Relation Between Transcardiac and Transthoracic Current During Defibrillation in Humans

Bruce B. Lerman and O. Carlton Deale

Conceptually, transthoracic defibrillation threshold current can be considered a function of at least two quantities. It is directly proportional to the transthoracic threshold current and inversely proportional to the transcardiac threshold fraction (\( F_C \)) or the ratio of transcardiac and transthoracic current. Although experimental and theoretical estimates of \( F_C \) have been as high as 45%, previous measurements in humans have not been made. This study was designed to quantify \( F_C \) in humans. During intraoperative testing of the automatic implantable cardioverter defibrillator, transthoracic rescue shocks of 200–400 J were delivered when the device failed to defibrillate. Simultaneous transthoracic voltage (\( V_T \)) and transcardiac voltage (\( V_C \)) were measured. The ratio, \( V_C/V_T \), was 0.04±0.03 (mean±SD) in 10 patients. In 16 dogs, a comparison was made between direct measurement of \( F_C \) and \( V_C/V_T \). \( F_C \) was determined with a specially designed electrode system, which was calibrated to account for field distortion introduced by the electrodes. There was no significant difference between \( F_C \) and \( V_C/V_T \), which were both approximately 0.05, suggesting that \( V_C/V_T \) was statistically equivalent to \( F_C \). The results of this study, therefore, indicate that during transthoracic defibrillation in humans, approximately 4% of transthoracic current traverses the heart. This relatively small percentage of current results from the existence of parallel pathways, such as the thoracic cage and lungs, which shunt current around the heart. (Circulation Research 1990;67:1420–1426)

Transcardiac defibrillation is dependent on both threshold myocardial current density and the field distribution throughout the remainder of the thorax. Therefore, the transcardiac current fraction (\( F_C \)), defined as the ratio of transcardiac threshold current (\( I_C \)) to transthoracic defibrillation threshold current (\( I_T \)), is one of the determinants of transthoracic defibrillation threshold.

Although measurements of \( F_C \) in humans have not been made, previous experimental and theoretical estimates have been between 3% and 45%.1–6 The reliability of these data is questionable, however, because of methodological considerations. For example, previous approaches have included comparisons of mean transcardiac to mean transthoracic current thresholds from different groups of subjects,1 comparisons of these two quantities in the same animals, with shocks delivered from both epicardial and transthoracic electrodes,2,3 direct measurement of myocardial current by a ring transformer during delivery of alternating current from electrodes positioned on the extremities4,5 and theoretical calculations based on field theory and a homogeneous slab torso model.6

The present study was designed to quantify \( F_C \) in humans. Measurements were made during rescue transthoracic shocks delivered during intraoperative testing of the automatic cardioverter defibrillator.

Methods

Rationale

This study was designed to quantify \( F_C \) in humans. For ethical reasons, \( F_C \) could not be measured directly in humans, because this would have required the implantation of experimental electrodes to measure transcardiac current and the delivery of unnecessary shocks. However, during intraoperative implantation and testing of the automatic cardioverter defibrillator, the voltage between the epicardial patch electrodes (transcardiac voltage, \( V_C \)) was mea-
sured simultaneously with the transthoracic voltage \( V_T \) (measured between transthoracic electrodes) when transthoracic rescue shocks were required. To determine the relation of the ratio \( V_C/V_T \) and \( F_C \), these quantities were directly compared in a separate protocol performed in dogs.

**Experimental Protocol**

**General.** All studies were performed in accordance with the guidelines of the American Physiological Society. Mongrel dogs weighing 20–30 kg were anesthetized with sodium pentobarbital, 30 mg/kg, and maintained with supplemental doses. The animals were ventilated with a respirator (Harvard Apparatus, South Natick, Mass.), and electrocardiographic surface lead II and arterial pressure (Statham Instruments, Inc., Oxnard, Calif.) were continuously monitored on an electrostatic recorder (model ES-1000, Gould, Houston). Arterial blood gases were monitored at 30-minute intervals.

