Use of implantable cardioverter defibrillators in the New Zealand context from 2000 to 2007

Peter D Larsen, Praveen De Silva, Scott A Harding, Ellen Woodcock, Nigel A Lever

Abstract

Background Implantable Cardioverter Defibrillator (ICD) therapy is now standard of care for prevention of sudden cardiac death in high-risk patient groups. In order to determine if the potential benefit of ICD therapy is being realised, ongoing monitoring of ICD therapy is required. This study was conducted to examine ICD therapy in two New Zealand tertiary hospitals.

Methods We retrospectively audited patient notes for all patients receiving a first ICD between 2000 and 2007 at two tertiary referral hospitals in New Zealand.

Results 702 patients received their first ICD within the study period, 73% male, mean age 53 years (range 1 to 83), with 73% of devices for secondary prevention. The implant rate increased from 15/million in 2000 to a peak of 44/million in 2004. Antitachycardia pacing was delivered to 21% of patients, appropriate defibrillation to 26% and inappropriate defibrillation to 16% of patients, with frequency of all types of therapy increasing with time since implantation. All cause mortality was 8.6%, and only 7 (1%) died as a consequence of sudden cardiac arrest.

Conclusions While increasing across the study period, the ICD implant rate remains low, with a high therapy rate and low mortality rate. This suggests that those receiving ICD therapy are benefiting, but may also imply that the group of patients receiving ICDs is too restricted.

Implantable cardioverter defibrillators (ICDs) have revolutionised the management of patients at high risk of sudden cardiac death since they were introduced to clinical practice in 1980. In the decade following their introduction, the design of the ICD was dramatically improved, and a series of observational studies demonstrated that the ICD appeared to be clinically useful. Following these observational studies, a number of large clinical trials were conducted, and these showed that for patients with previous symptomatic or sustained ventricular arrhythmias, mortality was significantly reduced in patients with ICDs when compared to optimal medical therapy. A second series of clinical trials were conducted demonstrating that ICDs also reduced mortality in populations with impaired left-ventricular function due to previous myocardial infarction or heart failure. Survival benefits of ICDs in patients with a range of channelopathies has also been demonstrated. There is now widespread agreement that the ICD is standard of care for reducing mortality in a wide range of patients at increased risk of sudden cardiac death. This has led to the development of robust, evidence-based guidelines for ICD therapy.
While the clinical indications for ICD therapy are now widely accepted, it is not clear that clinical practice is consistent with these guidelines. \(^{11-13}\) In order to realise the potential reduction in sudden cardiac death that is offered by ICD therapy, it is necessary to both audit current practice and outcomes in patients receiving ICDs, and to identify barriers to ICD therapy. This study was conducted to address the first of these goals by auditing the use of ICDs in two of the four tertiary referral hospitals offering this service in New Zealand over the period from 2000 to 2007.

**Methods**

We conducted a retrospective review of all patients receiving their first ICD implant at either Wellington Hospital or Auckland City Hospital between 1 January 2000 and 31 December 2007, and included clinical data from this time period. The study was reviewed by the Central Regional Ethics Committee, and found to conform to the New Zealand Ethical Guidelines for Observational Studies.

We collected data on indications for ICD implantation and co-morbidities, details of the type of device, and follow-up details of all delivered therapy, hospitalisations and deaths. This was done by reviewing implant records, arrhythmia clinic notes including device printouts following interrogation, cardiologist letters and hospital records for each patient. ICD indications were classified as primary or secondary prevention.

The secondary prevention group were the patients who had a previous ventricular tachycardia (VT) or ventricular fibrillation (VF) cardiac arrest, syncopal VT, or sustained VT. Non-sustained non-syncopal VT was classified as primary prevention, as were all other indications in which VT/VF had not occurred prior to implantation. Therapy was classified into anti-tachycardia pacing (ATP), appropriate shocks and inappropriate shocks.

