD therapy are listed in Table 2 .

## IMPLANTATION TECHNIQUE

The current generation of ICD leads are placed transvenously, via the subclavian, axillary, or cephalic vein. Previously, pulse generators were placed in the

**TABLE 2: Contraindications for ICD therapy**

- Syncope of undetermined cause, patient has no inducible ventricular tachyarrhythmias and no structural heart disease.
- Incessant VT or VF.
- VF or VT resulting from arrhythmias amenable to surgical or catheter ablation (eg, atrial arrhythmias associated with the Wolff-Parkinson White syndrome, RV outflow tract VT, idiopathic LV tachycardia).
- Ventricular tachyarrhythmias due to a transient or reversible disorder.
- Significant psychiatric illnesses.
- Terminal illness with projected life expectancy  $<6$  months.
- CAD with LV dysfunction and prolonged QRS duration in the absence of spontaneous or inducible sustained or non-sustained VT in patients undergoing coronary bypass surgery.
- NYHA Class IV drug-refractory congestive heart failure in patients who are not candidates for cardiac transplantation.

abdominal region, but modern devices are small enough to be implanted in the pectoral region, either subcutaneously or submuscularly, with a preference for the left side. With the recent compact models, conscious sedation and deep sedation during DFT-testing are the preferred methods of anesthesia. One advantage for avoiding general anesthesia is that many drugs, that could alter the defibrillation threshold (eg, halothane, isoflurane, and fentanyl), are not administered.<sup>17</sup>

In addition to standard recommended monitoring, invasive arterial blood pressure measurement is prudent in patients who have very severely depressed cardiac function. Cerebral satometry is useful and increasingly available in specialized cardiac centres. This is a noninvasive method for measuring cerebral oxygen saturation and its principle of operation is similar to that of pulse oximetry.<sup>18</sup> As a result, cerebral oxygen saturation and hemodynamics can be assessed noninvasively and continuously (Figure 1).

## COMPLICATIONS ASSOCIATED WITH ICDs

### Early complications

Perioperative mortality, which was as high as 5% using the thoracotomy method, has fallen to <1%.<sup>13,19</sup> Other perioperative complications include bleeding from vascular access, hematoma formation at the subcutaneous site and, if subclavian access is used, a small risk of pneumothorax. RV perforation is another rare complication during lead placement. Cardiac events due to defibrillation during implantation are also possible. Cerebrovascular events may occur, since repeated induction of VF during DFT-testing may result in transient cerebral hypoperfusion caused by reduced cardiac output.<sup>20</sup>

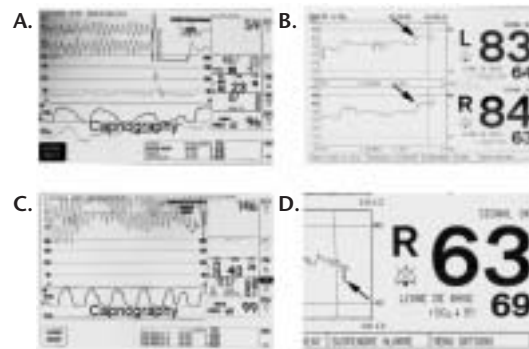
### Delayed complications

The infection rate is 1%-3%; the most common organisms are Gram-positive staphylococci (eg, *Staphylococcus epidermidis* and *Staphylococcus aureus*).<sup>18-21</sup> Lead-related problems are common and include infection, lead failure, lead fracture, dislocation, and vein thromboses, the latter being the most dangerous for patients.

### Functional complications related to ICDs

- Multiple defibrillator shocks can be seen in the context of ICD therapies in the presence of ventricular tachyarrhythmias related to electrical storms, high defibrillation thresholds, lead-related complications (eg, fracture, dislodgment), or inappropriately low energy shocks. Inappropriate shocks can also be seen in the presence of an oversensed signal from either supraventricular rhythms, a separate pacing system, electromagnetic interference, or from intracardiac signals.
- Failure to deliver an appropriate therapy for VT can be caused by a VT rate that is slower than the detection rate, undersensing of the electrical activity caused by lead-related complications (eg, fracture, dislodgment), or battery depletion.

**FIGURE 1: Brain oximetry (Invos 4100, Somanetic) during the implantation of a pace-defibrillator.**



(A) A 63-year-old man is scheduled for the insertion of a pace-defibrillator. During testing, the respiratory rate is reduced on the capnography signal following the addition of propofol.

(B) This was associated with a gradual increase in bilateral brain saturation from a baseline value of 64% and 63% on the left and the right to 83% and 84% (arrows) When ventricular fibrillation (reduction after vertical line) was induced a small decrease in brain saturation was observed.

