Transvenous Defibrillation in Humans via the
Coronary Sinus

Gust H. Bardy, MD, Margaret D. Allen, MD, Rahul Mehra, PhD, George Johnson, BSEE,
Shlomo Feldman, MD, H. Leon Greene, MD, and Tom D. Ivey, MD

A consistently effective transvenous defibrillation system for use in automatic defibrillators
could significantly alter the approach to patients at risk of sudden death. Transvenous
defibrillation systems that use a right ventricular (RV) electrode only or an RV electrode in
combination with a chest patch are relatively inefficient at applying current to the posterolat-
eral left ventricle. An RV electrode combined with a coronary sinus (CS) electrode, however,
may improve current distribution to the posterolateral left ventricle. The purpose of this
investigation, therefore, was to evaluate the effectiveness and safety of a specially designed
transvenous lead system with a CS electrode capable of current delivery to this relatively
inaccessible region of the heart. In 20 survivors of cardiac arrest, we determined defibrillation
efficacy immediately before defibrillator surgery for monophasic pulses delivered between an
RV catheter electrode and a CS catheter electrode system and compared these findings with an
RV catheter electrode–thoracic patch defibrillation system. Subsequently, we referenced the
efficacy of both transvenous systems to an epicardial patch electrode system at the time of
defibrillator implantation. The mean delivered-energy defibrillation threshold for the CS-RV
electrode system was 17.5±7.9 J, which was substantially lower than the RV electrode–thoracic
patch system (25.6±11.4 J, p=0.0016 [46% more]). Defibrillation threshold voltage was
529±123 V for the CS-RV electrode system and 647±164 V (22% more) for the RV
electrode–thoracic patch system (p=0.0013). The delivered energy and voltage required for
defibrillation with epicardial patch–patch electrodes were substantially lower than with either
transvenous systems: 6.0±3.6 J (p<0.00001) and 304±97 V (p<0.00001), respectively. The
CS-RV electrode system would have defibrillated 18 of the 20 patients (90%) when used with
presently available pulse generators limited to a stored energy of 30 J. The RV electrode–
thoracic patch system would have defibrillated only 12 of the 20 patients (60%) given the same
30-J limit. Furthermore, the transvenous CS-RV electrode system provided, on average, an 8 J
wider defibrillation safety margin. Thus, the use of a CS electrode may be effective in the use of
transvenous defibrillators. (Circulation 1990;81:1252–1259)

The need for thoracic surgery to insert an
automatic defibrillator limits the usefulness
of such devices in the prevention of sudden
death. A consistently effective transvenous defibrillation
system could significantly alter the role of implant-
able antiarrhythmia devices. For example, a trans-
venous defibrillation system could be more easily
implemented for prevention of sudden death in high-
risk patients who have yet to manifest ventricular
tachycardia or ventricular fibrillation as well as in those
patients who have. However, before such opportunities
arise, substantial improvements must be made in the
effectiveness of transvenous defibrillators.

The concept of transvenous defibrillation is not
new. In 1954, Hopps and Bigelow investigated the
possibility of achieving ventricular defibrillation with
a shock delivered between a catheter pacing elec-
trode and an electrode on the thorax in a dog. Other
animal studies followed that eventually led to the use
of transvenous countershock in humans first by Jain et
al for termination of atrial fibrillation and then by
Mirowski et al for termination of ventricular fibril-
atation. Although transvenous lead systems have
already been used in humans for defibrillation, the
percentage of patients amenable to any particular
transvenous approach for use with implantable

From the Division of Cardiology, Department of Medicine, and
the Division of Cardiothoracic Surgery, Department of Surgery,
University of Washington, Seattle, Washington.

Supported in part by grants R23-HL-36170-03 and R01-
HL-31472-01A3 from the National Institutes of Health, by a grant
from the American Heart Association, Washington Affiliate, and
by a grant from the Northwest Tachycardia Research Foundation.

Address for correspondence: Gust H. Bardy, MD, Division of
Cardiology (Mail Stop RG-22), University of Washington Medical
Center, Seattle, WA 98195.

Received June 21, 1989; revision accepted December 6, 1989.
Defibrillators is not known. Defibrillation has often required high energies to restore sinus rhythm in these studies, perhaps secondary to the difficulty of applying high enough current densities to the posterolateral and basilar left ventricle (LV) with a right heart catheter system. The purpose of this investigation is to advance the effectiveness of transvenous defibrillation by examining a new system in humans that uses the coronary sinus and to compare this system with presently available alternatives.