Transthoracic shocks were delivered with a commercially available defibrillator (Lifepak 6, Physio-Control Corp., Redmond, Wash.). As previously described,7 the defibrillator was calibrated in units of current and was discharged through a precision control system that regulated electrode force. The electrodes were positioned over the right and left lateral chest walls at the tranverse level of the heart. Electrode area (60 cm², circular stainless steel) and force (50 N) were held constant. A fresh, thin film of electrode paste (Redux Paste, Hewlett-Packard Co., Palo Alto, Calif.) covered the surfaces of the electrodes and was maintained throughout the protocol. The endotracheal tube was clamped at peak inspiration before the system was balanced and the shock was delivered.

\( V_T \) was measured with a 1,000:1 voltage divider in parallel with the defibrillator output, and delivered current was measured with a 0.10-Ω resistor in series with the defibrillator output. Voltage and current waveforms were displayed on a triggered-sweep storage oscilloscope (model 5113, Tektronix, Beaverton, Ore.).

**Protocol 1: Determination of the transtcaric current fraction.** To measure transtcaric current during transthoracic shocks, a median sternotomy was performed and the pericardium was reflected. A pair of two-sided rectangular foil electrodes (7×7 cm) were molded around virtually the entire epicardial surface and anchored to the posterior thoracic musculature (Figure 1). Each pair of electrodes was insulated with polyethylene, and from each set of electrodes, twisted-pair signal cables were connected to an external circuit for measuring transtcaric current. The pericardium was then closed, and the chest was tightly apposed with wire sutures. The cables from the electrodes were externalized through the median sternotomy. One transthoracic shock (10–30 A) was delivered in sinus rhythm, and \( V_T \) and transthoracic current were measured.

![Figure 1. Foil electrode (one of two) used to measure cardiac signals during transthoracic shocks.](image)

Transmyocardial current was measured by developing voltage signals across 0.1-Ω resistors with the switching circuit illustrated in Figure 2. During each transthoracic shock, the left and right switches were closed, and the short circuit switch was opened. Transthoracic current entered the outer left epicardial (or heart) electrode (\( I_{HR} \)); passed through the left 0.1-Ω resistor, the left switch, and the inner left epicardial electrode; traversed the heart; entered the right inner epicardial electrode through the right switch and the right 0.1-Ω resistor; and finally exited via the outer right epicardial electrode (\( I_{HR} \)).

Because the presence of the foil electrodes distorts the field in the myocardium, a calibration protocol, which has been described in detail,8 was performed. Briefly, by varying dose strength during transthoracic shocks, current measured with a single foil electrode on one side of the heart was compared with current measured by the same electrode when the field was perturbed by the presence of an additional electrode on the opposite side of the heart. The slope of the resultant linear regression equation obtained from the calibration protocol (Figure 3) was equivalent to the correction factor for a single electrode, 0.937, and as has been previously derived,8 the correction factor for a pair of foil electrodes is the square of the slope, or 0.878. In other words, the current measured by the electrodes was larger than the true current value. The corrected transtcaric current was calculated as 0.878 \( (I_{HR}+I_{HR})/2 \), and \( F_C \) was calculated by the ratio of transtcaric current transthoracic current.

**Protocol 2: Determination of transtcaric voltage transthoracic voltage \( V_C/V_T \) during transthoracic shocks (small epicardial patch electrodes).** Since during the clinical protocol only \( V_C/V_T \) could be measured (see below), protocols 2 and 3 were designed to
determine whether $V_C/V_T$ determined over a series of dogs was equivalent to $F_C$.

Foil electrodes were removed from each dog, two small epicardial patch electrodes were implanted, and the pericardium and sternum were closed. The patch electrodes consist of titanium mesh, with the outer surface insulated with silicone rubber (model 0040, Cardiac Pacemakers, Inc., St. Paul). The electrode surface area is 14 cm$^2$. The patch electrodes were sutured to the anterior right and left posterolateral ventricular surfaces and were oriented longitudinal to the long axis of the heart. After the sternum was closed, one sinus rhythm shock was delivered (at the current dose selected earlier in protocol 1) and $V_C$ and $V_T$ were measured. $V_C$ was measured between the inner left and right epicardial electrodes. As shown in Figure 2, the left and right switches were closed, and the short circuit switch was opened to measure $V_C$ with a 1,000:1 voltage divider.

