A Permanent Transvenous Lead System for an Implantable Pacemaker Cardioverter-Defibrillator
Nonthoracotomy Approach to Implantation

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Gerard M. Guiraudon, MD; Colette M. Guiraudon, MD;
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A transvenous lead system for implantable defibrillators would obviate a surgical thoracotomy and reduce the morbidity and mortality associated with implantation. We evaluated the clinical performance of a new nonthoracotomy lead system that included a defibrillation lead in the coronary sinus. At the time of defibrillator implantation, transvenous defibrillation leads were inserted percutaneously through the left subclavian vein into the right ventricular apex (RVA), superior vena cava (SVC), and distal coronary sinus (CS) under fluoroscopic guidance. A subcutaneous patch electrode (SQ) was also available if required. The first single- or dual-pathway electrode configuration that successfully terminated three of four ventricular fibrillation episodes using 18 J or less was implanted. Eleven men and three women aged 39–77 years (60.0±10.1 years) with left ventricular ejection fraction ranging from 16% to 63% (33.4±13.1%) were evaluated. Nine presented with ventricular tachycardia, three had ventricular fibrillation, and two had both. A totally transvenous lead system (RVA/CS/SVC) was implanted in seven patients (50%) with a mean defibrillation threshold of 15.6±2.9 J (10–18 J). Four patients received a partial transvenous lead system (RVA/CS/SQ). An effective nonthoracotomy lead system was not found in three patients; they received epicardial electrodes. After cumulative follow-up of 73 patient-months, nine patients remain alive and free of problems related to the implanted nonthoracotomy leads. One patient died of respiratory failure 3 months after defibrillator implant, and the leads from another patient were removed at 9 months because of bacterial infection. A transvenous lead system that includes a defibrillation lead in the coronary sinus is a safe, reliable, and, at least in the short term, effective nonthoracotomy approach for automatic defibrillator implantation. (Circulation 1992;85:196–204)

Implantation of automatic defibrillators for patients with malignant ventricular tachyarrhythmias generally requires a surgical thoracotomy because of the need to insert one or more epicardial leads. This exposes the patient to increased surgical morbidity and mortality and limits the type of patient eligible for this treatment modality. One recently developed electrode system that could be implanted without a thoracotomy used a transvenous defibrillation catheter coupled to a submuscular patch electrode in the left chest wall to distribute current more uniformly through the myocardium.

We have described an alternative nonthoracotomy triple-electrode system that employs transvenous defibrillation leads only, incorporating a coronary sinus lead as a substitute for the submuscular or subcutaneous chest wall electrode. The initial leads described were prototypes, but they did demonstrate that transvenous coronary sinus defibrillation was feasible and safe. In this report, we describe the clinical performance of this transvenous lead system for defibrillation implanted with a pacemaker cardioverter defibrillator and early results of this new nonthoracotomy approach.

Methods
Patients referred to University Hospital, London, Ontario, for the management of drug-refractory ven-
tricular tachycardia or fibrillation were considered for this study. Patients considered candidates for an implantable pacemaker cardioverter-defibrillator were asked to participate in the study in accordance with the guidelines of the Institutional Review Board for research involving human subjects at the University of Western Ontario, London, Ontario. Informed written and verbal consent was obtained.

**Nonthoracotomy Lead System**

The transvenous defibrillation leads (Figure 1) were designed for implantation with the pacemaker cardioverter-defibrillator (models 7216A and 7217B, Medtronic Inc., Minneapolis, Minn.). The device has been described in detail. It is a multiprogrammable unit capable of delivering graded therapy for ventricular tachycardia (antitachycardia pacing or cardioversion shocks), defibrillatory shocks at energy up to 34 J, and bradycardia pacing. Programming and stored information related to detected tachycardia events and the results of automatic therapy are accessible through bidirectional telemetry.