**Methods**

After giving informed consent, 20 survivors of cardiac arrest underwent preimplant comparison of two different transvenous defibrillation lead systems before receiving a standard epicardial lead system for an automatic implantable cardioverter/defibrillator. One transvenous defibrillation system consisted of a right ventricular (RV) catheter electrode (cathode) and a cutaneous patch electrode (anode) (Figure 1). The other transvenous defibrillation system consisted of the RV catheter electrode (anode) and a coronary sinus (CS) catheter electrode (cathode) (Figure 1). The study population of 20 patients was derived from an initial group of 23 patients. Three patients were excluded from the study because of the inability to cannulate the CS.

The RV lead (model 10284, Medtronic, Minneapolis, Minnesota) was inserted into the right internal jugular vein and positioned under fluoroscopic control in the RV apex. The 10.5F lead consisted of three distinct platinum-iridium electrodes: two electrodes for sensing and pacing and one continuous-coil electrode for defibrillation. The cathodal electrode used for sensing and pacing was an active fixation helical screw that also helped maintain lead stability. The anodal electrode used for sensing and pacing was a 3-mm long ring electrode and was 1 cm proximal to the screw electrode. The defibrillation electrode was an uninterrupted uniform coil and was positioned immediately proximal to the pacing and sensing electrodes extending 6 cm in length, typically to the tricuspid anulus. The defibrillation coil surface area was 426 mm². The insulation material for the RV lead was silicone.

The CS lead (model 10285, Medtronic) was inserted into the left subclavian vein and placed in the distal CS. This 6F lead had three platinum-iridium electrodes: one for defibrillation and two for atrial sensing. The defibrillation electrode was a continuous coil 5 cm in length and had a surface area of 283 mm². The defibrillation coil electrode began 1 cm from the lead tip, which was composed of flexible silicone. Two 2-mm ring electrodes separated by 1 cm were used for atrial sensing and lead positioning and were located 6 cm proximal to the distal end of the defibrillation electrode. These atrial sensing electrodes were used to record signals from the proximal CS when the lead tip was positioned properly in the distal CS. Insulation material for the CS lead, like the RV lead, was silicone. The CS lead was always positioned so that the distal end of the defibrillation electrode was adjacent to the left atrial appendage. Anteroposterior, right anterior oblique, and left anterior oblique chest radiographic views of the CS and RV electrodes were obtained in each patient to document lead location (Figure 2).

To simulate a subcutaneous patch electrode, a stainless steel 8-cm diameter disk electrode was placed on the chest wall (Figure 1). This electrode had a surface area of 50.3 cm² and was centered over the left lateral thorax in the anterior axillary line in the fifth intercostal space. Given the temporary nature of the transvenous component of the study, actual implantation of a subcutaneous patch could not be justified ethically. Fortunately, previous work showed that a cutaneous patch approximates well a subcutaneous patch for defibrillation testing, because the transdermal voltage drop during defibrillation is insignificant.⁹

After the leads were in place, defibrillation thresholds were measured. One of the nonthoracotomy defibrillation methods was selected randomly to be tested first. Alternating current (60 Hz) was used to initiate ventricular fibrillation by the RV bipolar pacing electrodes, and defibrillation was attempted 10 seconds after the application of current and the onset of ventricular fibrillation.

The pulse waveform used for both defibrillation methods was a truncated exponentially decaying 120-μF capacitor discharge with a waveform tilt of 65%.
This waveform was chosen for study so that the trial could be referenced to the pulsing methods used with the standard automatic implantable cardioverter/defibrillator. Voltage and current waveforms of the defibrillation pulse were recorded by the use of methods previously reported. Briefly, two differential amplifiers (model AM502, Tektronix) and two digitizing oscilloscopes (model 2230, Tektronix) in combination with an IBM-AT computer enabled on-line waveform storage and analysis for determination of delivered energy and resistance.

The external pulse generator used for the study was a Medtronic model 2394. The initial pulse used for both of the transvenous systems had a 500 V leading-edge voltage (approximately, 15 J stored energy). In the instance where the initial 500-V defibrillation pulse was successful, ventricular fibrillation was reinduced, and defibrillation was attempted with a 400-V pulse. If a voltage of 400 V was successful, decrements in leading-edge voltage were subsequently made in 50-V steps. This process was repeated until the lowest amplitude pulse failed to terminate ventricular fibrillation and the patient required defibrillation with a 200-J transthoracic rescue pulse.