Protocol 3: Determination of $V_C/V_T$ (large epicardial patch electrodes). To assess the effect of epicardial electrode size on $V_C/V_T$, measurements were also made with large patch electrodes. The small epicardial electrodes were removed, large epicardial patch electrodes (total surface area 28 cm$^2$, model 0041, Cardiac Pacemakers) were implanted on the right and left ventricular epicardial surfaces, and the pericardium and chest were closed. $V_C$ and $V_T$ were determined as in protocol 2.

Clinical Protocol: Determination of $V_C/V_T$ During Transthoracic Defibrillation

Measurements were made during transthoracic rescue shocks during intraoperative implantation of epicardial patch electrodes and the automatic implantable defibrillator in 10 patients. Epicardial patch electrodes were sutured to the right anterior and left posterolateral ventricular surfaces through a small left thoracotomy. Transthoracic self-adhesive electrodes (R2 Corp., Skokie, Ill.) were positioned at the second right intercostal space along the right sternal border and at the fifth intercostal space adjacent to the apex. The epicardial patch electrodes were connected to an external cardioverter defibrillator (model 2800, Cardiac Pacemakers). For routine intraoperative transthoracic threshold testing, the chest retractor was removed first, ventricular fibrillation was induced with alternating current, and shocks of incremental energy were delivered until the heart

![Figure 2. Voltage divider and switching circuit for measuring transcardiac voltage and current. The left and right switches ($S_L$ and $S_R$, respectively) were closed, and the short circuit switch ($S_{SC}$) was opened. Transcardiac current flowed through the left and right epicardial (or heart) foil electrodes ($I_{HL}$ and $I_{HR}$, respectively) and developed voltage signals ($V_{HL}$ and $V_{HR}$) across the 0.1-$\Omega$ resistors. Transcardiac voltage appeared between the inner left and right epicardial electrodes and developed a signal $V_{HR}$ at the output of a 1,000:1 voltage divider. $R_H$, heart resistance.](image)

![Figure 3. Effect of the foil electrodes on the measured transcardiac current. Current measured with a single foil electrode ($I_E$) was compared with the current measured with the same electrode when the field was perturbed by an electrode on the opposite side of the heart ($I_E$). The slope of the regression equation quantified the effect that the second foil electrode had on the current measured by the first foil electrode.](image)
was successfully defibrillated. If a 25-J shock failed to defibrillate the heart, a transthoracic rescue shock of 200–400 J was delivered from an unmodified damped sine wave defibrillator (Lifepak 6). Simultaneous Vc between the epicardial patch electrodes and VT were measured during the rescue shock, as illustrated in Figure 4.

Statistical Analysis

Differences between Fc and Vc/VT for large and small epicardial patch electrodes were analyzed by analysis of variance (ANOVA). Data are expressed as mean±SD. Differences were considered significant for p<0.05.

Results

Animal Data: Comparison of Fc and Vc/VT

Fc determined in 16 dogs was 0.05±0.01 (Table 1). These data were comparable to Vc/VT measured with the small epicardial patch electrodes in the same dogs, 0.05±0.03, p=NS (paired t test). In eight of these dogs, Vc/VT was also determined with large epicardial patches. For these eight dogs, there was no significant difference between Fc (0.04±0.02), Vc/VT (small patch), 0.04±0.02, and Vc/VT (large patch), 0.05±0.01 (ANOVA).

