Lead Systems for Internal Ventricular Fibrillation

G. Michael Deeb, M.D., Bartley P. Griffith, M.D., Mark E. Thompson, M.D., Alois Langer, Ph.D., M. Stephen Heilman, M.D., and Robert L. Hardesty, M.D.

SUMMARY We examined the feasibility of using a standby automatic implantable defibrillator and established thresholds for internal defibrillation. The implantable defibrillator senses ventricular fibrillation and delivers an electrical impulse for defibrillation. Two lead systems for the device have been investigated. System I consists of two 12-cm² silicone-covered titanium mesh patches attached to the atrial and diaphragmatic pericardial surfaces. System II has an identical diaphragmatic patch and a titanium spring catheter, with a 12-cm² surface area of conductivity, placed transvenously in the right atrium. Both systems were implanted by thoracotomy in 12 dogs (mean weight 20 kg) and by a subxiphoid approach in 10 pigs (mean weight 20 kg). The defibrillation threshold (lowest energy required for 80% success) was determined periodically for 54 weeks in the dogs (615 trials) and at 6 weeks for the pigs (100 trials).

In dogs, the mean defibrillation threshold with system I leads at 4 weeks was 10.5 J and did not change significantly over a 54-week period (p > 0.05). Similar results were obtained in the pig at 4 weeks. The defibrillation thresholds for both lead systems in dogs and pigs using a transpleural thoracotomy or a subxiphoid approach are satisfactory for an implantable defibrillator that produces 20–35 J.

THE ONLY EFFECTIVE TREATMENT for ventricular fibrillation is immediate termination by countershock. In an effort to facilitate treatment in a select group of high-risk patients, an AID (Medrad, Inc./Intec Systems, Inc.) implantable defibrillator has been proposed.1 Mirowski et al.2 and Schuder et al.3 demonstrated the feasibility of using such a device in animal preparations. Mirowski et al.2 successfully defibrillated nine of 11 normothermic patients using 5–15 J and a low-energy intraventricular catheter.4

Mirowski et al.5 and Langer et al.7 described an implantable system acceptable in size for implantation in humans. This device delivers up to 30 J, which may be sufficient for direct defibrillation of the human heart.8 We describe two new lead systems for the automatic implantable defibrillator. These electrode systems can be implanted using a subxiphoid approach and eliminate the need for a transpleural or transsternal approach to the mediastinum. These electrodes function as efficiently as the apical cup and catheter system.9

Materials and Methods

Electrode system I consists of two flexible silicone-covered titanium patches (fig. 1). The surface area available for electrical conductivity on each patch is 12 cm². The basilar patch electrode is sutured to the atrial pericardial surface, and the apical patch electrode is sutured to the diaphragmatic pericardial surface. Electrode system II consists of the same apical diaphragmatic patch and a transvenous titanium spring catheter with an electrically active surface area of 12 cm². The ideal position for the apical patch was determined by comparing defibrillation thresholds with a fixed basilar patch electrode over the right atrium and varying the position of the apical patch over the right and left ventricle. In system II the best position for the basilar transvenous spring catheter was determined by comparing defibrillation thresholds with a fixed apical patch electrode over the left ventricle and varying the position of the spring catheter from the junction of the azygos vein with the superior vena cava to a position within the right atrium adjacent to the tricuspid valve.

Most defibrillation thresholds described in the literature10–11 were established using dogs; therefore, dogs were used in establishing the baseline defibrillation thresholds for systems I and II. The midline pericardial–phrenic ligament prevented a subxiphoid approach, so we used a transpleural thoracotomy for

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insertion of electrodes in dogs. After establishment of comparable defibrillation thresholds with other systems, the efficiency of systems I and II using a subxiphoid approach was studied in 10 20-kg pigs.

For implantation, anesthesia consisted of i.v. sodium pentobarbital, 35 mg/kg, and inhalational 1% halothane. Cefamandol Nafate (1 g) was given preoperatively and continued postoperatively (2 g) for 48 hours.

In dogs, implantation was achieved by thoracotomy through the fourth intercostal space. The apical patch was sutured to the pericardium overlying either the right or left ventricle. Once the ideal position for the apical patch was established, all subsequent apical patches were sutured to the pericardium overlying the left ventricle for system I. The basilar patch was sutured to the pericardium overlying the right atrium. In system II, the basilar spring catheter was inserted through the external jugular vein and positioned by palpation at the junction of the aygos vein and the superior vena cava or adjacent to the tricuspid valve. A fibrillating lead was sutured to the interior of the pericardium (fig. 2).

In pigs, a subxiphoid approach was used to place the defibrillating leads. In system II the spring-catheter electrode was positioned with fluoroscopic guidance via the external jugular vein. In system I the basilar patch was positioned in the plane between the sternum and the anterior pericardium. The proximal end of the patch was sutured to the anterior pericardium. The apical patch for both systems was positioned in the plane between the pericardium and the diaphragm and stabilized with a suture. A transvenous electrode to be used for inducing fibrillation was directed into the right ventricle under fluoroscopic guidance. The terminals of the electrodes were buried submuscularly in the abdominal wall (fig. 3).

During testing, the animals were anesthetized with i.v. sodium pentobarbital (30 mg/kg) and ventilated on a respirator with room air. The ends of the leads were retrieved from their subcutaneous sites. Fibrillation was induced with a 50-mA-60-Hz current transmitted to the right ventricle via the fibrillating lead.

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**Figure 1.** Leads for system I and system II. (A) Apical lead. (B) Basilar lead. Both leads are silicone-covered titanium mesh patches that have a 12 cm² surface area of conductivity. (C) Transvenous titanium spring catheter lead, which also has a 12-cm² surface area of conductivity. System I consists of an apical and a basilar lead. System II consists of an apical lead on a transvenous titanium spring catheter.

**Figure 2.** Open mediastinum showing lead positions for dogs with lead system I. The suture is suspending the apex of the heart. (A) The apical patch sutured to the pericardium over the left ventricle. (B) The basilar patch sewn to the pericardium over the right atrium. (D) The fibrillating lead sutured to the pericardium overlying the right ventricle.

**Figure 3.** Lateral chest x-ray showing the position of system I and system II leads. (A) Apical patch between diaphragm and inferior myocardial surface. (B) Basilar patch between anterior myocardial surface and the sternum. (C) Transvenous basilar catheter positioned in the right atrium.
After 15 seconds, defibrillation was achieved with a truncated exponential pulse of 4–30 J.

The energy levels for defibrillation were 4, 6, 8, 9, 10, 12.5, 15, 16, 20, 24, 25 and 30 J. The first defibrillation attempt was at 4 J, and if unsuccessful the animal was rescued with a shock of 30 J. Sequential attempts were performed until successful defibrillation occurred. The lowest energy that was successful was then used for defibrillation in 10 subsequent fibrillation-defibrillation trials. Failure to convert to normal sinus rhythm with the test energy level necessitated rescue with the next highest energy level in the sequence. The defibrillation threshold was defined as the lowest amount of energy required for conversion to a normal sinus rhythm in 80% of the trials. Testing showed the trial energy level for defibrillation either failed miserably (< 20% conversion), necessitating rescue or successfully defibrillated in 100% of the trials. The data were analyzed using the t test for intersystem comparison. Data were recorded as the mean ± SEM.

**Results**

Using system I, with the apical patch over the left ventricle, the mean defibrillation threshold in dogs at 4 weeks was 10.5 J and did not change significantly over 54 weeks (p > 0.05). Using system II, the mean threshold in dogs with the basilar spring catheter in the right atrium near the tricuspid valve was 11.2 J at 4 weeks and did not change significantly over 54 weeks (p > 0.05) (table 1).

The position of the apical patch was critical. Defibrillation with as much as 30 J was unsuccessful in many animals when the patch was located over the right ventricle. The threshold varied according to the position of the transvenous catheter. For system II, the mean threshold was 25.0 J with the catheter at the junction of the superior vena cava and azygos vein.

**Table 1. Stability of Thresholds**

<table>
<thead>
<tr>
<th>Time (weeks)</th>
<th>No. of dogs</th>
<th>Trials</th>
<th>Threshold (J) (mean ± SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>System I</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>6</td>
<td>92</td>
<td>10.5 ± 6.3</td>
</tr>
<tr>
<td>8</td>
<td>6</td>
<td>93</td>
<td>11.2 ± 5.6</td>
</tr>
<tr>
<td>12</td>
<td>6</td>
<td>91</td>
<td>11.5 ± 5.4</td>
</tr>
<tr>
<td>54</td>
<td>3</td>
<td>33</td>
<td>10.0 ± 2.0</td>
</tr>
<tr>
<td>System II</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>6</td>
<td>93</td>
<td>11.2 ± 5.3</td>
</tr>
<tr>
<td>8</td>
<td>6</td>
<td>92</td>
<td>12.6 ± 6.7</td>
</tr>
<tr>
<td>12</td>
<td>6</td>
<td>90</td>
<td>12.6 ± 6.7</td>
</tr>
<tr>
<td>54</td>
<td>3</td>
<td>31</td>
<td>11.7 ± 3.8</td>
</tr>
</tbody>
</table>

Mean threshold for defibrillation in dogs over 54 weeks with system I and system II leads. There is no significant change in the threshold within each system or between both systems (p > 0.05).

**Table 2. Catheter Position, System II**

<table>
<thead>
<tr>
<th>Position</th>
<th>No. of dogs</th>
<th>No. of trials</th>
<th>Threshold (mean ± SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SVC-AZ</td>
<td>3</td>
<td>30</td>
<td>25.0 ± 5.0</td>
</tr>
<tr>
<td>RA-AV</td>
<td>3</td>
<td>160</td>
<td>10.3 ± 4.5</td>
</tr>
</tbody>
</table>

Abbreviations: SVC-AZ = basilar catheter at the junction of the superior vena cava-azygos vein; RA-AV = basilar catheter in the right atrium near the tricuspid valve.

**Table 3. Mean Threshold for Both Systems Using the Subxiphoid Approach for Lead Implantation**

<table>
<thead>
<tr>
<th>System</th>
<th>No. of pigs</th>
<th>No. of trials</th>
<th>Threshold (mean ± SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>5</td>
<td>50</td>
<td>15.8 ± 4.9</td>
</tr>
<tr>
<td>II</td>
<td>5</td>
<td>50</td>
<td>17.0 ± 2.7</td>
</tr>
</tbody>
</table>

There is no significant difference in the thresholds between the two systems (p > 0.05).

When the catheter was advanced into the right atrium at the level of the tricuspid valve, the mean threshold in the same animals decreased to 10.3 J (p < 0.05) (table 2).

The mean threshold for defibrillation in five pigs with system I at 6 weeks was 15.8 J and with system II it was 17 J. There was no significant difference in the threshold between the two systems (p > 0.05) (table 3).

**Discussion**

The inability to control life-threatening arrhythmias with antiarrhythmic drugs has prompted the development of an automatic implantable defibrillator. Many systems have been designed, among them is the AID. The AID is encased in titanium and hermetically sealed with an electron beam weld; it weighs 250 g and has a volume of 145 ml. All materials in contact with body tissue are biocompatible. The AID functions by the probability density mechanism, and it is the absence of the peak at the null point during ventricular fibrillation which triggers the device. A truncated exponential pulse is delivered as much as 30 J about 15–20 seconds after the onset of the arrhythmia and can recycle as many as three times if the first shock is ineffective.

A lead system should reliably and reproducibly defibrillate within the energy range of the device, the threshold should remain stable over time, and it should be easy to insert. Both systems I and II reliably and reproducibly defibrillated the animals within the energy levels of the AID. The threshold remained stable for both lead systems over 54 weeks for 615 trials (p < 0.05). Two dogs had a stable threshold for 110 weeks. Both systems can be easily inserted, using a minor subxiphoid approach, as performed in the pigs.

**References**

Effects of Sudden Change in Cycle Length on Human Atrial, Atrioventricular Nodal and Ventricular Refractory Periods

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SUMMARY In the steady state, the refractory periods of the human atrium, atrioventricular (AV) node, and ventricle are a function of cycle length. We compared the change in refractoriness that occurred when these refractory periods were measured after eight beats at a shorter cycle length with the change that occurred when these refractory periods were measured after a single beat at the shorter cycle length. For a decrease in cycle length of 235 ± 63 msec, the atrial effective refractory period shortened 31 ± 24 msec (p < 0.01) when measured after eight beats at the shorter cycle length and 26 ± 24 msec (p < 0.01) when measured after a single beat at the shorter cycle length. Similar changes were seen in atrial functional refractory period. For a decrease in cycle length of 214 ± 63 msec, the AV nodal effective refractory period increased 30 ± 39 msec (p < 0.05) when measured after eight beats and 31 ± 34 msec (p < 0.05) when measured after a single beat. The AV nodal functional refractory period showed moderate shortening with decreases in cycle length, both when measured after eight beats and when measured after a single beat (p = NS). For both the atrium and AV node, there was no significant difference between the change in refractoriness after a single beat at the shorter cycle length and after eight beats at the shorter cycle length. For a decrease in cycle length of 175 ± 52 msec, the ventricular effective refractory period shortened 26 ± 10 msec (p < 0.01) when measured after eight beats and 16 ± 12 msec (p < 0.01) when measured after a single beat at the shorter cycle length. Thus, a single beat at the shorter interval produced 60% of the shortening of refractoriness produced by eight beats at the shorter interval (p < 0.01). These findings have implications for the performance and interpretation of stimulation studies and provide insight into the mechanism of initiation of tachycardia by premature beats.

REFRACTORYNESS of cardiac tissue is a function of cycle length. This relationship, first recognized in isolated heart tissue, has subsequently been shown in both the canine and human atrium, atrioventricular (AV) node and ventricles.1-4 There is some controversy as to whether changes in refractory periods in the canine heart occur after a single beat at the new cycle length or represent a cumulative effect of multiple beats at the new cycle length.5-7 There are no data in humans on whether cycle-length-dependent changes in refractoriness are seen after a single beat at the new cycle length. In this report, we describe the effects of sudden changes in cycle length on the refractory period of the human atrium, AV node and ventricle.

Methods

Patients

There were 23 males and seven females, average age 49.9 ± 19 years. Six patients had atherosclerotic cardiovascular disease, four had cardiomyopathy, two had hypertensive heart disease, one patient had...
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