If defibrillation was unsuccessful at 500 V, a rescue pulse was delivered, and ventricular fibrillation was reinduced. Defibrillation was then attempted at 600 V. This process was repeated for a pulse amplitude of 700 V and finally to a maximum of 800 V. A value of 800 V was chosen as our maximum voltage, because the maximum capacitor voltage available with present technology for implantable defibrillators is 800 V. If defibrillation was not possible at 800 V (only seen during RV–thoracic cutaneous patch shock delivery in patients 8, 9, and 11), the defibrillation threshold was arbitrarily and conservatively designated to be 800 V.

At the end of the protocol for evaluating the two nonthoracotomy defibrillation systems, the patient underwent a sternotomy for implantation of the automatic internal cardioverter/defibrillator. In each patient, a uniform defibrillation lead system with two large patch electrodes (model 0041, CPI) was applied over the anterior RV (cathode) and the posterolateral left ventricle (anode). Defibrillation thresholds for the epicardial lead system were then determined with the same pulse waveform and comparable testing methods as used in the two nonthoracotomy defibrillation systems.

Statistical Analysis

Statistical analysis of the transvenous defibrillation data was performed with paired Student’s t tests to compare the two nonthoracotomy electrode configurations with respect to leading-edge voltage, leading-edge current, resistance, and delivered energy at the defibrillation threshold values. In addition, percent efficacy curves were constructed for voltage and delivered energy. Analysis of variance was used to assess the statistical significance of the results of epicardial patch–patch defibrillation in comparison with those from both of the transvenous methods.

Results

The clinical data for the 20 patients studied are summarized in Table 1. The study population consisted of 10 men and 10 women. The mean age was 56±12 years. Five patients had coronary artery disease only, five had a dilated cardiomyopathy only.
(idiopathic or alcoholic), four had hypertrophic cardiomyopathy, two had both coronary artery disease and a cardiomyopathy, one had both coronary artery disease and valvular heart disease, and three had primary electrical disease. The mean ejection fraction was 0.45±0.19. One patient had disopyramide, one had flecainide, and one had amiodarone present in the cardiac tissues at the time of defibrillation testing.

The defibrillation threshold data are detailed in Table 2. Only 12 of the 20 patients (60%) with the RV–thoracic cutaneous patch system were defibrillated within a 30-J stored-energy limit. On the other hand, 18 of the 20 patients (90%) were defibrillated with the CS-RV lead system within the 30-J limit. Furthermore, the mean defibrillation threshold values for the CS-RV defibrillation system were substantially lower than the values with the RV–thoracic cutaneous patch electrode system. The mean leading-edge defibrillation threshold voltage was 529±123 V when the CS-RV electrode system was used and 648±164 V (22% more) when the RV–thoracic cutaneous patch system was used, p=0.0013 (Figure 3). Mean leading-edge defibrillation threshold current was 9.2±2.7 A when the CS-RV electrode system was used and 10.2±3.5 A (11% more) when the RV–thoracic cutaneous patch system was used, p=0.1972. The leading-edge resistance for the CS-RV electrode system was 59±8 Ω and 67±15 Ω (14% more) for the RV–thoracic cutaneous patch system, p=0.0079, reflecting the differences in current pathways. The delivered-energy defibrillation threshold was 17.5±7.9 J when the CS-RV electrode system was used and 25.6±11.4 J (46% more) when the RV–thoracic cutaneous patch system was used, p=0.0016 (Figure 4).

Epicardial defibrillation thresholds with two large patch electrodes were 304±97 V, 7.0±2.7 A, 46±13 Ω, and 6.0±3.6 J, respectively, for leading-edge voltage, leading-edge current, resistance, and delivered energy. Not unexpectedly, these four variables of epicardial defibrillation were significantly less than the same variables for either transvenous system. The p values for the epicardial defibrillation threshold voltage, current, resistance, and delivered energy compared with both transvenous systems were <0.00001, =0.0050, <0.00001, and <0.00001, respectively.