(C) A 33-year-old man showed increased respiratory rate rapidly despite similar sedation.

(D) When ventricular fibrillation was induced, a decrease in brain saturation (after vertical line) was observed below baseline value.

These observations could be explained by the increase in PCO<sub>2</sub> with reduced ventilation. Hypercapnia increases cerebral blood flow and could attenuate the obligatory reduction in brain perfusion during ventricular fibrillation.

- ICD-induced pro-arrhythmias can occur, especially with the degeneration or acceleration of VT after antitachycardia pacing or low-energy cardioversion. Pro-arrhythmias can also be caused by post-shock bradycardia, a post-shock increase in pacing threshold resulting in failure to capture, and pacemaker-induced tachycardia in dual chamber ICDs.
- There are different sources of electromagnetic fields in every-day living and in hospitals that can cause ICD malfunction. This can manifest as either inhibition of pacing, asynchronous pacing, inappropriate tachytherapy, or inhibition of tachytherapy. Table 3 summarizes the main concerns.
- Magnet behaviour in an ICD depends on the manufacturer. Previously, ICDs from different companies had different responses to magnet placement. The response of current ICDs tends to be more simplified. Generally, when a magnet is placed over the generator, the tachyarrhythmias and defibrillation therapy are de-activated, and pacemaker function is not affected.

## ANESTHETIC MANAGEMENT

**Preoperative evaluation:** The general status of the patient is of primary importance. Underlying cardiac disease, whether it is CAD, congenital heart disease, or valvular heart disease, should be evaluated by a thorough medical history and appropriate physical examination. Associated systemic disease

**TABLE 3: Concerns and recommendations regarding ICDs and sources of electromagnetic interference**

Device	Concerns	Recommendations
<b>SOURCES IN DAILY LIFE</b>		
Household appliances	None	None
Cell phones	Limited data. Studies show no interference.	Keep phone >15 cm from ICD. <sup>22</sup> Do not use during ICD programming.
Security systems (airports, shopping centres)	None if exposure is limited. <sup>23</sup>	Inform personnel about metal detection.
Electrical equipment and tools.	Insulation and grounding should be insured.	Wear gloves. Insure adequate grounding.
<b>HOSPITAL SOURCES</b>		
Diagnostic radiology (including computed tomography [CT] scan)	None.	None.
Laser surgery	Probably safe.	Deactivation of ICD.
Surgical electrocautery	Device malfunction, reset, permanent damage to device, myocardial damage	Deactivation of ICD. Short, intermittent cautery bursts. Bipolar electrocautery if possible. If unipolar electrocautery is used, position plate far from device/leads. <sup>24</sup>
External cardioversion/defibrillation	Device malfunction, reset, permanent damage to device, myocardial damage	Position paddles far (>15 cm) from ICD, and perpendicular to it. Use standard resuscitation procedures. Confirm ICD function after procedure.
Transcutaneous electrical nerve stimulation (TENS)	Direct current interference	Contraindicated.
Electromyography, nerve conduction velocity tests.	Direct current interference	Contraindicated.
Radiofrequency (RF) ablation	ICD malfunction.	Deactivate device. Avoid contact with ICD. Confirm function after.
Therapeutic radiation	ICD malfunction.	Shield device. Consider changing device position (eg breast cancer). <sup>25</sup>
Diathermy	ICD damage from heat.	Deactivate ICD. Avoid direct contact.
Lithotripsy	Mechanical damage.	Deactivate ICD. <sup>26</sup>
Magnetic resonance imaging (MRI)	ICD malfunction. MRI malfunction.	Contraindicated. <sup>27</sup>
Direct current (DC ablation)	ICD malfunction.	Contraindicated.

should be noted and, if optimization is possible, it should be done specifically for elective procedures.

For elective procedures, a cardiology consultation should be sought to clarify the initial indications for ICD implantation, the date of implantation, whether symptoms have improved since implantation, and whether complications related to the device have occurred. Information should also be sought about the last device interrogation, such as battery status, lead performance, and adequacy of current settings. It is very important that instructions about de-activating the device be provided.<sup>28</sup>

For emergency surgery, if a cardiology consult is not immediately available, the device identification card should be reviewed, if possible. This card contains valuable information, such as the manufacturer's phone number, the name of the cardiologist, and the clinic where the device was implanted.

**Preoperative investigations:** No special laboratory test or chest radiographs are needed for the patient with an ICD. Investigations should be done accord-

ing to the patient's general status and coexisting diseases.