Human Data

Ten patients were studied, including eight men and two women, with a mean age of 55±20 years (Table 2). Six patients had coronary artery disease, two had idiopathic cardiomyopathy, one had mitral valve prolapse, and one subject had no structural heart disease. Left ventricular ejection fraction in these patients was 36±15%, with a range between 15% and 65%. The most frequent electrode configuration included a small epicardial patch electrode implanted on the anterior surface of the right ventricle and a large patch electrode sutured to posterolateral epicardial surface of the left ventricle (six patients). Two patients received two small patch electrodes, and two others received two large patch electrodes. All but two patients received a rescue shock of 300 J; one patient received 200 J and one patient received 400 J.

A representative analog recording for one patient is shown in Figure 5. The range of transthoracic current

<table>
<thead>
<tr>
<th>Dog</th>
<th>Fc</th>
<th>Vc/VT (Small patches)</th>
<th>Vc/VT (Large patches)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
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<td>0.07</td>
<td>0.06</td>
</tr>
<tr>
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<td>0.05</td>
<td>0.04</td>
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<tr>
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<td>0.03</td>
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<tr>
<td>5</td>
<td>0.07</td>
<td>0.08</td>
<td>0.06</td>
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<tr>
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<td>0.05</td>
<td>0.02</td>
<td>0.03</td>
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<tr>
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<td>0.07</td>
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<tr>
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</tbody>
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Fc, transthoracic current fraction; Vc, transthoracic voltage; VT, transthoracic voltage.
for rescue shocks was between 25 and 48 A. The transthoracic impedance was 65±14 Ω (range, 46–85 Ω). The ratio of $V_C/V_T$ was 0.04±0.03, with a range between 0.01 and 0.10. There was no correlation between $V_C/V_T$ and size of the epicardial patch electrode.

**Discussion**

The major finding in this study is that during transthoracic defibrillation in humans, approximately 4% of the total current traverses the heart. These data are significant because myocardial current density mediates defibrillation, and the magnitude of current density is dependent on the $F_C$ for a given shock dose.

There are two primary hypotheses of electrical defibrillation. According to the critical mass hypothesis,9 defibrillation is achieved when a sufficient mass of excitable cells (but not all cells) is simultaneously depolarized, thereby extinguishing activation wave fronts within a critical mass of myocardium. An alternative hypothesis, the upper limit of vulnerability hypothesis,10 maintains that defibrillation is mediated not only by depolarizing fully excitable cells, but also by depolarizing cells that are in their relative refractory period. This is achieved by delivering a stimulus that exceeds the upper limit of vulnerability (stimulus strength that can induce fibrillation during the vulnerable period) to all regions of the myocardium. Implicit in either hypothesis is that a threshold current density must be reached within the myocardium to achieve defibrillation.

During transthoracic defibrillation (e.g., during cardiac surgery or caused by discharge from an implantable defibrillator),11 myocardial current distribution determines success. In transthoracic defibrillation, another important determinant of success is the intrathoracic current distribution. This factor determines the amount of current that will traverse the

![Figure 5. Analog recordings from patient 10 during a 300-J transthoracic shock. $V_C$, transcardiac voltage; $V_T$, transthoracic voltage; $I_T$, transthoracic current.](image-url)
myocardium (Ic) from the total delivered transthoracic current (It). The ratio of these two quantities (Ic/It) is FC.

Studies of the thoracic volume conductor have been primarily based on analytical or numerical analyses. Analytical studies are, however, inadequate for detailed modeling of the complicated inhomogeneities in the volume conductor, and numerical computations obtained using finite element and integral equation methods have been applied primarily to studying body surface potentials by using passive electrodes rather than the active electrodes that are used for defibrillation. These approaches do not provide direct quantitative information on thoracic current distribution during defibrillation. Recently, studies have been performed using finite element and boundary integral models of the thoracic volume conductor during defibrillation. However, these studies did not directly determine the amount of current flow through the heart during transthoracic defibrillation.