The percent efficacy of epicardial patch defibrillation for delivered energy is shown in reference to the two transvenous methods in Figure 5. At any delivered-energy level, the percent efficacy for defibrillation was greatest for epicardial patch–patch defibrillation, almost as high for transvenous CS–RV defibrillation, and lowest for transvenous RV–thoracic cutaneous patch defibrillation. At 30 J, 100% of patients could be defibrillated with epicar-

### Table 1. Clinical Data

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Underlying structural heart disease</th>
<th>Clinical arrhythmia</th>
<th>LV ejection fraction</th>
<th>Antiarrhythmic drugs present during defibrillation testing</th>
<th>Therapeutic procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 E.A.</td>
<td>72</td>
<td>F</td>
<td>IDC</td>
<td>VF×2</td>
<td>0.28</td>
<td>None</td>
<td>AICD</td>
</tr>
<tr>
<td>2 B.K.</td>
<td>45</td>
<td>F</td>
<td>CAD, DMI, SP MVR</td>
<td>VF</td>
<td>0.49</td>
<td>None</td>
<td>AICD</td>
</tr>
<tr>
<td>3 T.B.</td>
<td>57</td>
<td>M</td>
<td>CAD, AMI</td>
<td>VF</td>
<td>0.18</td>
<td>None</td>
<td>CABG, AICD</td>
</tr>
<tr>
<td>4 H.M.</td>
<td>81</td>
<td>M</td>
<td>PED</td>
<td>VF</td>
<td>0.60</td>
<td>None</td>
<td>AICD</td>
</tr>
<tr>
<td>5 M.V.</td>
<td>52</td>
<td>M</td>
<td>CAD, DMI</td>
<td>VF</td>
<td>0.42</td>
<td>None</td>
<td>CABG, AICD</td>
</tr>
<tr>
<td>6 V.M.</td>
<td>46</td>
<td>F</td>
<td>IDC</td>
<td>VF</td>
<td>0.33</td>
<td>None</td>
<td>AICD</td>
</tr>
<tr>
<td>7 L.O.</td>
<td>54</td>
<td>F</td>
<td>IDC</td>
<td>VF</td>
<td>0.20</td>
<td>None</td>
<td>AICD</td>
</tr>
<tr>
<td>8 E.W.</td>
<td>47</td>
<td>F</td>
<td>CAD, AMI</td>
<td>VF</td>
<td>0.26</td>
<td>None</td>
<td>AICD</td>
</tr>
<tr>
<td>9 J.S.</td>
<td>67</td>
<td>M</td>
<td>CAD, AMI, IDC</td>
<td>VT</td>
<td>0.18</td>
<td>None</td>
<td>CABG, AICD</td>
</tr>
<tr>
<td>10 J.O.</td>
<td>30</td>
<td>M</td>
<td>HCM</td>
<td>VF</td>
<td>0.85</td>
<td>Disopyramide</td>
<td>AICD</td>
</tr>
<tr>
<td>11 D.S.</td>
<td>49</td>
<td>F</td>
<td>Apical HCM</td>
<td>VF</td>
<td>0.49</td>
<td>None</td>
<td>AICD</td>
</tr>
<tr>
<td>12 D.S.</td>
<td>52</td>
<td>M</td>
<td>PED</td>
<td>VF</td>
<td>0.60</td>
<td>None</td>
<td>AICD</td>
</tr>
<tr>
<td>13 J.E.</td>
<td>51</td>
<td>F</td>
<td>CAD, DMI, ACM</td>
<td>VF</td>
<td>0.34</td>
<td>None</td>
<td>AICD</td>
</tr>
<tr>
<td>14 H.B.</td>
<td>60</td>
<td>F</td>
<td>PED</td>
<td>VF</td>
<td>0.64</td>
<td>Amiodarone</td>
<td>AICD</td>
</tr>
<tr>
<td>15 W.A.</td>
<td>59</td>
<td>M</td>
<td>CAD, DMI</td>
<td>VF</td>
<td>0.59</td>
<td>None</td>
<td>AICD</td>
</tr>
<tr>
<td>16 M.G.</td>
<td>73</td>
<td>F</td>
<td>HCM</td>
<td>VF</td>
<td>0.61</td>
<td>Flecainide</td>
<td>AICD</td>
</tr>
<tr>
<td>17 F.C.</td>
<td>67</td>
<td>M</td>
<td>ACM</td>
<td>VF</td>
<td>0.22</td>
<td>None</td>
<td>AICD</td>
</tr>
<tr>
<td>18 W.H.</td>
<td>50</td>
<td>M</td>
<td>CAD, DMI</td>
<td>VF</td>
<td>0.57</td>
<td>None</td>
<td>CABG, AICD</td>
</tr>
<tr>
<td>19 D.B.</td>
<td>52</td>
<td>M</td>
<td>IDC</td>
<td>VF×2</td>
<td>0.52</td>
<td>None</td>
<td>AICD</td>
</tr>
<tr>
<td>20 W.T.</td>
<td>65</td>
<td>M</td>
<td>HCM</td>
<td>VF</td>
<td>0.67</td>
<td>None</td>
<td>AICD</td>
</tr>
</tbody>
</table>

