Anaesthetic management of a patient with an automatic implantable cardioverter defibrillator in situ

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Summary

The automatic implantable cardioverter defibrillator (AICD) is becoming used more frequently in patients with refractory malignant ventricular arrhythmias. The anaesthetic implications of patients with an AICD in situ presenting for surgery are discussed. (Br. J. Anaesth. 1997; 78: 102–106)

Key words

Equipment, defibrillators. Complications, tachycardia.

Malignant recurrent ventricular arrhythmias cause sudden cardiac death in an estimated 40 000 people annually in the United States. Survivors of cardiac arrest secondary to ventricular tachycardias have a 30% risk of a recurrent arrest within 1 yr and a 45% risk at 2 yr. The mortality rate is approximately 60% within 2 yr in spite of antiarrhythmic prophylaxis considered adequate at that time. A major advance in the management of recurrent malignant ventricular tachycardia has been the development of the automatic implantable cardioverter defibrillator device (AICD) by Mirowski, Mower and Staewen. Since the first device was implanted in 1980, technological advances have led to a marked increase in the insertion of AICDs, especially with the availability of systems that do not require thoracotomy for implantation and further miniaturization of the generator. Anaesthetists are therefore likely to encounter patients with an AICD in situ presenting for surgery unrelated to the defibrillator.

This article describes the anaesthetic management of a patient with a third-generation AICD in situ presenting for total hip replacement and discusses the management issues relevant to anaesthetists.

Case report

A 62-yr-old man, a Jehovah’s Witness with an implanted AICD, presented for left total hip replacement. He had a long history of hypertension and ischaemic heart disease for which he underwent coronary artery bypass surgery 5 yr and resection of a posterobasal ventricular aneurysm 2 yr previously. As he developed recurrent ventricular tachycardia (VT) and fibrillation (VF) with episodes of atrial flutter after the aneurysectomy, a third-generation AICD (Medtronic 7219C) with two transvenous leads and a subcutaneous patch electrode at the cardiac apex was implanted. The AICD was programmed to provide anti-tachycardia pacing of VT at 130–190 beat min⁻¹, low energy cardioversion for VT greater than 190 beat min⁻¹ and defibrillation at 25–35 J for VF with anti-bradycardia pacing at 50 beat min⁻¹. His current medications were sotalol 80 mg twice daily, captopril 25 mg daily, lisinopril 5 mg and frusemide 20 mg daily. He also had a history of mild obstructive sleep apnoea which was confirmed by sleep studies.

On examination, the patient was slightly overweight (90 kg) with an arterial pressure of 150/80 mm Hg and a heart rate of 60 beat min⁻¹. His ejec-
tion fraction was 30% and preoperative investigations were within normal limits. The preoperative electrocardiogram showed sinus rhythm with Q waves in the inferior leads.

All medications except frusemide were continued and he was premedicated with morphine 10 mg, 1 h before surgery. Before induction of anaesthesia, invasive arterial pressure, central venous pressure via an antecubital catheter, and ECG monitoring together with pulse oximetry were established. Adhesive external defibrillator pads were applied laterally on the chest away from the subcutaneous patches. A left femoral nerve block with 1% lignocaine 25 ml was performed. The patient’s lungs were preoxygenated and anaesthesia was induced with increments of thiopentone to a dose of 175 mg i.v. Neuromuscular block was produced with vecuronium 10 mg and tracheal intubation performed. The patient’s lungs were preoxygenated and anaesthesia was induced with increments of thiopentone to a dose of 175 mg i.v. Neuromuscular block was produced with vecuronium 10 mg and tracheal intubation performed. The patient’s lungs were ventilated with 0.5–1% isoflurane and nitrous oxide in oxygen, and normocapnia was maintained with the aid of a capnograph. Normothermia (36.1 °C) was maintained with the use of a warm air convective blanket (Bair Hugger, Augustine Medical, USA) and an i.v. fluid warmer.

A programmer from the cardiology department was in attendance and the anti-tachycardia, cardioversion and defibrillation functions of the AICD were deactivated and the anti-bradycardia pacing function left intact. During operation, there was a transient episode of atrial flutter with a slow ventricular rate resulting in mild hypotension (75/50 mm Hg) and this triggered the anti-bradycardia pacing...
function of the AICD. The patient responded to metaraminol 0.5 mg and arterial pressure was maintained at 120–150/60 mm Hg with a CVP of 9–19 mm Hg.

Diathermy using bipolar leads was used throughout the procedure. A Muller cemented hip prosthesis was inserted. Blood loss was estimated to be 400 ml and the patient received 1 litre of normal saline. At the end of the procedure, neuromuscular block was antagonized with neostigmine 2.5 mg and glycopyrronium 0.4 mg.

In the recovery ward, the AICD was reactivated to its preoperative parameters. Analgesia was provided with morphine i.m. On the first postoperative day, the AICD was tested to ensure it was functioning appropriately. The patient was monitored in a high dependency area of the orthopaedic ward. However, the patient developed VT on the third and 10th postoperative days which was terminated successfully by anti-tachycardia pacing.

Discussion

The AICD is proving to be an effective and innovative device for the treatment of patients with drug-resistant malignant ventricular arrhythmias, reducing the 1 yr mortality rate from 66% to approximately 8.5%.

An understanding of these devices is essential for anaesthetists who are likely to encounter these patients with an AICD in situ presenting for surgery unrelated to the device.

AICD technology has become increasingly sophisticated and complex (table 1). The original first-generation AICD had only defibrillation capability without any programmability. The second-generation devices had limited programmability of rate threshold parameters and shock energy. The third-generation devices provide anti-bradycardia pacing, anti-tachycardia pacing, low energy cardioversion and standard defibrillation capabilities. The fourth-generation devices use a single right ventricular coil electrode and the body of the generator as the other pole for the delivery of the shock and are referred to as active or hot can devices.

Fifth-generation devices which have DDD pacemaker functions and can identify and treat atrial arrhythmias are currently undergoing clinical trials.

The AICD consists of a pulse generator and a lead electrode system for sensing, pacing and delivery of shocks for cardioversion or defibrillation. The pulse generator is made up of a battery source (lithium vanadium silver pentoxide cells), capacitors for high voltage generation, and a storage and microprocessor logic system which analyses incoming signals and makes decisions regarding appropriate therapy based on algorithms set by the cardiologist. Older pulse generators weigh 200 g with a volume of 80–100 ml but newer devices may weigh 100 g with a volume of 60–100 ml. Larger generators are implanted in the left anterior abdominal wall behind the rectus muscle. The newer smaller generators can be implanted in the pectoral region either subcutaneously or subpectorally.

Before 1990, the AICD used lead systems comprising epicardial patches which therefore required a thoracotomy. Non-thoracotomy lead systems have become available over the past few years and consist of a ventricular lead with a distal sensing pacing tip and a defibrillation coil placed in the right ventricle. A second defibrillation coil is placed in the superior vena cava (SVC) or right atrium (RA), either on the same lead or on a separate lead positioned in the SVC or anchored to the RA. A third lead system usually comprising an array of leads placed subcutaneously over the cardiac apex may be required to achieve satisfactory defibrillation thresholds. The most recent advance has been the development of the active can system which uses a single ventricular electrode.

The sensing components of the AICD continuously monitor and analyse the patient’s heart rate (using the R-R interval) and waveform morphology by measuring the variance from baseline. The absence of isoelectric segments is typical of VF or sinusoidal tachycardias. Different emphasis on either the rate detection channel or the waveform channel may be selected by the cardiologist. Emphasis on the rate detection channel improves the detection of non-sinusoidal tachycardia but may sense and inappropriately deliver a shock in sinus tachycardia, supraventricular tachycardia or atrial fibrillation with a rapid ventricular response. With the fourth-generation devices, it is possible to programme to sense the rate of change in heart rate, therefore minimizing delivery of shock while the heart is in sinus rhythm. Dual sensing models have a better

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<th>Manufacturer/Model</th>
<th>Detection Morph. Rate</th>
<th>Pacing AB AT</th>
<th>Therapy DF LEC Prog.</th>
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<td>First-generation Intec aid</td>
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<td>Second-generation CPI Ventak 1600</td>
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<td>Third-generation CPI Ventak PRX 1700</td>
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<td>Medtronics PCD 7215</td>
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specificity for non-malignant arrhythmias but may not detect non-sinusoidal ventricular tachycardia. A malignant ventricular tachycardia is usually present for 5–15 s before the preset rate and morphology criteria are met, at which point the AICD charges its capacitor and delivers a shock of 25 J. The entire process takes 35 s and if VT persists, subsequent shocks of 30 J are delivered. After four shocks, the AICD resets in 35 s and if VT persists, another series of four shocks are delivered. The characteristics of the shock waveform (monophasic/biphasic) and current-pathway (single, bidirectional or sequential) used in the AICD vary depending on the model and lead system used. The latest advanced AICD are capable of different responses to one or more tachycardias classified by rate and a programmable sequence or graded (or “tiered”) therapy to an arrhythmia by providing adaptive pacing sequences to low energy cardioversion to high energy defibrillation. With the fourth-generation devices, the use of the large surface area of the “can” or body of the device as a pole for delivery of the shock decreases defibrillation thresholds and conserves battery life. If the malignant VT is shown to be terminated by pacing in the electrophysiology laboratory, algorithms may used to attempt pace termination of malignant arrhythmia. However, current devices may not be able to distinguish some forms of supraventricular and ventricular tachycardias, although atrial fibrillation may be identifiable if the variability in the RR interval is programmed into the device.

In the preoperative evaluation of patients with AICD presenting for non-cardiac surgery, the underlying cardiac disease (ischaemic or valvular heart disease, cardiomyopathy) and the status of their ventricular function should be taken into account. It is important that these patients are receiving optimal medical therapy and a knowledge of preoperative antiarrhythmic medications for each individual patient would be useful if intraoperative VT or VF occurs. Preoperative investigations should be directed towards detecting any electrolyte imbalance or drug toxicity which may predispose to intraoperative complications or cause inappropriate discharge of the AICD. In the patient described, frusemide was omitted to reduce problems associated with hypovolaemia and haemodynamic instability. Patients with AICD frequently suffer from depression as a result of the stresses associated with frequent discharges of the AICD and may be receiving antidepressant medication.

A knowledge of the model of the AICD is important as there is wide variation in the functional characteristics of different models.57 Patients usually carry a medic alert bracelet and identification data that would provide information on the model, lead systems, therapy options and contact details of the cardiologist. It would be prudent for the anaesthetist to consult the cardiologist involved. An overpenetrated radiograph of the pulse generator would identify the model of the AICD. A chest x-ray would reveal the lead systems, position of the patches and pulse generator which are important for the intraoperative management of these patients.

AICD malfunction may be related to battery depletion, failure of sensing as a result of altered thresholds, and fracture or migration of lead systems. Cardiac symptoms may indicate the complications of AICD implantation such as infection, pericarditis, pericardial effusion, cardiac fibrosis or progression of the underlying cardiac disease.

Although there is little information available, the anaesthetic management of patients with an AICD is similar to that for patients with rate-responsive permanent pacemakers. In minor and short surgical procedures, monitoring should consist of ECG and pulse oximetry with a device that has a plethysmographic display. For major surgical procedures or patients with severe impairment of left ventricular function, intra-arterial pressure monitoring is mandatory as it would demonstrate the adequacy of perfusion in the event of intraoperative tachyarrhythmia. Central venous access and pressure monitoring is useful as it provides a route for vasoactive drugs and may aid in the optimization of fluid therapy. The use of a pulmonary artery catheter is dictated by the patient’s cardiac function but its benefits must be weighed against the risks of inducing VT or VF and displacing the lead systems of the AICD during its insertion. An external cardioverter/defibrillator should be available in the operating theatre. Adhesive external defibrillator patches should be placed appropriately on the patient’s chest and it is important to ensure that the appropriate connectors are available. These enable effective cardioversion/defibrillation without Contamination or interference of the surgical field.

Although no specific anaesthetic techniques have been advocated, the main objectives are to avoid factors and drugs that induce VT or VF and to maintain haemodynamic stability. Adequate analgesia is essential to reduce sympathetic overactivity during and after operation.

In general, all AICD should be deactivated during surgery as they can inappropriately discharge because of extraneous signals, such as myopotentials caused by shivering, or fasciculations induced by suxamethonium, diathermy, orthopaedic instrumentation, and by false sensing as a result of sinus tachycardia or rapid atrial fibrillation. However, with the third- and fourth-generation AICD, selective deactivation of the anti-tachycardia, low energy cardioversion and defibrillation functions can be achieved with the anti-bradycardia pacing function remaining reactivated. This is useful if the patient develops severe bradycardia during operation, as seen in this patient. Deactivation of the AICD is accomplished best using its programming device. Deactivation of the first- and second-generation devices can also be achieved by placing a “ring” or “donut” magnet over the pulse generator for 30 s which is indicated by a continuous tone. Removal of the magnet maintains the pulse generator in its inactive mode. Reactivation of the AICD may be achieved by placing the ring magnet over the inactive pulse generator for 30 s, and pulse tones synchronous with the QRS complexes indicate that the generator is reactivated. However, the tones emitted from these devices are
extremely difficult to hear. With the proliferation of new devices, the effects of a magnet are not consistent for all models of AICD.\textsuperscript{13} The third- and fourth-generation devices are inhibited and continue to sense but not deliver therapy in the presence of a magnet.

Electromagnetic interference can lead to malfunction of the AICD.\textsuperscript{10,13} High frequency electromagnetic interference can inhibit the AICD while lower frequency electromagnetic interference may cause the AICD to incorrectly sense and thus inappropriately deliver a shock. During surgery, if diathermy is used, the AICD should be deactivated to avoid inappropriate discharge and reactivated when diathermy is not required. In the patient described, diathermy was required to minimize intraoperative blood loss as he was a Jehovah’s Witness. With the third-generation or multi-function AICD, VT and VF pacing function intact. Diathermy can also cause burns at the myocardial–electrode interface leading to raised pacing thresholds, reprogramming of the AICD and damage to the circuit of the pulse generator. When diathermy is used, the following precautions should be undertaken.\textsuperscript{15} Bipolar diathermy with the lowest current output should be used to prevent damage to the pulse generator. The ground or indifferent electrode of the diathermy should be placed as far away as possible from the generator. Diathermy must not be applied close to the generator as it may damage its circuitry and cause burns to the surrounding tissues.

Magnetic resonance imaging is contraindicated in patients with AICD\textsuperscript{13} as strong magnetic fields produced can exert mechanical forces causing physical pain and damage to the generator, or cause inappropriate delivery of a shock. Radiotherapy (linear accelerator, betatron, radioactive cobalt) may damage the complementary metal oxide semiconductor circuits of the AICD, especially with cumulative doses.\textsuperscript{13,16} Therefore, shielding of the generator should be undertaken and the device evaluated after its exposure to radiation. Theoretically, extracorporeal shock wave lithotripsy can produce mechanical and electromagnetic forces which may damage the piezoelectric crystal timing device of the AICD or cause inappropriate delivery of a discharge. However, bench analysis\textsuperscript{17} and a case report\textsuperscript{18} suggest that contralateral lithotripsy can be performed safely in patients with an AICD \textit{in situ}. The safety of lithotripsy applied ipsilateral to the AICD is unknown. As electroconvulsive therapy (ECT) frequently induces transient arrhythmias, the AICD should be deactivated just before treatment and reactivated immediately after ECT.\textsuperscript{13} Grounding of the patient should be avoided as the delivered current may be rerouted to the heart via the defibrillator electrodes resulting in VF.

The presence of an AICD should not discourage standard cardiopulmonary resuscitation.\textsuperscript{12,13} External defibrillation does not damage the AICD and should be administered promptly on the assumption that the implanted device is not operative. With the first- or second-generation AICD, the presence of the epicardial patches may increase the energy required for external defibrillation as a result of the shielding effect of the insulating portions of the patches or the shortening of energy away from the myocardium through the conducting portions of the patches.\textsuperscript{13} An anterior–posterior placement of external defibrillation pads has been shown to be efficacious in such patients.\textsuperscript{19} In patients with subcutaneous electrodes requiring external defibrillation, significant interaction may occur if the chest paddles are applied over the subcutaneous electrode. After external defibrillation, the pulse generator should be examined for correct function and to ensure that the programmed parameters have not been reset. However, internal defibrillation can damage the AICD. Personnel administering cardiopulmonary resuscitation may experience a mild electric shock on the patient’s body surface if an AICD discharges but this should not pose a risk to the personnel during resuscitation. Further, as rapid supraventricular tachycardias (SVT) with adequate perfusion are common during resuscitation, internal defibrillator discharges may be triggered by the SVT and result in VT or VF. Thus in a controlled environment it may be desirable to deactivate the AICD during cardiopulmonary resuscitation.

In the postoperative period, hypoxaemia, hypercapnia and inadequate analgesia causing increased sympathetic activity may predispose to VT and VF. Continuous monitoring is essential until vital signs are stable. Spontaneous skeletal muscle contractions caused by shivering can generate myopotentials which may inappropriately trigger the AICD. Low frequency electromagnetic interference may arise from transcutaneous electrical nerve stimulation (TENS) leading to inappropriate delivery of shocks and therefore are contraindicated.\textsuperscript{15} It is imperative that the devices are tested before patients are discharged from hospital.

As automatic implantable cardioverter defibrillator therapy has become more popular and sophisticated, these devices present the anaesthetist with several diagnostic and management issues and hence an understanding of the functions of these devices is essential for the optimal care of the patient.

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