Previous analytical and experimental studies have estimated FC to be between 3% and 45%. Several different methods have been used, each of which has significant methodological limitations. Researchers have compared estimates of mean transcardiac defibrillation thresholds from one group of subjects with mean transthoracic defibrillation thresholds from another group and obtained a value of 25%. Others have compared transmyocardial and thoracic thresholds in the same animals (FC = 20–30%). Comparison of thresholds under this set of conditions to determine FC is not optimal, because it is assumed that the myocardial field distribution (and, hence, myocardial threshold) is identical whether shocks are delivered from epicardial or transthoracic electrodes. Such an assumption cannot be justified because the electrode-epicardial interface will not have the same field configuration as the lung-epicardial interface. FC has been estimated to be 45% based on elementary field equations and a homogeneous slab torso model. The only direct measurement of FC was performed in dogs that had a ring transformer placed around the heart. Electrodes were placed in various orientations on the extremities, and FC was determined to be 3% with a foreleg-to-foreleg electrode configuration. However, FC was not determined with electrodes positioned on the thorax. Of note, these data compare favorably with our results obtained for FC (4%) determined from an experimental resistive-network model of the canine thorax.

Although transcardiac current in humans could not be directly measured in the present study, VC was measured in dogs in protocols 2 and 3 with epicardial patch electrodes, whereas Ic was measured in protocol 1 with calibrated foil electrodes. Because the construction, orientation, and size of the two types of electrodes were different, the field configuration for the two measurements also differed. Epicardial patch electrodes have been shown to alter the flow of current through the heart during transthoracic defibrillation. Therefore, the voltage VC measured with epicardial patch electrodes does not necessarily reflect the total voltage that produces current Ic through the heart (the ratio VC/Ic does not necessarily represent the total resistance of the heart to the current Ic), because the terminals for measurement of VC and Ic were different. This study, therefore, did not seek a relation between VC and Ic but rather a relation between the ratios VC/VT and Ic/It (FC).

A concern and possible limitation in this study was the method by which FC was measured. The foil electrodes, positioned between the pericardium and heart, introduce field distortion in the region of the pericardial-epicardial interface. To account for this perturbation, a calibration protocol was performed to derive a correction factor for this distortion. The assumptions and derivations of the calibration method have been presented in detail, and the correction factor obtained adjusts the cardiac current data to the ideal condition, when no electrodes are present.

The results between FC in dogs and humans were nearly identical. Although the transthoracic electrode positions were not identical in the two studies (the location in dogs was more lateral so that the electrodes would make optimal contact with the thorax), these results suggest that the proportion of transthoracic current that traverses the heart is similar in both species (4–5%). These data are consistent with a previous analysis based on a resistive-
network model of the canine thorax, which showed \( F_C \) to be approximately 4%. In the latter study, the major portion (82%) of transthoracic current was shunted by the thoracic cage, whereas 14% was shunted by the lungs.

For transthoracic defibrillation, only current traversing the myocardium has physiological importance. The significance of \( F_C \) is that it quantifies this fractional distribution of current. These data suggest that approximately 95% of thoracic current does not participate in the defibrillatory process. Because current flow through the thoracic volume conductor is determined by geometric factors as well as by the relative resistivities of the thoracic tissues, it is not unexpected that \( F_C \) could change significantly when thoracic resistances are altered by clinical conditions. For example, based on our canine thoracic resistive-network model, a 50% decrease in resistance (caused by pleural effusion) of the thoracic cage would increase the current shunted around the heart and decrease \( F_C \) from 4% to approximately 2%. If the myocardial defibrillation threshold remained unchanged, the amount of current required for transthoracic defibrillation would double. Therefore, relatively small absolute changes in \( F_C \) can have significant effects on transthoracic threshold. Patients in this study showed considerable variability in \( F_C \) with a range between 1% and 10%. It is likely that these differences are accounted for in part by thoracic geometry, although this was not systematically studied. It must be emphasized that these results can only be considered approximations of \( F_C \) and are limited by methodological considerations. The mean results, however, were in agreement with the animal data obtained in this study and are also consistent with our previous analysis, for which we used a different experimental approach. It therefore appears reasonable to conclude that during transthoracic defibrillation in humans, only approximately 4% of transthoracic current mediates the defibrillatory process.

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\textbf{KEY WORDS} • arrhythmia • automatic implantable defibrillator • ventricular fibrillation
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