Mean±SD 56±12 0.45±0.19

ACM, alcoholic cardiomyopathy; AICD, automatic implantable cardioverter/defibrillator; AMI, anterior myocardial infarction; CABG, coronary artery bypass grafting; CAD, coronary artery disease; EF, ejection fraction; F, female; DMI, diaphragmatic myocardial infarction; HCM, hypertrophic cardiomyopathy; LV, left ventricular; M, male; MVR, mitral valve replacement; PED, primary electrical disease; SP, status post; VF, ventricular fibrillation; VT, ventricular tachycardia.
TABLE 2. Defibrillation Threshold Data

<table>
<thead>
<tr>
<th>Patient</th>
<th>Voltage (V)</th>
<th>Current (A)</th>
<th>Resistance (Ω)</th>
<th>Delivered energy (J)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RV-CP</td>
<td>RV-CS</td>
<td>RV-CP</td>
<td>RV-CS</td>
</tr>
<tr>
<td>1</td>
<td>E.A.</td>
<td>676</td>
<td>619</td>
<td>15.6</td>
</tr>
<tr>
<td>2</td>
<td>B.K.</td>
<td>547</td>
<td>355</td>
<td>9.7</td>
</tr>
<tr>
<td>3</td>
<td>T.B.</td>
<td>700</td>
<td>579</td>
<td>8.9</td>
</tr>
<tr>
<td>4</td>
<td>H.M.</td>
<td>399</td>
<td>487</td>
<td>4.9</td>
</tr>
<tr>
<td>5</td>
<td>M.V.</td>
<td>686</td>
<td>486</td>
<td>10.5</td>
</tr>
<tr>
<td>6</td>
<td>V.M.</td>
<td>366</td>
<td>478</td>
<td>7.7</td>
</tr>
<tr>
<td>7</td>
<td>L.O.</td>
<td>378</td>
<td>346</td>
<td>6.9</td>
</tr>
<tr>
<td>8</td>
<td>E.W.</td>
<td>800</td>
<td>714</td>
<td>17.5</td>
</tr>
<tr>
<td>9</td>
<td>J.S.</td>
<td>800</td>
<td>670</td>
<td>12.3</td>
</tr>
<tr>
<td>10</td>
<td>J.O.</td>
<td>500</td>
<td>339</td>
<td>6.4</td>
</tr>
<tr>
<td>11</td>
<td>D.S.</td>
<td>800</td>
<td>379</td>
<td>15.1</td>
</tr>
<tr>
<td>12</td>
<td>D.S.</td>
<td>677</td>
<td>567</td>
<td>11.2</td>
</tr>
<tr>
<td>13</td>
<td>J.E.</td>
<td>493</td>
<td>658</td>
<td>6.3</td>
</tr>
<tr>
<td>14</td>
<td>H.B.</td>
<td>850</td>
<td>473</td>
<td>11.8</td>
</tr>
<tr>
<td>15</td>
<td>W.A.</td>
<td>628</td>
<td>528</td>
<td>9.4</td>
</tr>
<tr>
<td>16</td>
<td>M.G.</td>
<td>624</td>
<td>529</td>
<td>9.7</td>
</tr>
<tr>
<td>17</td>
<td>F.C.</td>
<td>856</td>
<td>732</td>
<td>8.5</td>
</tr>
<tr>
<td>18</td>
<td>W.H.</td>
<td>822</td>
<td>620</td>
<td>13.1</td>
</tr>
<tr>
<td>19</td>
<td>D.B.</td>
<td>826</td>
<td>621</td>
<td>13.1</td>
</tr>
<tr>
<td>20</td>
<td>W.T.</td>
<td>524</td>
<td>403</td>
<td>5.6</td>
</tr>
</tbody>
</table>

Mean±SD  648±164  529±123  10.2±3.5  9.2±2.7  67±15  59±8  25.6±11.4  17.5±7.9

p=0.0013  p=0.1936  p=0.0084  p=0.0016

CS, coronary sinus; RV, right ventricular; CP, chest patch.