ATP was considered to have been successful if the arrhythmia was terminated without required a shock, and to have failed if a shock was required. Shocks were defined as inappropriate if they were not due to VT/VF and appropriate if they were due to VT/VF.

Implantation rates were determined by dividing the number of devices by the referral population for the two tertiary referral hospitals. Population data was from New Zealand Statistics.\(^{14}\)

**Data analysis**—Continuous variables were compared between groups using unpaired t-test, while discrete variables were compared using Chi-Squared test. All statistical tests were performed using SPSS 11 (Chicago, IL).

**Results**

Over the 8-year study period 702 patients received their first ICD, and the median follow-up time was 40 months (interquartile range 18–64 months). The majority of these devices were implanted for secondary prevention (511 patients, 73%).

Demographic data are given for all patients in Table 1, as well as for the primary and secondary prevention groups. Overall, patients were predominantly male (73%), and had a mean age of 53 years (range 1 to 83 years).

Patients receiving devices for secondary prevention were more likely to have had a myocardial infarction (p=0.0001), and therefore also more likely to have had either angioplasty (p=0.001) or coronary artery bypass surgery (p=0.003), than those receiving primary prevention devices. Secondary prevention patients were also more likely to be on amiodarone at the time of implantation (0=0.002). Patients receiving devices for primary prevention were more likely to have either a non-ischaemic dilated (p=0.0001) or a hypertrophic cardiomyopathy (p=0.003), and were more likely to have an ejection fraction less than 30% (p=0.0001), and be taking spironolactone (p=0.0001) and/or another diuretic agent (p=0.001), suggesting a higher rate of heart
failure in this group. Primary prevention patients were younger (p=0.001), and included more females (p=0.02) than the secondary prevention group.

Table 1. Patient demographics.

<table>
<thead>
<tr>
<th></th>
<th>All patients n=702</th>
<th>Primary prevention n=191</th>
<th>Secondary prevention n=511</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>539 (77%)</td>
<td>135 (71%)</td>
<td>404 (79%)</td>
<td>0.02</td>
</tr>
<tr>
<td>Age</td>
<td>53 (1-83)</td>
<td>49 (6-78)</td>
<td>54 (1-83)</td>
<td>0.001</td>
</tr>
<tr>
<td><strong>Cardiac Disease:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myocardial Infarct</td>
<td>229 (33%)</td>
<td>30 (16%)</td>
<td>199 (39%)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Valve Disease</td>
<td>103 (15%)</td>
<td>23 (12%)</td>
<td>80 (16%)</td>
<td>0.28</td>
</tr>
<tr>
<td>Dilated Cardiomyopathy</td>
<td>123 (18%)</td>
<td>62 (32%)</td>
<td>61 (12%)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Ischaemic Cardiomyopathy</td>
<td>165 (24%)</td>
<td>46 (24%)</td>
<td>119 (23%)</td>
<td>0.82</td>
</tr>
<tr>
<td>Hypertrophic Cardiomyopathy</td>
<td>37 (5%)</td>
<td>18 (9%)</td>
<td>19 (4%)</td>
<td>0.003</td>
</tr>
<tr>
<td>Arrhythmogenic Right</td>
<td>33 (5%)</td>
<td>11 (6%)</td>
<td>22 (4%)</td>
<td>0.42</td>
</tr>
<tr>
<td><strong>Ventricular</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Cardiomyopathy</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Long QT</td>
<td>73 (10%)</td>
<td>25 (13%)</td>
<td>48 (9%)</td>
<td>0.22</td>
</tr>
<tr>
<td>Brugada Syndrome</td>
<td>9 (1%)</td>
<td>6 (3%)</td>
<td>3 (1%)</td>
<td>0.007</td>
</tr>
<tr>
<td>LVEF less than 30%</td>
<td>191 (27%)</td>
<td>72 (38%)</td>
<td>119 (23%)</td>
<td>0.0001</td>
</tr>
<tr>
<td><strong>Previous Arrhythmia:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VF</td>
<td>307 (44%)</td>
<td>–</td>
<td>307 (60%)</td>
<td></td>
</tr>
<tr>
<td>Sustained VT</td>
<td>220 (31%)</td>
<td>–</td>
<td>220 (43%)</td>
<td></td>
</tr>
<tr>
<td>Non sustained VT</td>
<td>163 (23%)</td>
<td>83 (43%)</td>
<td>80 (16%)</td>
<td>0.0001</td>
</tr>
<tr>
<td>AF</td>
<td>173 (25%)</td>
<td>46 (24%)</td>
<td>127 (25%)</td>
<td>0.83</td>
</tr>
<tr>
<td><strong>Previous Interventions:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percutaneous Intervention</td>
<td>75 (11%)</td>
<td>8 (4%)</td>
<td>67 (13%)</td>
<td>0.001</td>
</tr>
<tr>
<td>Coronary Artery Bypass Graft</td>
<td>126 (18%)</td>
<td>21 (11%)</td>
<td>105 (20%)</td>
<td>0.003</td>
</tr>
<tr>
<td><strong>Medications at Implant:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beta-Blocker</td>
<td>454 (65%)</td>
<td>121 (63%)</td>
<td>333 (65%)</td>
<td>0.65</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>172 (25%)</td>
<td>31 (16%)</td>
<td>141 (28%)</td>
<td>0.002</td>
</tr>
<tr>
<td>ACE Inhibitor</td>
<td>339 (48%)</td>
<td>107 (56%)</td>
<td>232 (45%)</td>
<td>0.01</td>
</tr>
<tr>
<td>Spironolactone</td>
<td>153 (22%)</td>
<td>60 (31%)</td>
<td>93 (18%)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Diuretic</td>
<td>237 (34%)</td>
<td>83 (43%)</td>
<td>154 (30%)</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Note: Data is given as number (percentage), and compared between primary and secondary prevention groups using Chi Square, apart from age, which is given as mean (range) and compared using unpaired Student’s t-test.