The 10.5F multipolar lead (model SP2100-6 or 6884, Medtronic Inc.) was designed for placement in the right ventricular apex and stabilized by active fixation. Two models of this lead were available during the course of the study. In both versions, the screw-in tip electrode served as the cathode; they differed only in the electrode used as the sensing anode. The model 6884 lead had a distal ring electrode 5 mm proximal to the tip for bipolar pacing and sensing, whereas the model SP2100-6 lead used the defibrillating coil electrode. The 50-mm-long defibrillation electrode located 22 mm from the tip provided a conductive surface area of 205 mm².

The second lead was a 7.0F monopolar defibrillation lead (model 6881, Medtronic Inc.) with a 50-mm coil electrode of 90-mm² surface area. This lead was intended for placement in the coronary sinus or the superior vena cava but possessed no mechanism for active or passive fixation.

A subcutaneous patch electrode with a total conductive surface area of 660 mm² was available for alternative nonthoracotomy lead configurations in combination with one or more transvenous leads in the event that a totally transvenous lead configuration could not be implanted.

**Intraoperative Evaluation Protocol**

The transvenous lead system and generator were tested and implanted in the operating room under fluoroscopic guidance. All antiarrhythmic drugs were discontinued at least 5 half-lives before defibrillator implantation except for amiodarone, which was discontinued at least 4 weeks before evaluation. The transvenous defibrillation leads were introduced percutaneously through the left subclavian vein using three separate punctures. The coronary sinus lead was inserted first, followed by the right ventricular lead. The third lead was positioned with the tip at the right atrial/superior vena cava junction (Figure 2). Adequate positioning of the right ventricular catheter was confirmed by measuring pacing threshold values of the bipolar pace/sense electrodes. Coronary sinus lead position was accepted if the entire coil electrode was within the coronary sinus, but optimally, the catheter tip was advanced to the left margin of the cardiac silhouette under the left atrial appendage. Measurement of shock pathway impedance by a 0.6-J test shock helped to confirm adequacy of lead positions.

A standard 6.0F multipolar catheter was inserted through the right internal jugular vein and used for induction of ventricular fibrillation. It was positioned in the right ventricle distant from the permanent defibrillation lead. An adhesive cutaneous defibrillation pad electrode (R2 pad™, Darox Corp., Niles, Ill.) was applied to the left chest wall at the anterior axillary line, centered at the fifth to sixth intercostal space, to simulate the subcutaneous patch electrode during testing. A pair of cutaneous adhesive defibrillation electrodes (R2 pad) were applied to the chest wall for transthoracic rescue shocks (200–360 J).

Because the pacemaker cardioverter-defibrillator generator provided a choice of three pulse waveforms (single, bidirectional simultaneous, or bidirectional sequential) delivered across two or three electrodes, many shock-pulse and lead configurations could have been evaluated. On the basis of previous studies indicating that dual-current-pathway shocks were superior to single-pathway shocks, three electrodes were implanted in all patients. The electrode configurations evaluated during surgery in order from the highest to lowest priority are listed in Table 1. Highest priority was assigned to implanting a totally transvenous lead system, because this obvi-
ated a separate pocket for the subcutaneous patch electrode. No attempt was made to compare defibrillation efficacy of all lead configurations listed. Testing proceeded until an effective lead configuration was identified or all had failed.

The defibrillation leads were interfaced to an external cardioverter-defibrillator unit (ECVD model 5355, Medtronic Inc.) that manually delivered single- or sequential-pulse (4-msec trapezoidal waveform) shocks of equal leading-edge voltage separated by 0.2 msec. Delivered waveform characteristics were monitored and analyzed automatically by the unit, which displayed the delivered peak voltage, pathway impedance, and energy of both pulses. A 0.6-J test shock was delivered to ensure proper functioning of the entire system before any electrode configuration was used for defibrillation testing.

Ventricular fibrillation was induced by 60-Hz AC for 5–10 seconds or rapid ventricular pacing to cycle length of 200–250 msec (Figure 3). When sustained ventricular fibrillation was established for at least 5 seconds, a single transvenous shock of 18 J (stored energy) was delivered, followed by a transthoracic rescue shock if the transvenous shock was unsuccessful. Another lead configuration was then evaluated. If defibrillation was successful, the initial energy for subsequent episodes was reduced by decrements to 15, 10, and 5 J until failure to defibrillate or 5 J was reached. The defibrillation threshold was defined as the lowest stored energy that successfully defibrillated. The tested lead system and defibrillator unit were implanted if three of four episodes of ventricular fibrillation (one failure at 18 J or less was allowed) were successfully defibrillated at 18 J or less. The transvenous leads were firmly anchored in the pectoral fascia and tunneled subcutaneously to the left subcostal region, where they were connected to the generator. Where a subcutaneous patch electrode (model 6895, Medtronic Inc.) was required, the lead was introduced through the left subcostal incision created for the generator and advanced subcutaneously until it was centered directly under the conductive area of the R2 pad used for testing.

After surgery, patients were confined to bed for 24–48 hours and then gradually allowed to walk. Chest radiograms were taken every 2–3 days until discharge to monitor lead stability. Before discharge, patients underwent noninvasive programmed stimu-

**TABLE 1. Electrode Configurations Tested**

<table>
<thead>
<tr>
<th>Configuration</th>
<th>Common cathode (-ve)</th>
<th>Pulse 1 anode (+1)</th>
<th>Pulse 2 anode (+2)</th>
<th>Pulse waveform</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>RV</td>
<td>SVC</td>
<td>CS</td>
<td>Sequential</td>
</tr>
<tr>
<td>2</td>
<td>RV</td>
<td>SVC</td>
<td>SQ</td>
<td>Sequential</td>
</tr>
<tr>
<td>3</td>
<td>RV</td>
<td>CS</td>
<td>SQ</td>
<td>Sequential</td>
</tr>
<tr>
<td>4</td>
<td>CS</td>
<td>RV</td>
<td>SQ</td>
<td>Sequential</td>
</tr>
<tr>
<td>5</td>
<td>RV</td>
<td>CS</td>
<td>...</td>
<td>Single</td>
</tr>
<tr>
<td>6</td>
<td>RV</td>
<td>SVC</td>
<td>...</td>
<td>Single</td>
</tr>
</tbody>
</table>

CS, coronary sinus; RV, right ventricle; SQ, subcutaneous; SVC, superior vena cava.
ulation using the implanted device under neuroleptic anesthesia. Information regarding the efficacy of pacing, cardioversion, and defibrillation therapies was used to program an electrical prescription that best suited the characteristics of each patient's arrhythmias. Patients were seen every 1–3 months.

Data are expressed as the mean±SD unless otherwise indicated. Shock energy reported herein refers to stored energy.

**Results**

Fourteen patients, 11 men and three women, participated in this clinical study (Table 2). The patients ranged in age from 39 to 77 years (60.0±10.1 years). Eleven patients had coronary artery disease with remote myocardial infarction. One patient had concomitant aortic insufficiency with previous prosthetic aortic valve replacement. Two patients had chronic mitral regurgitation and associated mild left ventricular dysfunction not sufficiently severe to warrant valve repair. One patient had a left ventricular lipoma resected 2 years previously without evidence of recurrence at the time of defibrillator implantation. The presenting arrhythmia was recurrent ventricular tachycardia in nine patients; three patients presented with sudden cardiac death and documented ventricular fibrillation, and two patients had both ventricular tachycardia and fibrillation. Left ventricular ejection fraction measured by the radionuclide method ranged from 16% to 63% (33.4±13.1%). A nonthoracotomy approach was favored in eight patients because of previous cardiac surgery, severe pulmo-

**TABLE 2. Patient Characteristics**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Diagnosis and associated conditions</th>
<th>LVEF (%)</th>
<th>Arrhythmia</th>
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</thead>
<tbody>
<tr>
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<td>77</td>
<td>M</td>
<td>IHD, MI, AVR</td>
<td>20</td>
<td>VT</td>
</tr>
<tr>
<td>2</td>
<td>55</td>
<td>F</td>
<td>IHD, MI, Crohn's</td>
<td>30</td>
<td>VF</td>
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<tr>
<td>3</td>
<td>75</td>
<td>M</td>
<td>IHD, MI, COPD</td>
<td>35</td>
<td>VF</td>
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<td>4</td>
<td>65</td>
<td>M</td>
<td>IHD, MI, diabetes</td>
<td>40</td>
<td>VT</td>
</tr>
<tr>
<td>5</td>
<td>50</td>
<td>M</td>
<td>IHD, MI, CABG</td>
<td>20</td>
<td>VT</td>
</tr>
<tr>
<td>6</td>
<td>68</td>
<td>M</td>
<td>IHD, MI</td>
<td>30</td>
<td>VT</td>
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<tr>
<td>7</td>
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<td>M</td>
<td>IHD, MI</td>
<td>25</td>
<td>VF</td>
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<tr>
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<td>63</td>
<td>M</td>
<td>IHD</td>
<td>63</td>
<td>VT</td>
</tr>
<tr>
<td>9</td>
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<td>M</td>
<td>Cardiac lipoma</td>
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<td>VT</td>
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<tr>
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<td>54</td>
<td>F</td>
<td>MVR</td>
<td>42</td>
<td>VT</td>
</tr>
<tr>
<td>11</td>
<td>55</td>
<td>M</td>
<td>IHD, MI, CABG</td>
<td>37</td>
<td>VT/VF</td>
</tr>
<tr>
<td>12</td>
<td>64</td>
<td>M</td>
<td>IHD, MI</td>
<td>22</td>
<td>VT</td>
</tr>
<tr>
<td>13</td>
<td>61</td>
<td>F</td>
<td>MVR</td>
<td>52</td>
<td>VF</td>
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<tr>
<td>14</td>
<td>52</td>
<td>M</td>
<td>IHD, MI, CABG</td>
<td>16</td>
<td>VT</td>
</tr>
</tbody>
</table>

**TABLE 2. Patient Characteristics**

AVR, prosthetic aortic valve replacement; CABG, coronary artery bypass graft surgery; COPD, chronic obstructive pulmonary disease; F, female; IHD, ischemic heart disease; LVEF, left ventricular ejection fraction; M, male; MI, myocardial infarction; MVR, mitral valve regurgitation; VF, ventricular fibrillation; VT, ventricular tachycardia.
nary emphysema, brittle insulin-dependent diabetes mellitus, and Crohn's disease treated with cortico-steroid therapy.

The transvenous defibrillation leads, including the coronary sinus electrode, were positioned without major difficulty in all 14 patients (Table 3). Seven patients met the implantation criteria with lead configuration 1 (totally transvenous lead system). The mean defibrillation threshold was 15.6±2.9 J (10–18 J). In six of these seven patients, the coronary sinus electrode served as the pulse 1 anode; in one patient the superior vena cava lead provided better defibrillation efficacy when serving as the pulse 1 anode. The superior vena cava electrode was replaced by the cutaneous patch electrode (configuration 2, 3, or 4), and four more patients met the implant criteria for a nonthoracotomy lead system with a defibrillation threshold of 10 J (one patient) or 15 J (three patients). In three of these patients, the right ventricular electrode continued to serve as the common cathode, but the coronary sinus electrode was used as the cathode in one patient. Despite trials of up to six single- or dual-current-pathway lead configurations, no consistent effective nonthoracotomy lead configuration could be identified in three patients, and the approach was abandoned. At the time of epicardial patch electrode implantation, no abnormalities were identified on the epicardial surface in the region of the coronary sinus that could be attributed to repeated coronary sinus defibrillation.

The number of lead configurations tested ranged from one to six (3.4±1.6), and ventricular fibrillation was induced from five to 14 times (10.7±2.5) (Table 3). The cumulative stored energy given during intraoperative testing ranged from 71 to 540 J (307.1±122.9 J). The coronary sinus electrode participated in the cumulative delivery of 36–267 J (146.8±63.2 J), with the highest values received by the three patients in whom no nonthoracotomy lead system could be implanted. Despite this, no complications were encountered intraoperatively.

The sensed ventricular electrogram amplitudes during sinus rhythm recorded from the pace/sense electrodes of the right ventricular lead in patients receiving the nonthoracotomy lead system ranged from 9.5 to 28.8 mV (17.3±7.0 mV), with slew rates of 0.8–3.1 V/sec (1.8±0.9 V/sec). The mean pacing threshold using a stimulus of 0.5-msec pulse width was 0.9±0.4 V (0.6–1.8 V), with measured currents of 2.0±0.8 mA (1.1–3.5 mA) and lead impedance of 474.1±84.1 Ω (328–542 Ω).

No complications occurred in any patient during surgery for implantation and testing of the nonthoracotomy lead system. There were no instances of pneumothorax, subclavian artery, thoracic duct, or brachial plexus trauma secondary to subclavian puncture. Within the first 24 hours after implantation, the coronary sinus electrode in only one patient (patient 8) dislodged because of traction on a loosely anchored lead. This patient required a minor surgical procedure to reposition the lead and reconfirm defibrillation efficacy.

The 11 patients given nonthoracotomy lead systems have been followed for 2–13 months with cumulative follow-up of 73 patient-months. One patient (patient 4) with brittle insulin-dependent diabetes mellitus developed a generator pocket infection 9 months after initial implantation. Despite conservative measures, the bacterial infection could not be eradicated, and the generator and nonthoracotomy leads were removed. The coronary sinus and right ventricular leads were explanted by gentle traction on the lead body, and no complications occurred. The patient was placed on amiodarone and
has had no recurrence of ventricular tachycardia after 6 months of follow-up.

A second patient (patient 3) died of respiratory failure secondary to severe chronic emphysema 3 months after implantation of his transvenous defibrillator system. The documented cardiac rhythm at death was ventricular pacing with electromechanical dissociation. Before death, this patient had a total of 16 transvenous shocks, including five for documented rapid atrial fibrillation for a cumulative stored energy of 150 J, of which 74 J were delivered between the right ventricular and coronary sinus electrodes. At autopsy, the coronary sinus defibrillation lead was firmly adherent to the distal wall of the great cardiac vein. The coronary sinus and major venous tributaries were patent. A thin layer of fibrous tissue surrounded the defibrillation coil electrodes in the right ventricle and superior vena cava. No antemortem clot was found on the surface of the transvenous leads or right atrium. There was no evidence of pulmonary embolization. Histological examination showed thin strands of fibrous tissue stretching from the endocardium to the electrode coil (Figure 4). Only one small recent nonocclusive mural thrombus was seen in the coronary sinus. The left circumflex artery adjacent to the coronary sinus electrode showed no fibrosis, hemorrhage, or any acute or chronic changes secondary to coronary sinus defibrillation.

In total, 87 spontaneous tachycardia episodes were detected in four patients and were treated automatically by the pacemaker cardioverter-defibrillator. The device detected 85 episodes as ventricular tachycardia and two, according to the rate criteria, as ventricular fibrillation. Five episodes treated by the device as ventricular tachycardia were documented to have been rapid atrial fibrillation (patient 3) and resulted in five shocks of 5 J each. Although uncomfortable for the patient, the shocks did not have any serious clinical consequences, and further inappropriate shocks were prevented by drug therapy. Seventy-seven episodes of ventricular tachycardia (90.6%) were treated first by antiarrhythmic pacing sequences, which were successful 76 times (98.7%). Three episodes terminated spontaneously, and automatic therapy was aborted. The two episodes of ventricular fibrillation were both terminated by 34-J shocks. None of the patients have suffered any complications secondary to the presence of a lead in the coronary sinus or from the delivery of repeated low-energy coronary sinus shocks. No instances of late transvenous lead dislodgement or lead malfunction have been seen on follow-up. Antiarrhythmic drugs were discontinued in all patients after defibrillator implantation, but adjunctive pharmacological therapy was eventually reinstated in three patients to suppress frequently recurring ventricular tachyarrhythmias.

**Discussion**

The use of transvenous defibrillation catheters was pioneered by a number of investigators before and after the introduction of the automatic implantable defibrillator, but the limited efficacy of such systems prevented their wider clinical use. When the automatic defibrillator was first introduced for patient implantation, the initial lead system was a spring coil lead in the superior vena cava combined with an epicardial cup/patch electrode. This was supplanted by the epicardial patch/patch electrode configuration, which had demonstrated superior defibrillation efficacy and avoided problems such as lead migration. Implantation of epicardial electrodes requires a thoracotomy and is associated with a significant surgical mortality reported to be less than 5%. Long-term morbidity must also be considered; there are concerns that the fibrous pericardial reaction incited by large epicardial electrodes might lead to constrictive pericarditis or difficulty with subsequent transthoracic defibrillation. Bacterial infections can easily spread from the generator pocket along the leads directly to the heart, which necessitates the removal of the entire defibrillator system and makes subsequent reimplantation particularly difficult. Subsequent cardiac surgery such as coronary artery bypass graft or valve replacement is rendered increas-
ingly hazardous should it become necessary at a later date.

The principal challenge has been to design nonthoracotomy electrodes with acceptable defibrillation efficacy for the majority of candidates requiring an implantable defibrillator. In 1989, Saksema et al. reported the implantation of a nonthoracotomy lead system with an automatic defibrillator. The components were a transvenous lead with two defibrillation coil electrodes (located in the right ventricle and right atrium) and a submuscular patch in the left chest wall, which could be used in a variety of electrode combinations to deliver single- or dual-current-pathway shocks.

The new nonthoracotomy lead system described in this report differs in that it incorporates a separate electrode in the coronary sinus–great cardiac vein in lieu of a submuscular or subcutaneous patch electrode to distribute shock current across the left ventricular myocardium. This eliminates the need for a separate chest wall incision and reduces the risk of complications. Eleven patients (79%) received a nonthoracotomy lead system with acceptable defibrillation threshold values, and seven of these patients received a totally transvenous lead system. Even in the four patients who required the subcutaneous patch, the coronary sinus electrode was essential in achieving sufficient defibrillation efficacy. We identified only one patient in whom single-pathway defibrillation efficacy without the coronary sinus electrode was superior to any configuration using the coronary sinus electrode. Because other lead configurations were not evaluated if one involving a coronary sinus electrode was acceptable for implantation, a comparison of the defibrillation efficacy of the various lead configurations was not performed; it is possible that a lead configuration not involving the coronary sinus may be superior in individual patients.

There are several potential areas of concern related to the implantation of a coronary sinus lead for defibrillation. First, DC shocks delivered in the coronary sinus during catheter ablation of accessory atrioventricular pathways have resulted in rupture of the vessel, and defibrillation shocks could cause similar traumatic injury. This possibility is remote because the surface area of the coronary sinus electrode is nearly 10 times that of standard electrodes used for catheter ablation, and the energy, current, and voltage delivered are substantially less. In the extreme case of a maximum stored energy shock of 34 J (corresponding leading-edge current is about 12 A), the maximum current density would be approximately 130 mA/mm². In previous studies involving 53 patients receiving coronary sinus defibrillation shocks, none suffered an acute complication such as coronary sinus rupture or thrombosis, and there was no clinical evidence to suggest injury to the adjacent circumflex coronary artery as a result of repeated shocks. The pathological data obtained from the patient who died provided further critical evidence. Despite having received a total of 105 J during intraoperative testing and five shocks during follow-up, this patient showed no pathological evidence of damage to the circumflex vessel wall. The lead was firmly adherent to the vessel, indicating that a stable position was attained within 3 months and that there was no significant cumulative injury to the coronary vessels during that time.

Second, the presence of a lead with an exposed metal defibrillation coil might promote thrombotic occlusion of the coronary sinus. The use of the coronary sinus as a site for permanent leads is not new, although its involvement as a defibrillation pathway is a recent development. Before the introduction of leads for chronic atrial pacing with a fixation mechanism, permanent pacing leads were infrequently inserted into the coronary sinus if a stable position in the right atrium was not attainable. Histological evidence obtained from the patient who died showed minimal thrombus formation over the defibrillation coil and mild fibrotic reaction without occlusion of the coronary sinus after 3 months. This would suggest that thrombotic occlusion is not a major problem.

While coronary venous obstruction remains a possibility, a deleterious effect upon left ventricular function is by no means inevitable. Recently, McLellan et al. showed that the hemodynamic effects of coronary sinus occlusion were dependent upon the site of obstruction, the degree of occlusion, and the rate at which occlusion progresses. Proximal total occlusion near the orifice by ligation resulted in profound interstitial edema with rapid deterioration of ventricular contractility and, invariably, death of the animals. Partial occlusion of the coronary sinus or total occlusion of the great cardiac vein but sparing the middle cardiac vein caused substantially less edema and left ventricular dysfunction with improved probability of survival, probably because of venous drainage through collateral vessels, including the Thebesian veins.

Third, the smaller electrode surface areas of these transvenous leads might reduce defibrillation efficacy. This could reduce the proportion of patients in whom transvenous defibrillation lead systems would be sufficiently efficacious to be implanted. Newer developments such as the use of biphasic shock waveforms may offset this effect. In a recent study using temporary leads similar in design to those permanently implanted, we estimated that up to 60% of patients tested may be suitable candidates for a totally transvenous lead system incorporating a coronary sinus lead. A further increment in the number of patients able to receive a nonthoracotomy lead system was possible if a subcutaneous patch electrode was also available for alternative nonthoracotomy lead configurations. Although a significant proportion of patients will continue to require epicardial lead systems because nonthoracotomy leads do not provide adequate defibrillation efficacy, the availability of nonthoracotomy leads for a substantial number
of patients requiring defibrillators represents a significant advance.

Finally, the viability of any transvenous lead system also depends on lead stability. Only the right ventricular lead had active fixation, yet neither the superior vena cava nor the coronary sinus leads showed any tendency toward migration or dislodgement. The single case of the coronary sinus electrode dislodgement resulted from excessive traction on a loosely anchored lead and was easily corrected. Dislodgement would not appear to be a major problem beyond the immediate postoperative period. The histological data available in the one patient who died suggested that firm adherence to the coronary sinus was achieved within 3 months, yet the fibrous reaction was not so pronounced as to impede removal of the nonthoracotomy leads in the patient who developed a late infection. Although the need for some active or passive fixation mechanism for these leads would appear to be limited, the serious consequences that could result from lead instability after implantation argue strongly for continued investigation in this area.

Implantation of the nonthoracotomy lead system was based on evaluation at surgery of defibrillation threshold in the same manner as for epicardial lead implantation. The long-term durability, reliability, temporal stability of defibrillation threshold values, and pacing and sensing characteristics of the right ventricular electrodes require longer follow-up. In a recent study, Bardy et al.\(^2\) reported marked fluctuations in defibrillation threshold during a 3-month period following implantation. They found increases in defibrillation threshold energy of up to \(204\%\) and recommended that devices be programmed on follow-up to deliver energy twice that of the defibrillation threshold. By allowing the lead system to be implanted only if the defibrillation threshold was \(18\) J or less, we ensured a safety margin of approximately \(100\%\), which should allow for any chronic change in defibrillation energy requirements.

We arbitrarily prioritized a series of lead configurations to be implanted and tested in this limited number of initial patients, and it remains to be determined which lead configuration will be most applicable to a broader range of patients. The purpose of this study was not to compare the defibrillation efficacies of various possible lead configurations, but rather to assess the clinical performance of the nonthoracotomy leads during testing at surgery and on short-term follow-up. It is probable that no single electrode configuration will be found to be superior to any other and that the optimal lead system will be one that will afford the greatest flexibility and variety of lead configurations.\(^2\) Nonetheless, a transvenous lead system that incorporates an electrode in the coronary sinus for defibrillation is a reliable, safe, and effective nonthoracotomy approach for automatic defibrillator implantation.

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