Voltage is measured leading-edge volume. Current is measured leading-edge current. Resistance is calculated from leading-edge voltage and current.

Discrepancies between actual leading-edge voltage settings and measured leading-edge voltages are a function of patient resistance, of component tolerances, and of internal resistances found in the pulse generator used in this study.

dial patch electrodes, 90% with the transvenous CS-RV electrode system, and 60% with the transvenous RV–thoracic cutaneous patch system.

No patient suffered a complication from the study. In each instance, the CS was visually examined after sternotomy and found to have no evidence of burn or thrombosis.

Discussion

The history of transvenous, nonthoracotomy cardioversion and defibrillation encompasses a variety of alternative approaches. Most of the initial studies of transvenous defibrillation explored an RV–superior vena caval electrode system, a right atrial–chest wall electrode system, or an RV–chest wall electrode system. Only one of these three transvenous systems has been used in humans: the RV–superior vena caval approach. Of the human studies that used an RV–superior vena caval system, none has recorded data on defibrillation threshold or percent efficacy sufficient enough to allow comparison with our findings. Review of the patients treated...
in these previous studies, however, suggests that many patients required energies greater than 30 J to cause defibrillation. Thus, although the RV—superior vena caval electrode system has worked in some patients, whether it can be safely applied to most patients needing implantable defibrillators is unclear.

More recently, efforts were made to improve on these earlier transvenous lead systems. In one method, Sakse na et al.17,22–23 used an RV—superior vena caval electrode system in combination with a left anterior thoracic patch electrode in an attempt to develop an effective nontoracotomy system. With this approach, the superior vena caval and thoracic patch electrodes are connected in parallel at the anode, and a single monophasic pulse is delivered to the cathodal RV electrode. This system has been used primarily for ventricular tachycardia cardioversion and appears to be effective. Defibrillation efficacy, however, is less clearly demonstrated, although preliminary results are promising. At this time, it is uncertain what percentage of patients will be effectively defibrillated with this method within a reasonable safety margin.

The role of the CS for countershock of ventricular arrhythmias was first examined by Jackman and Zipes.16 In their canine study, a 10F electrode was inserted into the CS and used in conjunction with an RV electrode. Cardioversion efficacy of ventricular tachycardia with this system was then compared with cardioversion efficacy for shocks delivered between an RV electrode and a superior vena caval electrode. The results of the study showed no differences between these two methods in cardioversion efficacy of ventricular tachycardia. Ventricular defibrillation was not examined. The failure to demonstrate any improved cardioversion efficacy with a CS electrode in that study may be a consequence of three factors: the differences between ventricular tachycardia and ventricular fibrillation, the anatomic differences between humans and dogs, and the differences in the electrode systems.

Kallok and Maraccini24 have also examined the role of a CS electrode on arrhythmia termination in dogs. In their study, defibrillation efficacy rather than cardioversion efficacy was investigated with single-pulse and sequential-pulse defibrillation. Single-pulse defibrillation was conducted through an RV apical and a superior vena caval catheter electrode. The CS electrode’s effect on defibrillation efficacy, on the other hand, was only examined in the context of sequential pulsing where it served as the second anode in a pulse sequence that used the superior vena caval electrode as the first anode and the RV apical electrode as the common cathode. With this method, sequential-pulse defibrillation proved 20% more efficacious than single-pulse defibrillation. What component of the 20% improvement in defibrillation thresholds was secondary to the CS electrode and what component was secondary to the use of sequential pulses is unclear. Nevertheless, this study at least demonstrated the potential clinical usefulness of a CS electrode.

Applying higher current densities to the lateral LV should decrease voltage requirements for transvenous defibrillation systems. Mapping studies of electric fields during defibrillation pulses between an RV and a superior vena cava electrode have shown insufficient defibrillation voltage gradients along the posterolateral and basalar LV freewall with transvenous systems isolated to the right heart.25 With one electrode in the CS, however, a means is provided for applying a higher transmyocardial voltage gradient to the lateral and basalar LV than with those transvenous lead systems using only the right heart.