The number of new implants per year and the new implant rate per million are shown in Figure 1. These graphs show a marked increase in both number and rate across the study period. The implant number and rate both peaked in 2004 at 123 devices and 44 devices per million respectively. In order to get national figures, we contacted Christchurch Hospital and Waikato Hospital, which are the other two public centres in New Zealand that implant ICDs.

Including new implant figures from these two hospitals, the national implant rate increased from 18 per million in 2000 to 49 per million in 2006 (Figure 1). The national data show that the implant rate in Auckland and Wellington is slightly lower than in the other two centres. The percentage of implants for primary prevention is shown in Figure 2, and showed an increase across the study period, from 24% in 2000 to 37% in 2007.
Device details—Over the study period 74% of implanted devices were single chamber ICDs, 23% were dual chamber and 3% were cardiac resynchronisation (CRT) devices. With the exception of 2001, where 90% of devices were single chamber, the proportion of single chamber devices was very consistent across the 8-year study period (Figure 3). CRT devices were first implanted in 2003, and while the percentage of these devices being implanted increased, they still accounted for less than 5% of new implanted devices in 2006 and 2007.

Figure 1. Total number of new implants by year (A) and implant rate per million population by year (B) are given for the study population (histogram bars) and for the entire country (line plot).
Figure 2. Percentage of new ICD implants for primary prevention by year.

![Percentage Implants for Primary Prevention](image)

Figure 3. Percentage of each device type (single chamber, dual chamber or cardiac resynchronisation – CRT devices) for new ICD implants by year.

![Device Type Percentage](image)
Device therapy—Overall 21% of all patients had received ATP, 26% had received appropriate shocks for VF or VT, and 16% had received inappropriate shocks. Only 57% of patients had ATP zones programmed at the time of implantation (39% of the primary prevention group, and 62% of the secondary prevention group), although a number of these patients may have had ATP zones programmed subsequently.