**Intraoperative management:** Although there is no special monitoring or anesthetic technique required for the patient with an ICD, basic ECG, hemoglobin oxygen saturation, and blood pressure measurement should be continuously monitored. ICD function should be turned off at all times by placing a magnet over the generator. This will avoid the delivery of shocks to the patient when tachyarrhythmias (caused by the interference of electromagnetic fields, eg, cautery) are sensed. External defibrillation pads should be placed and ready to use in case of a tachyarrhythmia.<sup>29-31</sup>

**Postoperative management:** Postoperatively, ICD function must be re-activated by a programming unit or removal of the magnet, if used. If there are concerns about major electromagnetic field interference and/or if external cardioversion or defibrillation were used during the procedure, the integrity and function of the device should be evaluated by the cardiologist.

## SUMMARY

It is apparent that with the appearance of more sophisticated devices and increasing indications for ICD implantation, anesthesiologists will have more frequent contact with patients with ICDs. The assessment and management of these patients require special attention and an understanding of the ICD system, indications, and complications (eg, electromagnetic interference [EMI]) in the operating room setting are essential for safe anesthesia practice. If a cardiologist is not immediately available, information drawn from the patient's device identification card that carries cardiologist, institution, and manufacturer information, can be useful.

---

**Dr. Kaprelian** is an anesthesiologist at St-Luc Hospital of the Centre Hospitalier de l'Université de Montréal (CHUM); **Dr. Haaddad** is a cardiologist at the Montreal Heart Institute; **Dr. André Denault** is an anesthesiologist at the Montreal Heart Institute and intensive care specialist at Hôtel-Dieu of CHUM; **Dr. Dubuc** is a cardiologist at the Montreal Heart Institute; **Dr. Pierre Couture** is an anesthesiologist at the Montreal Heart Institute.

---

## References

1. Mirowski M, Reid PR, Mower MM, et al. Termination of malignant ventricular arrhythmias with an automatic defibrillator in human beings. *N Engl J Med* 1980;303:322-324.
2. Connolly SJ, Hallstrom AP, Cappato R, et al. 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;21:2071-2078.
3. Buxton AE, Lee KL, Fisher JD, et al. A randomized study of the prevention of sudden death in patients with coronary artery disease. Multicenter Unsustained Tachycardia Trial Investigators. *N Engl J Med* 1999;341:1882-1890.
4. Mushlin AI, Hall WJ, Zwanziger J, et al. The cost-effectiveness of automatic implantable cardiac defibrillators: results from MADIT. Multicenter Automatic Defibrillator Implantation Trial. *Circulation* 1998;97:2129-2135.
5. Greenberg H, Case RB, Moss AJ, et al. Analysis of mortality events in the Multicenter Automatic Defibrillator Implantation Trial (MADIT-II). *J Am Coll Cardiol* 2004;43:1459-65.
6. Bardy GH, Lee KL, Mark DB, et al; Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT) Investigators. Amiodarone or an implantable cardioverter-defibrillator for congestive heart failure. *N Engl J Med* 2005;352(3):285-287.
7. Niebauer MJ, Wikoff BL. Implantable Cardioverter-Defibrillators: Technical Aspects, Cardiac Electrophysiology. In: Zipes DP, Jalife J, Eds. *From Cell To Bedside*, 3rd Edition. Philadelphia: WB Saunders; 2000:949-957.
8. Block M, Breithardt G. Implantable Cardioverter-Defibrillators: Clinical Aspects, Cardiac Electrophysiology. In: Zipes DP, Jalife J, Eds. *From Cell To Bedside*, 3rd Edition. Philadelphia: WB Saunders; 2000:957-970.
9. Lee KL, Lau CP. Implantable cardiac defibrillators. In: Crawford MH, DiMarco JP, Paulus WJ, Eds. *Cardiology*. 2nd edition. Oxford, England: Mosby; 2004:805-819.
10. Nasir N Jr, Pacifico A, Doyle TK, et al. Spontaneous ventricular tachycardia treated by antitachycardia pacing. *Am J Cardiol* 1997;79:820-822.
11. Barold S, Zipes DP. Cardiac Pacemakers and Antiarrhythmic Devices. In: Braunwald E, Ed. *Heart Disease*. 5th edition. Philadelphia: WB Saunders; 1997:705-741.
12. Gregoratos G, Abrams J, Epstein AE, et al. ACC/AHA/NASPE 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmic Devices: Summary Article. A report of the American College of Cardiology/American Heart Association task force on practice guidelines. *Circulation* 2002;106:2145-2161.
13. Moss AJ, Zareba W, Hall WJ, et al. Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. *N Engl J Med* 2002;346:877-883.

14. Nishimura RA, Holmes DR Jr. Hypertrophic obstructive cardiomyopathy. *N Engl J Med* 2004;350:1320-1327.
15. Gemayel C, Pelliccia A, Thompson PD. Arrhythmogenic right ventricular cardiomyopathy. *J Am Coll Cardiol* 2001;38:1773-1781.
16. Brugada P, Brugada R, Brugada J, Geelen P. Use of the prophylactic implantable cardioverter defibrillator for patients with normal hearts. *Am J Cardiol* 1999;83:98-100.
17. Weinbroum AA, Glick A, Copperman Y, Yashar T, Rudick V, Flaishon R. Halothane, isoflurane, and fentanyl increase the minimally effective defibrillation threshold of an implantable cardioverter defibrillator: First report in humans. *Anesth Analg* 2002;95:1147-1153.
18. McCormick PW, Stewart M, Goetting MG, Dujovny M, Lewis G, Ausman JI. Noninvasive cerebral optical spectroscopy for monitoring cerebral oxygen delivery and hemodynamics. *Crit Care Med* 1991;19:89-97.
19. Kron J, Herre J, Renfro EG, et al. Lead- and device-related complications in the antiarrhythmics versus implantable defibrillator trial. *Am Heart J* 2001;141:92-98.
20. Murkin JM, Baird DL, Martzke JS, Yee R. Cognitive dysfunction after ventricular fibrillation during implantable cardioverter/defibrillator procedures is related to duration of the reperfusion interval. *Anesth Analg* 1997;84:1186-1192.
21. Rosenqvist M, Beyer T, Block M, et al, on behalf of the European 7219 Jewel ICD Investigators. Adverse events with transvenous implantable cardioverter-defibrillators: A prospective multicenter study. *Circulation* 1998;98:663-670.
22. Fetter JG, Ivans V, Benditt DG. Digital cellular telephone interaction with implantable cardioverter-defibrillators. *J Am Coll Cardiol* 1998;31:623.
23. Pinski SL, Trohman RG. Interference in implanted cardiac devices, part I. *Pacing Clin Electrophysiol* 2002;25:1367-1381.
24. Levine PA, Balady GJ, Lazar HL, Belott PH, Roberts AJ. Electrocautery and pacemakers: management of the paced patient subject to electrocautery. *Ann Thorac Surg* 1986;41:313-317.
25. Venselaar JL, Van Kerloerle HL, Vet AJ. Radiation damage to pacemakers from radiotherapy. *Pacing Clin Electrophysiol* 1987;10:538-542.
26. Stroom SB. Contemporary clinical practice of shock wave lithotripsy: A reevaluation of contraindications. *J Urol* 1997;157:1197-1203.
27. Sommer T, Vahlhaus C, Lauck G, et al. MR imaging and cardiac pacemakers: in-vitro evaluation and in-vivo studies in 51 patients at 0.5 T. *Radiology* 2000;215:869-879.
28. Atlee JL, Bernstein AD. Cardiac rhythm management devices (part i), indications, device selection, and function. *Anesthesiology* 2001;95:1265-1280.
29. Atlee JL, Bernstein AD. Cardiac rhythm management devices (part ii), perioperative management. *Anesthesiology* 2001;95:1492-1506.
30. Stone KR, McPherson CA. Assessment and management of patients with pacemakers and implantable cardioverter defibrillators. *Crit Care Med* 2004;32 (S4):s155-s165.
31. Salukhe TV, Dob D, Sutton R. Pacemakers and defibrillators: anaesthetic implications. *Br J Anaesth* 2004;93:95-104.

**Editor's note:** In the January 2005 issue of *Anesthesiology Rounds* (Ultrasound-guided Brachial Plexus Anesthesia by Dr. S Williams), the journal for reference #21 was incorrectly listed. It should have stated: De Andres J, Sala-Blanch X. Ultrasound in the practice of brachial plexus anesthesia. *Reg Anesth Pain Med* 2002;27(1):77-89.

Change of address notices and requests for subscriptions to *Anesthesiology Rounds* are to be sent by mail to P.O. Box 310, Station H, Montreal, Quebec H3G 2K8 or by fax to (514) 932-5114 or by e-mail to [info@snellmedical.com](mailto:info@snellmedical.com). Please reference *Anesthesiology Rounds* in your correspondence. Undeliverable copies are to be sent to the address above. Publications Post #40032303

---

This publication is made possible by an educational grant from  
**Organon Canada Limited**

---