Although we believe the CS is an effective lead location for transvenous defibrillation, two concerns must be addressed: CS thrombosis and CS perforation. Each heart in our study was visually inspected at the time of epicardial patch placement, and there was no gross evidence of CS thrombosis or perforation, and there was not any evidence of coronary artery, LV, or left atrial injury, hemorrhage, or discoloration. Although none of our 20 patients had evidence of CS occlusion or injury during the procedure, the status of the CS during long-term implantation of a defibrillation lead remains unclear. There is evidence, however, from long-term CS pacing studies in humans that thrombosis and perforation of the CS do not occur even with leads that are considerably larger than the leads used in this study.26–28

We have also performed histologic studies in dogs that had a CS lead implanted for 12 weeks. During the 12-week period, the catheter was used repetitively for defibrillation. At least ten 34-J (or 760-V) shocks were delivered to this electrode at the time of the initial surgery and at weeks 1, 2, 3, 4, 6, 8, and 12 after implantation. In total, more than 80 high-energy shocks were delivered. On histologic examination of these animals, only mild intimal fibrosis was found in each animal on the side of the CS wall adjacent to the electrode. There was no histologic evidence of tissue injury to the coronary arteries, left atrium, or LV. Thus, no significant cardiac injury was observed.
Concerns regarding CS thrombotic occlusion should be balanced against published observations that indicate CS occlusion may actually have a salutary effect. For example, acute occlusion of the CS in dogs has been shown to improve oxygen delivery to LV muscle and to increase the ventricular fibrillation threshold. In addition, even if occlusion or partial occlusion of the CS does occur, sufficient collaterals exist through the thebesian and anterior RV venous systems to offset possible alterations in coronary circulation and to prevent any change in ventricular mechanical function. CS injury is a legitimate concern for long-term follow-up. However, our findings, together with previous work, suggest that transvenous defibrillation with a CS lead system is probably safe.

Although this study demonstrates that the CS lead facilitates defibrillation, it is reasonable to acknowledge that the RV–thoracic cutaneous patch system could have performed better had the chest patch location been different. In choosing the chest patch location at the anterior axillary line in the fifth intercostal space, we hoped to place the chest patch electrode in closest proximity to the LV. It should be recognized that this may not have been the optimal chest patch lead location. However, testing of other chest patch positions, as well as other CS electrode positions, was precluded by the clinical restrictions of repetitive inductions and terminations of ventricular fibrillation. Therefore, the chest patch electrode location chosen was the one believed most likely to result in the highest current density to the LV.

In addition to considering alternative lead positions, one must also consider the effect of electrode polarity on the outcome of the study. We tried to optimize both transvenous systems with respect to polarity by using as the anode the electrode with the largest surface area adjacent to the most myocardium. This decision was based on earlier work suggesting that anodal pulses excite fibrillating, partially refractory myocardium better than do cathodal pulses. Given the RV, CS, and chest patch electrode surface areas, we made the RV electrode the anode for the RV-CS configuration and made the chest patch electrode the anode for the RV–chest patch configuration. Other electrode polarities could have given better or worse results for either lead configuration examined.

In conclusion, our study has demonstrated the feasibility and safety of a transvenous defibrillation system with a CS–RV catheter lead system in cardiac arrest survivors. Compared with an RV–thoracic cutaneous patch lead system for human defibrillation, the CS–RV catheter system may lead to a more consistently reliable nonthoracotomy defibrillation method for use in implantable automatic antitachycardia devices.

References

7. Saksena S, Lindsay BD, Parsonnet V: Developments for future implantable cardioverters and defibrillators. PACE 1987; 10:1342–1358
12. Schuder JC, Stockehl H, West JA, Keskar PY: Relationship between electrode geometry and effectiveness of ventricular defibrillation in the dog with catheter having one electrode in right ventricle and other electrode in superior vena cava, or external jugular vein, or both. Cardiovasc Res 1973; 7:629–637
22. Ciccone JM, Saksena S, Shah Y, Pantopoulos D: A prospective randomized study of the clinical efficacy and safety of


**KEY WORDS**

- transvenous defibrillation
- sudden death
- ventricular fibrillation
- automatic defibrillators
Transvenous defibrillation in humans via the coronary sinus.
G H Bardy, M D Allen, R Mehra, G Johnson, S Feldman, H L Greene and T D Ivey

Circulation. 1990;81:1252-1259
doi: 10.1161/01.CIR.81.4.1252
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1990 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/81/4/1252

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/