Of those receiving ATP 22% had only 1 episode, 33% had to 2-5 episodes of ATP, 15% had 6-10 episodes and 30% had more than 11 episodes of tachyarrhythmia treated with ATP. ATP failed to terminate the tachyarrhythmia in 23% of episodes.

Of those receiving appropriate shocks 36% had only one VT/VF event, 38% had 2-5 events, 11% 6-10 events, and 16% had more than 11 events requiring defibrillation. The first shock was successful in terminating VT/VF in 94% of cases. In total 31 patients had episodes of VT/VF that were not terminated by the first shock.

In all but 2 cases, subsequent device shocks terminated the arrhythmia. In the 2 patients who had episodes of VF that were not terminated at all by the ICD, 1 was rescued by external defibrillation, and in the other case the patient died as a result of refractory VF.

Appropriate therapy was more likely the longer the device had been implanted, increasing to 45% for those with an ICD for 6 to 8 years. Appropriate therapy was also significantly more likely in secondary prevention patients (p=0.001).

Overall 16% of patients received inappropriate shock therapy, most commonly because of atrial fibrillation (50% of patients) or SVT (35% of patients). A smaller number experienced inappropriate therapy because of over-sensing (11%), or lead problems (2%). In 2 patients the cause of inappropriate therapy was not clearly documented. Of those experiencing inappropriate therapy, 38% had one episode, 41% had 2-5 episodes, 12% had 6-10 episodes and 8% experienced more than 11 episodes.

As with appropriate therapy, inappropriate therapy increased with time since device implantation, reaching 29% at 6-8 years. Inappropriate therapy was more likely for patients with previous AF (p=0.04) patients with underlying ischaemic cardiomyopathy (p=0.04), patients with single chamber devices (p=0.02) and in patients without ATP zones programmed on their device (p=0.01).

Deaths—At the end of the study period 61 (8.6%) of the patients had died. Only 13 (21%) of these patients had their device interrogated following death. In 23 (36%) patients no clear cause of death was determined. Of the remaining deaths, 9 (15%) were non-cardiac in nature, and 29 (48%) were due to cardiac causes. Of the cardiac deaths, 22 were attributed to progressively deteriorating heart failure, and 7 were attributed to sudden cardiac arrest. Of the 7 cardiac arrests that were fatal, 5 were due to pulseless electrical activity after VF was successfully treated by the ICD, 1 was due to refractory VF as mentioned above, and 1 was due to a head injury sustained from a fall at the time of cardiac arrest.

Discussion

The use of ICD therapy to combat sudden cardiac death increased across the study period. The proportion of devices implanted for primary prevention increased from 24% to 37% across the study period. Observed treatment rates were consistent with
international experience, with 26% of the patients received shocks from their device for VT/VF, and 21% receiving ATP for VT. Overall mortality was 8.6%, which is low by international standards.

While the implantation rate at our 2 institutions has increased over the study period, the rate has not continued to rise since 2004. The national figures differ slightly, in that a more continual increase across the study period is seen. The peak national implant rate of 49 per million is low by international standards. An abstract presented at the European Society of Cardiology meeting in 2007 reported that European implant rates varied between 1 and 226/million, and argued that this represented under-utilisation of the therapy.

The Italian registry reports an implant rate of 125/million in 2004, the Spanish registry a rate of 62/million in 2005, and the Danish Registry a rate of 117/million in 2006. In Australia an implant rate of 142/million in 2005 has been reported. Given that the New Zealand burden of cardiovascular disease is similar or greater than these countries, our implant rate is probably too low. Further investigation is required to determine how many patients who meet the criteria for ICD implantation are not receiving treatment and the extent to which limited resources and funding act as barriers to treatment.

Our patient group had a mean age of 53 years, which is young when compared to the Spanish (mean 61 years), Danish (mean 66 years) or Italian (mean age 68 years) populations receiving this therapy. While there is evidence that patients over 75 receive the same benefit as younger patients from ICD therapy in terms of preventing sudden cardiac death, the older age group have higher all cause mortality rates. For this reason it is possible that older patients, with higher levels of co-morbidities are less likely to receive ICD therapy.

The use of ICDs for primary prevention increased across the study period from 24% in 2000 to 37% in 2007. This is consistent with expanding indications for ICD therapy, and international trends. The Spanish registry reported in 2005 that 39% of devices were for primary prevention and the Italian Registry 24% in 2004. Despite this increase in primary prevention use, the population of patients with primary indications for ICDs is likely to exceed the secondary population, but still accounts for considerably less than half all implanted devices.

The overall mortality rate in our study (8.6%) was low when compared to other series with a similar length follow-up, which range between 19% and 36%. Mortality rate in the defibrillator arm of the large primary and secondary prevention studies was also higher that seen in our series. However, as commented on above, our patient group is young for an ICD population, and potentially we are choosing not to offer ICD therapy to patients with significant risks of death due to factors other than sudden cardiac arrest, both of which would lower overall mortality rates. There was a very low rate of device interrogation following death. It is therefore possible that there is a considerably larger portion of sudden cardiac deaths in this population than we have detected, and some of these may be due to a failure to terminate VT/VF.

Overall, 26% of patients in this study had received appropriate shocks from their device, 21% ATP, and 15% inappropriate treatment. The rate of ATP in our series is relatively low, given that there is evidence that up to 70% of VT episodes may be
adequately treated by ATP, and not require shock treatment.\textsuperscript{24,25} The relatively low rate of ATP is consistent with the limited initial programming of ATP, and this of particular concern, as our study is consistent with previous work demonstrating that ATP programming reduces the probability of inappropriate shock therapy.\textsuperscript{26} Despite the low use of ATP, the inappropriate therapy rates in our study were similar to those described in other series.\textsuperscript{21,27,28} Our findings that patients with previous atrial fibrillation,\textsuperscript{28} and with single chamber devices\textsuperscript{27} were more likely to experience inappropriate shocks are also consistent with previous work.

This study is a retrospective audit, and as such suffers from a number of inherent limitations. Gaps exist in the data, and data has not always been recorded according to tightly defined criteria. The study does not represent the entire country. The two hospitals studied, Auckland and Wellington, provide the ICD service to approximately 68\% of the New Zealand population. The implant rate at the other two hospitals, Christchurch and Waikato, is slightly higher and the practice may differ slightly in other ways. We have also not captured the number of ICDs that are implanted within the private sector, but this is a very small group as the major medical insurers in New Zealand do not currently fund this therapy. Few of the patients who died had their ICD returned for interrogation and the cause of death was unclear in a significant number. It is therefore possible that there were considerably more arrhythmic deaths than we are aware of within the study.

We have observed an increasing rate of ICD implantation across the study period, with an increase in the proportion of patients receiving these devices for primary prevention of sudden cardiac death. The proportion of patients receiving appropriate shocks for VT/VF is high, demonstrating that an appropriately high risk group of patients are being selected for ICD therapy. However, the possibility that this therapy is not available to a wide enough patient group is supported by the still low implant rate, the young average age of implant recipients and the low overall mortality rate. Further work on the potential benefits of more widely available ICD therapy in the New Zealand context is required.

Competing interests: None known.

Author information: Peter D Larsen, Associate Professor \textsuperscript{1}; Praveen De Silva Medical Student\textsuperscript{1}; Scott A Harding Cardiologist\textsuperscript{2}; Ellen Woodcock, Assistant Lecturer\textsuperscript{1}; Nigel A Lever, Senior Lecturer\textsuperscript{3}

1. Department of Surgery and Anaesthesia, University of Otago, Wellington
2. Cardiology Department, Wellington Hospital
3. Department of Medicine, University of Auckland

Acknowledgement: This study was supported by a grant from the Wellington Surgical Research Trust.

Correspondence: PD Larsen, University of Otago, Wellington, PO Box 7343, Wellington, New Zealand. Fax: +64 (0)4 389 5318; email: peter.larsen@otago.ac.nz

References:


