Intrathoracic Current Flow During Transthoracic Defibrillation in Dogs

Transcardiac Current Fraction

O. Carlton Deale and Bruce B. Lerman

To achieve transcardiac threshold current during transthoracic defibrillation, a considerably larger current must be delivered to the thorax to compensate for the shunting effect of the lungs, the thoracic cage, and other elements of the torso. This shunting effect is thus an important determinant of transthoracic defibrillation threshold and can be quantified by the transcardiac current fraction \( F_C \), the ratio of transcardiac to transthoracic threshold currents. Previous estimates of \( F_C \) have ranged from as low as 3% to as high as 45%. The purpose of this study was to quantify both \( F_C \) and the major intrathoracic current pathways. Transthoracic and intrathoracic voltages and currents were simultaneously measured during high-voltage transthoracic shocks in 20 dogs. With correction factors determined from another set of 12 dogs, these raw data were corrected to compensate for field distortion caused by the presence of the intrathoracic electrodes, and the adjusted data were fit to a resistive network model. The results showed that 82% of the transthoracic current was shunted by the thoracic cage, while 14% was shunted by the lungs. The remaining 4% \( (F_C) \) is the portion that passed through the heart. There was good agreement between the two independent methods used to calculate \( F_C \). Analysis based on the model indicated that \( F_C \) was 3.7%, whereas \( F_C \) determined by direct measurement with calibrated electrodes was 4.2%. Therefore, the results of this study, in contrast to earlier estimates of \( F_C \), show that defibrillation in dogs is achieved by only 4% of the total transthoracic current. (Circulation Research 1990;67:1405–1419)

Both the critical mass\(^1\) and upper limit of vulnerability\(^2\) hypotheses predict that defibrillation occurs when current density in the myocardium reaches a critical or threshold level. This threshold current density is in turn associated with a transcardiac threshold current \( I_C \). To achieve \( I_C \) during transthoracic defibrillation, a considerably larger current must be delivered to the thorax to compensate for the shunting effect of the lungs, the thoracic cage, and other elements of the torso. This shunting effect is thus an important determinant of transthoracic defibrillation threshold and can be quantified by the transcardiac current fraction \( F_C \), defined as the ratio of \( I_C \) to transthoracic threshold current \( I_{th} \). Previous estimates of \( F_C \) have been between 3% and 45%,\(^3-8\) but the reliability of these data are questionable because of methodological limitations (see “Discussion”).

The purpose of the present study was to quantify \( F_C \) and the major intrathoracic current pathways during transthoracic defibrillation. A resistive network model was developed to describe the major current pathways. Transthoracic and intrathoracic voltages and currents were measured experimentally in dogs during high-voltage transthoracic shocks. Intrathoracic data were corrected to compensate for field distortion caused by the presence of the measuring electrodes and then were applied to the model to determine the relative magnitudes of the resistive intrathoracic pathways and intrathoracic currents during defibrillation.

Methods

Development of the Network Model

Transthoracic defibrillation is generally performed in dogs by placing electrodes on the shaven right and left lateral chest walls at the transverse level of the heart (Figure 1). With this electrode arrangement, nearly all current passing through the heart is conducted by the lungs, and there is little or no potential...
difference across the heart in a direction normal to the axis of the transthoracic electrodes. That is, equipotential surfaces pass through the myocardium approximately normal to the electrode axis. This type of field configuration has been demonstrated by Guha et al with a two-dimensional human torso model and by Claydon et al both experimentally and with a realistic canine volume conductor model.

The heart-lung current pathway corresponding to the above electrode placement is modeled in Figure 1 as a series string of resistances. Each resistance of this string represents a lumped current path through which all transcardiac current flows. Current from the right paddle electrode passes through the right chest wall \( R_{CW1} \), the right lung \( R_{LS1} \), the pericardium and heart \( R_{PH} \), the left lung \( R_{LS2} \), and the left chest wall \( R_{CW2} \) and into the left paddle electrode. In addition to the above series pathway for current through the heart, the lungs also shunt current around the heart through a parallel pathway. This alternate parallel pathway is provided by the lungs contacting each other and is designated \( R_{LP} \).

Another shunting pathway is provided for current by the thoracic cage. The rather low resistivity of the skeletal muscle \( 280 \, \Omega \times \text{cm}^{11} \) is compared with the lungs at peak inspiration \( 2,390 \, \Omega \times \text{cm}^{12} \) which causes a majority of the applied current to be shunted between electrodes by the thoracic cage.13–15 This pathway is designated \( R_{TC} \).

The various current pathways described above are illustrated in Figure 1 as a network of resistances connected between the transthoracic electrodes. Parallel or shunting resistances are shown as single elements, whereas for series resistances, both right and left elements are shown and are designated 1 and 2, respectively. For example, \( R_{CW1} \) is the right chest wall resistance component, and \( R_{CW2} \) is the left chest wall resistance component. The network is redrawn in Figure 2A, with the input terminals at the left representing the transthoracic electrodes and the input resistance to the network designated by \( R_T \), the transthoracic resistance. The network is further simplified in Figure 2B, where \( R_{CW} \) is equal to the sum of \( R_{CW1} \) and \( R_{CW2} \) and \( R_{LS} \) is the sum of \( R_{LS1} \) and \( R_{LS2} \). The resistive ladder network in Figure 2B is the model used in this investigation (see “Appendix, Resistive Ladder Network Model”).

**Experimental Protocol**

Mongrel dogs were anesthetized with sodium pentobarbital \( 30 \, \text{mg/kg} \), maintained with supplemental doses, and ventilated with a respirator (Harvard Apparatus, South Natick, Mass.). Electrocardiographic surface lead II and arterial pressure were continuously monitored (model P23Db transducer, Statham Instruments, Inc., Oxnard, Calif.) on a direct-writing recorder (model ES1000, Gould, Inc., Houston). The femoral vein was cannulated for venous access. A median sternotomy was performed, and the pericardium was reflected.

**Figure 1.** Schematic representation of thorax. The thoracic electrodes are shown located on the midlateral chest walls. The following resistances are represented: \( R_{CW} \), right chest wall; \( R_{CW2} \), left chest wall; \( R_{LS} \), right lung series; \( R_{LS2} \), left lung series; \( R_{PH} \), pericardium and heart; \( R_{LP} \), parallel lung current path; \( R_{TC} \), thoracic cage current path.

**Figure 2.** Resistive network model of the canine thorax. Panel A: Resistive network of Figure 1 redrawn. \( R_T \) is the transthoracic resistance (input resistance of network). Panel B: Resistive ladder network when \( R_{CW} = R_{CW1} + R_{CW2} \) and \( R_{LS} = R_{LS1} + R_{LS2} \). The following resistances are represented: \( R_{CW1} \), right chest wall; \( R_{CW2} \), left chest wall; \( R_{LS} \), right lung series; \( R_{LS2} \), left lung series; \( R_{PH} \), pericardium and heart; \( R_{LP} \), parallel lung current path; \( R_{TC} \), thoracic cage current path.
A pair of foil electrodes (7×7 cm) were molded around the pericardium, anchored to the posterior thoracic musculature, and positioned between the pericardium and medial surfaces of the lungs (Figure 3). The chest was tightly closed with wire sutures, and cables from the electrodes were externalized through the median sternotomy. Each electrode was composed of two square sheets of aluminum foil separated by polyethylene insulation (Figure 4). The electrode cables were connected to an external circuit to measure the current entering and leaving the pericardium and heart and to measure the voltage drop across the pericardium and heart (Figure 5A) (see below).

Transthoracic damped sine wave shocks of 100 J were discharged during sinus rhythm at 5-minute intervals from a commercial defibrillator (Lifepak 6, Physio-Control Corp., Redmond, Wash.). Circular stainless steel electrodes (60 cm²) were covered with electrode paste (Redux Paste, Hewlett-Packard Co., Palo Alto, Calif.) and were placed over the shaven right and left lateral chest walls at the transverse level of the heart. Electrode force was set at 50 N and held constant by a specially designed control system, which has been described previously. The endotracheal tube was clamped at peak inspiration before the system was balanced and the shock was delivered.

Transthoracic voltage (Vₚ) and current (Iₚ) measurements were made using the circuit illustrated in Figure 5B. The voltage signal corresponding to Iₚ is developed across a 0.1-Ω resistor and sent to an isolation amplifier. The voltage signal corresponding to Vₚ is developed using a 1,000:1 voltage divider, and this signal is sent to a second isolation amplifier.

Intrathoracic currents were measured with the switching circuit shown in Figure 5A. Signals developed across the two 0.1-Ω resistors were sent to a pair of isolation amplifiers. Intrathoracic voltage signals, developed using a 1,000:1 voltage divider, were sent to another isolation amplifier. The three switches illustrated allowed the measurement of voltages and currents in the normal configuration as well as open circuit voltages and short circuit currents.

These measurements were made using the following stages (Figure 5).

Stage 1: Pericardium and heart normal (PHN). The short circuit switch Sₐₜ was open, and the left and right switches were closed. Current flow through the pericardium and heart was measured by developing voltage signals across both 0.1-Ω resistors.

Stage 2: Pericardium short circuit (PSC). The left and right switches were open, and the short circuit switch was closed. Current from the lungs was bypassed around the heart through the short circuit switch and developed voltage signals across the 0.1-Ω resistors.

Stage 3: Pericardium open circuit (POC). All switches were open and, except for the high-impedance (1 MΩ) voltage divider, there was no path for current flow. The signal developed by the voltage divider, therefore, closely approximated the open circuit voltage between the outer pericardial electrodes in contact with the lungs.

In the above three stages, in addition to measuring intrathoracic voltages and currents with the circuit in Figure 5A, Vₚ and Iₚ were simultaneously measured using the circuit in Figure 5B.

Stage 4: Thoracic cage (TC). The thoracic cage was electrically isolated from the internal thoracic structures by fitting a sheet of polyester (Mylar, Read Plastics, Rockville, Md.) insulation around the inner circumferential chest wall as illustrated in Figure 6. The corresponding Vₚ and Iₚ were measured using the circuit of Figure 5B.

For high-voltage isolation and common-mode rejection, isolation amplifiers (model AD294A, Analog Devices, Norwood, Mass.) were used. Although the AD294A has a common mode rejection of 108 dB at 60 Hz, the high common-mode voltage applied to its input in these experiments can still result in a significant error signal at the output. In Figure 7, the lower
tracing represents the pulse signal at the output of the isolation amplifier (and contains both the differential mode signal and the common mode error signal combined), while the upper tracing shows only the common mode error signal at the output. The two tracings have different scales. Of particular significance is the fact that when the pulse signal reaches its peak value, the common-mode error signal (at the amplifier's output) has a zero crossing. Since, in the series of experiments conducted, the signal of interest was the peak of the pulse waveform, the common mode signal contributed essentially no error.

The high-impedance input, the floating reference of the input section, and the high common-mode rejection of the AD294A enable it to behave as an instrumentation amplifier. The isolation amplifiers

**Figure 5.** Voltage divider networks. Panel A: Intrathoracic voltage and current measurement. $I_{PHL}$, current entering (leaving) pericardium and heart from left; $I_{PHR}$, current leaving (entering) pericardium and heart from right; $R_{ph}$, resistance of pericardium and heart; $S_L$, switch controlling current flow from left side of pericardium and heart; $S_R$, switch controlling current flow from right side of pericardium and heart; $S_{sc}$, short-circuit switch for bypassing current around the pericardium and heart; $V_{PHL}$, voltage signal developed across 0.1-Ω resistor by $I_{PHL}$; $V_{PHR}$, voltage signal developed across 0.1-Ω resistor by $I_{PHR}$; $V_{PH}$, voltage signal from 1,000:1 voltage divider corresponding to voltage across the pericardium and heart. Each voltage signal is sent to a separate isolation amplifier. Panel B: Trans-thoracic voltage and current measurement. $R_T$ is the trans-thoracic resistance. Trans-thoracic voltage across $R_T$ is reduced by the 1,000:1 voltage divider, and the signal is sent to an isolation amplifier. Current through $R_T$ develops corresponding voltage signal across 0.1-Ω resistor, and this signal is sent to a second isolation amplifier.

**Figure 6.** Placement of polyester (Mylar) insulation between lungs and chest wall for determination of thoracic cage resistance.

**Figure 7.** Differential mode (defibrillator pulse) and common mode signals at output of isolation amplifier.
used in the transthoracic signal circuit were operated at unity gain, whereas the isolation amplifiers used in the intrathoracic signal circuit were operated with a gain of 10 to improve the signal-to-noise ratio.

The output signals from the isolation amplifiers were sent to a storage oscilloscope (model 5113, Tektronix, Beaverton, Ore.). Five different tracings were simultaneously stored by the oscilloscope and visually read from the screen. A Polaroid camera was used for making photographic records of some of the displays.

Relation Between Network Elements and Measured Quantities

Pericardium and heart normal. Normal transthoracic and intrathoracic voltages and currents are present in this condition (Figure 2B). Pericardium and heart current (\(I_{PH}\)), \(I_T\), and \(V_T\) are measured, and \(F_C\) is calculated (=\(I_{PH}/I_T\)).

Thoracic cage measurement. For this condition, the internal organs are insulated from the thoracic cage, and the transthoracic voltage, \(V_{TC}\), and current, \(I_{TC}\), are measured.

As shown in Figure 8A,

\[
R_{TC} = \frac{V_{TC}}{I_{TC}} \tag{1}
\]

Pericardium open circuit. The pericardium is insulated from the lungs so that no current flows through the pericardium and heart. The reduced circuit is shown in Figure 8B.

\[
\frac{V_{POC}}{V_{TPOC}} = \frac{R_{LP}}{(R_{CW} + R_{LP})} \tag{2}
\]

where \(V_{POC}\) is the pericardium open circuit voltage and \(V_{TPOC}\) is the transthoracic voltage for the pericardium open circuit condition and

\[
R_{TPOC}^{-1} = R_{TC}^{-1} + (R_{CW} + R_{LP})^{-1} \tag{3}
\]

where \(R_{TPOC}\) is the transthoracic resistance for the pericardium open circuit condition.

Solving for \(R_{LP}\) and \(R_{CW}\)

\[
R_{LP} = \frac{V_{POC}}{[V_{TPOC}(G_{TPOC} - G_{TC})]} \tag{4}
\]

where \(G_{TPOC}\) is the transthoracic conductance for the pericardium open circuit condition and \(G_{TC}\) is the conductance of the thoracic cage and

\[
R_{CW} = (G_{TPOC} - G_{TC})^{-1} - R_{LP} \tag{5}
\]

where

\[
G_{TPOC} = \frac{I_{TPOC}}{V_{TPOC}} \tag{6}
\]

\((I_{TPOC}\) is the transthoracic current for the pericardium open circuit condition\) and

\[
G_{TC} = R_{TC}^{-1} \tag{7}
\]

Pericardium short circuit. Current is shunted around the pericardium, and the network reduces to the circuit in Figure 8C.

\[
G_{TPSC} = \frac{I_{TPSC}}{V_{TPSC}} \tag{8}
\]

where \(G_{TPSC}\), \(I_{TPSC}\), and \(V_{TPSC}\) are the transthoracic conductance, current, and voltage, respectively, and

\[
R_{LS} = \frac{V_{RLS}}{I_{RLS}} \tag{9}
\]

\(=\frac{V_{TPSC}[1-(G_{TPSC} - G_{TC})R_{CW}]}{I_{TPSC}}\)

where \(V_{RLS}\) is the voltage drop across \(R_{LS}\), \(I_{RLS}\) is the current through \(R_{LS}\), \(R_{CW}\) is the chest wall resistance, and \(I_{TPSC}\) is the pericardium short circuit current.

The above equations give the values of \(R_{TC}\), \(R_{CW}\), \(R_{LP}\), and \(R_{LS}\) provided that the parameters \(V_{TC}\), \(I_{TC}\), \(V_{POC}\), \(V_{TPOC}\), \(I_{TPSC}\), \(I_{TPSC}\), and \(I_{TPSC}\) are determined. Once the values of the elements of the resistive ladder network are determined, the relative distribution of transthoracic current through each path can be calculated.

Experimental Variables

Transthoracic resistance (\(R_T\)), \(V_T\), and \(I_T\) are functions of delivered energy,\(^{17}\) electrode force,\(^{18}\) elec-
trode area, time interval between shocks, respiratory phase, and electrode-skin interface conditions. Methods were devised to set the different variables to constant values during the measurement process.

Energy was selected by the setting on the defibrillator (100 J), electrode area was held constant, and the time interval between shocks was set at 5 minutes. In addition, interface conditions were maintained relatively constant by shaving the contact area on the animal’s chest and periodically applying fresh Redux paste.

A method was also devised to fix the respiratory phase at peak inspiration during each shock. The endotracheal tube was clamped shut at peak inspiration, and the respirator was turned off just before the shock was delivered. Peak inspiration provided maximum contact of the lungs with the heart and chest wall and also gave the maximum resistivity for the lung tissue. Force (50 N) was maintained constant by the force control system.

**Linearity Tests**

The variation of $V_T$ and $I_T$ as a function of the number of shocks presented a unique problem, because the shock number could not be held constant unless all data were taken at a single shock. Because this was not experimentally possible, a method was devised to reduce each measured value from a given experiment to the same shock number by linear interpolation.

Thus, it was desirable to have a linear relation for each measured parameter with respect to shock number for accuracy of the interpolated data. Linearity was tested by performing experiments in which the parameter of interest was measured as a function of shock number, with all other variables held constant.

An initial pair of experiments was performed in closed-chest dogs to test the linearity of $V_T$ and $I_T$ as a function of shock number. Serial 100-J shocks were delivered, and $V_T$ and $I_T$ were recorded for each shock. A linearity test experiment was also performed for the PHN condition with foil electrodes in place, and $V_T$, $I_T$, and $I_{PH}$ were recorded at each 100-J shock.

Another experiment was conducted to test the linearity of $V_T$, $I_T$, and $I_{PH}$ in the PSC condition ($V_{TPSC}$, $I_{TPSC}$, and $I_{PSC}$, respectively). These variables were recorded at each 100-J shock in the PSC condition (which was alternated with the PHN condition).

The above linearity tests are further described in the “Results.”

**Symmetrical Protocol**

Based on the results of the linearity tests, a symmetrical protocol was designed such that all collected raw data could be reduced to the same shock number by linear interpolation. A series of shocks ($n=0, 1, 5$) was administered at the beginning of each experiment to ensure that the data would be stable, and then the main protocol began at $n=6$.

Figure 9 illustrates the symmetry of the protocol and shows the shock number where data for each condition were collected. For example, PHN data were taken at $n=6, 7, 23, 24$ with a mean $n$ of 15. Thus, all raw data for each condition were reduced to correspond to $n=15$ (“Appendix, Data Reduction”).

Figure 9 also shows why linearity tests for some conditions were not necessary. The TC condition consists of three adjacent data points, and linearity was assumed. Similarly, the POC condition was assumed to be approximately linear, because the two parts are separated only by the three thoracic cage shocks. Therefore, linearity tests were conducted only for the PSC and PHN conditions.

**Foil Electrode Correction Factors**

The presence of foil electrodes around the pericardium distorted the field and therefore the measured values of $I_{PH}$ and $V_{POC}$. Experimentally derived correction factors compensated for this distortion. For the two protocols below, all data were taken with the chest closed with wire sutures.

$I_{PH}$ calibration protocol (symmetrical). The purpose of this protocol was to quantify the effect that one foil electrode had on the current measured by a second foil electrode.

1) A single foil electrode was installed on one side of the heart and connected to the circuit illustrated in Figure 5A. Transthoracic shocks were then delivered at 5-minute intervals with the Lifepak 6 and a current divider that has previously been described. $I_T$ was decreased from 30 to 15 A in 3-A steps, and pericardial electrode current, $I_T$, and $V_T$ were recorded at each shock.

2) A second pericardial foil electrode was installed on the opposite side of the heart and was connected to the circuit in Figure 5A. $I_T$ was increased from 15 to 30 A in 3-A steps and then decreased back to 15 A in 3-A steps while the

![Figure 9](image-url)
pericardial electrode current from the first electrode was recorded at each shock along with \( V_T \) and \( I_T \).  
3) The second pericardial electrode was then removed, and \( I_w \) was increased from 15 to 30 A in 3-A steps with pericardial electrode current again recorded at each shock.

Data analysis for the \( I_{PH} \) calibration protocol consisted of first reducing the raw data to the same shock number for each experiment. Next, the mean of corresponding data points was taken over the set of six dogs, and finally, linear regression was performed between 1) current from the first foil electrode without a second electrode installed and 2) current from the first foil electrode with the second electrode installed. The square of the slope of the regression equation became the calibration factor used to correct the raw current data from the pericardial electrodes (“Appendix, Foil Electrode Current Calibration”).

\[ V_{POC} \text{ calibration protocol (symmetrical)} \]

The purpose of this protocol was to quantify the effect that foil electrodes had on the measured value of \( V_{POC} \).

Because of the strong linearity of the \( I_{PH} \) calibration protocol (see “Results”), the \( V_{POC} \) calibration protocol was simplified, and ratios of individual data points were taken rather than taking a range of data and determining the slope through linear regression as in the former protocol.

1) Polyethylene film insulation was installed around the pericardium to simulate the ideal POC condition. Point electrodes were installed on the inner left and right chest walls directly beneath the positions of the circular transthoracic electrodes. The point electrodes were constructed of solid tinned copper wire (gauge 22, American Wire Gauge) individually insulated with polyvinyl chloride insulation (Belden Wire and Cable, Richmond, Ind.). Insulation was removed from the ends of the wires, and the bare wire was formed into 2-mm-diameter loops that were then coated with solder. These loops were sutured to the inner chest wall. Left and right chest wall electrodes were paired for voltage measurement and were positioned as illustrated in Figure 10. Each electrode pair was connected to an individual voltage divider and isolation amplifier similar to those shown in Figure 5A. A pair of transthoracic shocks were delivered 5 minutes apart using the force control system and current divider as previously described, with a \( V_T \) of approximately 1,000 V. At each shock, \( V_T \), \( I_T \), and the three inner chest wall voltages were recorded.

2) The polyethylene insulation was removed from around the pericardium, and a pair of foil pericardial electrodes were installed. Another pair of shocks were delivered as described in step 1 above. \( V_T \), \( I_T \), and \( V_{POC} \) were recorded for both shocks.

3) The pericardial electrodes were removed, and polyethylene insulation was once again installed around the pericardium. Another pair of shocks as described in step 1 were delivered, and \( V_T \), \( I_T \), and the three inner chest wall voltages were again recorded.

Data analysis for the \( V_{POC} \) calibration protocol consisted of taking the mean of the inner chest wall voltages and the mean of the \( V_{POC} \) voltages and determining the ratio between the two mean values.

**Data analysis (main protocol).** Data were analyzed by performing the following sequence of steps.

1) Raw data (voltages and currents) obtained from each dog in a symmetrical protocol (Figure 9) were reduced to correspond to the same shock number (n=15) by linear interpolation. (This step was required because all measured voltages and currents were [linear] functions of n but could not all be measured at the same n.)

2) Voltages for each dog were normalized by dividing by \( V_T \) recorded during the PHN condition (\( V_{TPHN} \)). Likewise, currents were normalized by dividing by the transthoracic current for the PHN condition (\( I_{TPHN} \)). (Normalization of the data was necessary for combining data from different dogs.)

3) Means of corresponding normalized voltages and currents were taken over a set of 20 dogs to compensate for random experimental errors and variability from animal to animal.

4) Correction factors obtained from the calibration protocols were applied to \( I_{PH} \) and \( V_{POC} \).

5) Network elements \( R_{TC}, R_{CW}, R_{LP,} \) and \( R_{LS} \) were calculated using mean-normalized voltages and currents from the TC, POC, and PSC conditions and Equations 1–9. (Also, for mean-normalized data, \( R_T = 1. \))

6) \( F_C \) was calculated from the model by using the network elements found above and Equations A22, A25, and A26 (“Appendix”).

7) From the PHN condition (not used above), \( F_C \) was also determined from the mean-normalized value of \( I_{PH} \) (\( I_{PH} \) [normalized]=\( F_C \)).

8) The two independent values of \( F_C \) were compared to assess the validity of the model as well as the electrode calibration techniques.

9) After the above comparison was made, the value of \( R_{TH} \) was adjusted until \( F_C \) calculated from the model (Equations A22 and A25, “Appendix”)

![Figure 10. Placement of point electrodes for inner chest wall voltage measurement. \( V_1, V_2, \) and \( V_3 \) are measured voltage differences.](http://circres.ahajournals.org/)
linearity were -0.987, the determine (Figure condition respectively. obtained against linear change obtained were linear with respective. Because the value of RT now differed slightly from unity, the network was scaled by dividing all elements (including RT) by the calculated value of R. The resulting network has an input resistance of unity and is the result of experimental measurements from the TC, POC, PSC, and PHN conditions.

Results

Linearity Tests

Four experiments were performed to validate the use of linear interpolation for reduction of data obtained at different shock numbers to the same shock number. Figure 11 shows the results of the first experiment in which VT and IT were measured as a function of shock number. VT and IT were found to be linear with respect to shock number. The correlation coefficients were -0.962 and 0.963 for VT and IT, respectively. This protocol was repeated in a second experiment, and the correlation coefficients obtained were -0.945 and 0.950 for VT and IT, respectively.

Another experiment was performed to verify the linear change in measured VT, IT, andIPH with respect to shock number for the PHN condition (Figure 12). The correlation coefficients obtained were -0.987, 0.985, and 0.970, respectively.

A linearity test was also performed for the PSC condition (Figure 13). VTPS, ITPS, and IPS were plotted against shock number. The correlation coefficients obtained were -0.969, 0.974, and 0.929, respectively.

Pericardial Electrode Calibration

A series of 12 experiments was performed to determine the effect of the pericardial electrodes on the measured intrathoracic voltages and currents. The effect of pericardial electrodes on measured heart current was determined in six dogs for both left and right electrodes. The presence of the electrodes caused an increase in measured heart current and resulted in a correction factor of $\beta^2 = 0.507$. The constant $\beta$ ("Appendix, Foil Electrode Current Calibration") relates the measured electrode current in a distorted field to the measured electrode current in an undistorted field. This relation is shown in Figure 14, for which $\beta$ is the slope of the linear regression equation. The linearity between the two variables is demonstrated by a correlation coefficient of 0.998. The slope $\beta = 0.712$.

In another set of six dogs, the relation between measured POC voltage and the inner chest wall voltage was obtained. Because of the distortion of the field caused by the presence of the pericardial electrodes in the POC condition, the voltage measured by the electrodes is less than the inner chest wall voltage measured when the electrodes are not present. A correction factor of 2.04 was obtained.

Comparison of Model-Dependent Versus Model-Independent Results

Fc was calculated from the model by using the values for RT, RCW, RLp, RLS, and RT (1) as

\[
\text{FIGURE 11. Transthoracic voltage (VT) and current (IT) vs. shock number (n) for pericardium and heart normal condition.}
\]

\[
\text{FIGURE 12. Transthoracic voltage (VT), transthoracic current (IT), and pericardium and heart current (IPH) vs. shock number (n) for pericardium and heart normal condition.}
\]
only, since the mean-normalized value of $I_{PHN}$ is equal to $F_C$ (by definition). A value of 4.2% was obtained by this method.

Resistive Network Model

The complete resistive network model with the element values determined is shown in Figure 15, and all element values are scaled to give a transthoracic resistance of unity.

Also shown in Figure 15 are the currents through each branch of the network. As illustrated, 82% of $I_T$ is shunted between electrodes by the thoracic cage. Fourteen percent of $I_T$ is shunted by the lungs, whereas the remainder of the current traversing the pericardium and heart is approximately 4%.

Variation in $F_C$

The relative significance of each network element in the determination of $F_C$ can be seen by varying the elements one at a time and plotting the corresponding value of change in $F_C$ ("Appendix, Calculation of $F_C$ From Network Elements"): This effect is illustrated in Figure 16 for the elements $R_{TC}$, $R_{CW}$, $R_{LP}$,

![Graph showing transthoracic current (Ip) and pericardium-short circuit current (Ipsc) vs. shock number (n) for pericardium short-circuit condition.]

![Graph showing relationship between $I_E$ and $I_E'$ with equation $I_E = 0.712 I_E' + 0.188$ and correlation coefficient $r = 0.998$.]
FIGURE 16. The transcardiac current fraction (FC) as a function of the following resistances: thoracic cage (RTC), chest wall (RCW), parallel lung current path (RLP), lung series (RLS), and pericardium and heart (RPH).

\[ \text{RTC} \]
\[ \text{RCW} \]
\[ \text{RLP} \]
\[ \text{RLS} \]
\[ \text{RPH} \]

Changes in the normalized resistance from RTC, for example, can significantly alter FC because a 50% reduction in RTC decreases FC by approximately 50%.

**Variation in Transthoracic Threshold**

Variation in I_{TH} can also be plotted as a function of variation in the network elements ("Appendix, Calculation of FC From Network Elements and Variation in Transthoracic Threshold Current"). Figure 17 shows the percent change in I_{TH} as a function of RTC, RCW, RLP, RLS, and RPH. For example, doubling RTC reduces threshold current by approximately 40%.

**Discussion**

I_{TH} has been shown to be a deterministic quantity.\textsuperscript{23} For a given I_{TH}, there will be a corresponding I_{C}. FC is the ratio of the two. That is,

\[ \text{FC} = \frac{I_C}{I_{TH}} \quad \text{(10)} \]

and

\[ I_{TH} = I_C \cdot F_C^{-1} \quad \text{(11)} \]

That is, at a given defibrillation episode, I_{TH} is a function of two parameters, I_{C} and F_{C}. F_{C} and I_{C} are independent of each other, and although I_{C} is a threshold quantity, F_{C} is not. That is, F_{C} for sub-threshold, suprathreshold, and even sinus rhythm shocks should not significantly differ from F_{C} for threshold shocks. An assumption of this study was that F_{C} measured during sinus rhythm was equal to F_{C} measured in ventricular fibrillation with a threshold shock.

This study determined a mean F_{C} by fitting experimental data from 20 dogs to a resistive network model. Other assumptions were that transthoracic and in-
trathoracic impedances are primarily resistive\textsuperscript{17,24,25} and that \(I_c\) is not appreciably affected by the presence of the pericardium.\textsuperscript{26} Because the pericardium is a conductive medium surrounding the heart, it provides an additional shunting path for current around the heart. However, the fact that the pericardium is thin and in close proximity to the myocardium suggests that the amount of current shunted is probably small and that the overall effect can be neglected.\textsuperscript{26}

A potential source of error in this study was the introduction of metallic foil electrodes between the pericardium and lung. These electrodes distorted the field in the vicinity of the pericardium and lung interfaces so that the recorded intrathoracic voltages and currents were, in general, not equal to the voltages and currents that existed when the foil electrodes were not present. Although field lines passing across the pericardium-lung interface would be refracted because of the difference in electrical resistivities of the two media, the metal–lung tissue interface and the metal-pericardium interface would produce much greater refraction of the field lines.\textsuperscript{27} The lines entering and leaving the conductive foil would be at an angle of 90° to the foil, which in certain regions of the interface would represent extreme distortion. The crucial question was whether these measured values of intrathoracic voltages and currents could be related to the real values that existed when the foil electrodes were not present.

Three different cases were considered in answering the above question: PHN, PSC, and POC. In the PHN condition, the normal field pattern across the pericardium-lung interface is assumed, and the introduction of foil electrodes would be expected to distort the field as discussed above. The PSC and POC conditions, however, represent planned perturbations of the field. In the PSC condition, it is assumed that the heart is replaced with a conductor. The introduction of foil electrodes in this case would not change the field, because the foil electrodes are, themselves, conductors. However, in the POC condition it is assumed that the heart is replaced with an insulator. Therefore, the introduction of foil electrodes in this case would cause considerable distortion of the field. Ideally, the measured voltage \(V_{\text{POC}}\) in this configuration is equal to \(V_{\text{RLP}}\), which represents the voltage across the inner chest wall. Because of distortion of the field however, the measured value of \(V_{\text{POC}}\) may differ considerably from \(V_{\text{RLP}}\).

To resolve these problems, several assumptions were made: 1) the measured value of \(V_{\text{POC}}\) is proportional to \(V_{\text{RLP}}\), 2) \(I_{\text{PH}}\) in an undistorted field is linearly related to \(I_{\text{PH}}\) in a field distorted by a single foil electrode, 3) \(I_{\text{PH}}\) measured by a single foil electrode is linearly related to \(I_{\text{PH}}\) measured by that electrode when the field is distorted by a second electrode, and 4) the effect of the field distortion caused by the foil electrodes on the measured value of \(I_{\text{PH}}\) is approximately the same as the effect on the true value of \(I_{\text{PH}}\). (The relation between measured \(I_{\text{PH}}\) and true \(I_{\text{PH}}\) is derived in “Appendix, Foil Electrode Current Calibration.”)

Based on the above assumptions, experiments were performed to obtain correction factors for \(V_{\text{POC}}\) and for \(I_{\text{PH}}\). Assumption 3 was verified experimentally by linear regression (see “Results”) and tends to make assumption 2 plausible because of the similarity between assumptions 2 and 3. The calibration of the electrodes made the measured current independent of electrode area or shape.

To minimize the effect of random measurement errors and variability from animal to animal, mean-normalized data were used in the analysis. All intrathoracic voltages were normalized to \(V_{\text{IPH}}\) and all intrathoracic currents were normalized to \(I_{\text{IPH}}\) for each animal. The mean of each normalized value was taken over the set of 20 dogs, and the resulting mean-normalized voltages and currents were used to calculate the values of the resistive elements in the model by the use of Ohm’s Law and Kirchhoff’s Voltage and Current Laws.

Because mean-normalized data were used in the analysis, \(R_7=1\) (“Appendix, Calculation of \(F_C\) From Network Elements”). After determination of \(R_7\), \(R_{\text{CW}}, R_{\text{LP}}, \text{ and } R_{\text{LS}}\), sufficient information was available for calculation of \(F_C\). The calculated value of \(F_C\) from the model was 3.7%. The raw data used in this calculation came from the TC, POC, and PSC conditions and the POC calibration protocol.

The above calculated value of 3.7% was then compared with the normalized heart current (\(F_C\)) measured during the PHN condition (and corrected by the PHN calibration protocol). The measured and corrected value of \(F_C\) was 4.2%. Thus, the close agreement between the two values of \(F_C\) obtained by two different methods supports both the model and the electrode calibration procedures. After comparison of the two values of \(F_C\), the PHN data were incorporated into the resistive network, and the resulting elements were scaled to give an \(R_7\) of unity (see “Methods”). The resulting scaled network is shown in Figure 15. It is seen that the shunting path provided by the thoracic cage has a net resistance just above unity and therefore dominates \(R_7\). The relatively thin and conductive chest wall provides only a small series resistance. Although the lungs nearly fill the chest cavity at peak inspiration and provide a relatively large volume for conduction, the very high resistivity of the lungs at peak inspiration contributes to a net high resistance for \(R_{\text{LP}}\) and \(R_{\text{LS}}\). In spite of the high conductivity of the blood and myocardium, the relatively small size of the heart also produces a high resistance current path.

The relative magnitudes of the elements in the resistive network determine the net distribution of \(I_F\) through the network (Figure 15). The majority of the current is shunted between transthoracic electrodes by the thoracic cage, leaving only 18% to be distributed intrathoracically. Fourteen percent of the total \(I_F\) is shunted around the heart by the lungs, while only about 4% actually traverses the heart.
Previous estimates of Fc by others range from 3% to 45%. Kouwenhoven and Hooker\(^3\) compared the amount of transmyocardial alternating current (AC) required to defibrillate the heart with 1-in. brass disk electrodes to the amount of \(I_f\) required to defibrillate the heart with 2.75-in.\(^2\) transthoracic electrodes and obtained a mean value of 12%. In a similar manner, Guyton and Satterfield\(^4\) used AC defibrillation to estimate Fc as 20%. The difference between the results of these two studies is an indication of the degree of variability in calculated Fc by using the technique of comparing measured heart thresholds to transthoracic thresholds. Geddes\(^5\) made estimates of Fc by using heart threshold data normalized to body weight and transthoracic threshold data normalized to body weight and obtained values of approximately 20% for Fc.

A rather large value of Fc of 45% was estimated by Rush et al\(^6\) by the use of field theory and a slab torso model. However, the simplifications of the model limit the reliability of the calculated results because a cubic heart and uniform conductivity throughout the thorax was assumed. With these constraints, the current density over a cross section of the cubic heart was calculated, and the net current flow through the heart was then calculated using an assumed cross-sectional area.

Perhaps the most accurate experimental measurements of heart current were made by Kouwenhoven\(^6\) using a ring transformer. This device encircled the heart during the passage of transthoracic AC and produced a voltage that was proportional to the current flowing through the heart. The device was carefully calibrated using an electrolytic tank, and the measured results were probably quite accurate. The study was designed to determine the percentage of 60 Hz AC through the heart for various orientations of electrodes placed on the canine torso. Kouwenhoven et al\(^6\) obtained a value of 3% for Fc with a foreleg-to-foreleg electrode orientation and a value of 10% for a head-to-hind leg electrode configuration. Thus, the value of 4% for Fc obtained in the present study corresponds to the lower estimates of Fc in the literature but disagrees considerably with the higher estimates. The lower values of Fc obtained by Kouwenhoven and coworkers are probably more reliable because of his direct measurement technique and painstaking calibration of his instrumentation.

Although the previous studies have indeed provided estimates of Fc, each suffers from one or more serious limitations. The field theoretical model is limited by the simplifying assumptions. Estimates of Fc obtained by comparing heart threshold currents with transthoracic threshold currents implicitly assume that heart threshold current corresponding to the heart-lung interface is the same as heart threshold current corresponding to the heart–metal electrode interface. In addition, these thresholds were obtained during AC defibrillation at several hundred volts AC and capacitive discharge defibrillation. It is not known how well these results apply to defibrillation with a high-voltage damped sinusoidal pulse. Although the ring transformer studies were carefully performed, electrode placement did not correspond to present-day defibrillator electrode placement, and 60 Hz AC of only several hundred milliamperes was used. Because most present-day defibrillators are damped sine wave devices that deliver approximately 60 A into a 50-Ω load (400-J shock), it is difficult to make a direct comparison between the two techniques.

The present investigation sought to avoid some of the limitations of previous studies and is unique in several ways. Simultaneous measurement of transthoracic and intrathoracic voltages and currents was made using high-energy damped sinusoidal pulses. Experimental data were fit to a resistive network model for quantification of the intrathoracic current pathways. In addition to Fc, the percentage of \(I_f\) flowing through the other two major shunting pathways, \(R_{TC}\) and \(R_{LP}\), was determined. The magnitudes of the elements in the resistive network model were also quantified and used to investigate the determinants of Fc.

The effect of each network element on Fc (“Appendix, Calculation of Fc From Network Elements”) can be obtained by varying each network element about its base value and plotting the corresponding values of Fc (Figure 16). For example, if the shunting elements \(R_{TC}\) and \(R_{LP}\) are increased, the amount of current shunted away from the heart is decreased, resulting in a net increase of current through the heart and an increase of Fc. On the other hand, an increase in the series element \(R_{CW}\) or \(R_{LS}\) results in less current traversing the heart and a net decrease in Fc. Similarly, an increase in \(R_{PH}\) causes a decrease in Fc. The slope of each curve gives an indication of the relative importance of that element in determining Fc. Figure 16 shows that \(R_{TC}\), \(R_{PH}\), and \(R_{LS}\) have a major effect on Fc, while the effect of \(R_{CW}\) is insignificant. \(R_{LP}\) does not have a significant effect unless it gets very small.

These results can also be applied to the investigation of transthoracic threshold current (“Appendix, Variation in Transthoracic Threshold Current”). If heart threshold current is (mathematically) held constant, the change in \(I_c\) can be determined as a function of the change in a given network element. The effect of each network element on \(I_c\) is shown in Figure 17. As was the case with Fc, \(R_{CW}\) and \(R_{LP}\) have only minor effects, while \(R_{PH}\), \(R_{LS}\), and \(R_{TC}\) appear as primary determinants of threshold current.

In summary, the results of this study show that for canine transthoracic defibrillation, 82% of the total delivered current is shunted between the electrodes by the thoracic cage, 14% is shunted by the lungs, and only 4% traverses the heart. This 4% figure is predicted by the resistive network model whose elements have been determined from empirical data and is confirmed by an independent measurement of transcardiac current. Variations in the shunting paths provided by either the
thoracic cage or the lungs may have significant effects on transthoracic threshold current.

Appendix
Resistive Ladder Network Model

Thoracic cage. When the entire interior of the thorax is insulated from the thoracic cage, the only path for current is through the external thoracic cage resistance \( R_{TC} \). Thus the networks in Figures 2A and 2B reduce to a single resistance, \( R_{TC} \).

Pericardium open circuit. If the pericardium is insulated from the lungs and the other organs in the thorax, no path is provided for current flow through the pericardium and heart. For both networks, \( R_{PH} \to \infty \).

In Figure 2A, the current through resistor \( R_{LP} \) is

\[
I_{RLP} = \frac{V_T}{(R_{CW1} + R_{LP} + R_{CW2})} = \frac{V_T}{(R_{CW} + R_{LP})}
\]

(A1)

Then the voltage \( V_{POC} = V_{RLP} \) is the same as for Figure 2B.

Pericardium short circuit. If the heart were replaced by a conductor, then \( R_{PH} = 0 \), and the current through the conductor would be \( I_{PSC} \).

In Figure 2A,

\[
I_{PSC} = V_T \left[ R_{LP} \left( R_{LS1} + R_{LS2} \right) \right] / \left[ \left( R_{CW1} + R_{CW2} \right) + \left( R_{LP} \left( R_{LS1} + R_{LS2} \right) \right) \left( R_{LS1} + R_{LS2} \right) \right]
\]

(A2)

and reduces to

\[
I_{PSC} = V_T \left( R_{LP} \left[ R_{LS} \right] / \left[ \left( R_{CW} + \left( R_{LP} \left[ R_{LS} \right] \right) \right) R_{LS} \right] \right)
\]

(A3)

However, Equation A3 also gives \( I_{PSC} \) for Figure 2B. Therefore, the pericardium short circuit current \( I_{PSC} \) is the same for either network.

Data Reduction

For reduction of data collected in a symmetrical protocol, linear interpolation simply involves taking the arithmetic mean of two measured data points to provide the desired (but unmeasured) data point halfway between the two. For example, if a given voltage varies linearly with shock number \( n \) (\( n = 0, 1, \ldots \)),

\[
V_n = kn + V_0
\]

(A4)

where \( V_n \) is the voltage, which varies as a function of \( n \), \( k \) is the slope, and \( V_0 \) is the intercept.

Suppose the voltage at \( n = 15 \) (\( V_{15} \)) is desired but cannot be conveniently measured at that value of \( n \) and that \( V_{13} \) and \( V_{17} \) can be measured. Then

\[
V_{13} = 13k + V_0
\]

(A5)

and

\[
V_{17} = 17k + V_0
\]

(A6)

Adding Equations A5 and A6 gives

\[
V_{13} + V_{17} = 30k + 2V_0
\]

(A7)

and dividing by 2 gives

\[
(V_{13} + V_{17})/2 = 15k + V_0
\]

(A8)

However, by Equation A4

\[
15k + V_0 = V_{15}
\]

Therefore,

\[
V_{15} = (V_{13} + V_{17})/2
\]

(A9)

That is, the desired voltage at \( n = 15 \) is obtained by averaging the voltages measured at \( n = 13 \) and 17.

Foil Electrode Current Calibration

Refer to Figures A1 and A2.

**Definitions.** The mean current (\( I \)) through the heart and pericardium is defined as the mean of the currents through the left and right sides of the heart and pericardium over a set on \( N \) dogs.

\[
I = \frac{1}{N} \sum_{i=1}^{N} \left[ \frac{1}{2} (I_{Li} + I_{Ri}) \right]
\]

(A10)

Similarly, \( I' \) is the mean current through the heart and pericardium when the field is perturbed by a foil electrode on the opposite side of the heart.
RIGHT ELECTRODE CURRENT CALIBRATION

**Figure A2.** Current flow through right side of heart for different electrode combinations. Panel A: Current (IE) through right side of heart when no electrodes are present. Panel B: Current (IE) through right side of heart when field on left side is distorted by electrode. Panel C: Current (IE) through right side of heart when no electrode is on left side. Panel D: Current (IER) through right side of heart when field on left side is distorted by electrode.

\[ I'_E = \frac{1}{2N} \sum_{i=1}^{N} (I'_{Li} + I'_{Ri}) \]  
(A11)

\[ I_E = \frac{1}{2N} \sum_{i=1}^{N} (I_{Li} + I_{Ri}) \]  
(A12)

\[ I'_E = \frac{1}{2N} \sum_{i=1}^{N} (I'_{REi} + I'_E) \]  
(A13)

**Assumptions.** The current (through the pericardium and heart) corresponding to the unperturbed field is linearly related to the current corresponding to the perturbed field

\[ I = \alpha I' \]  
(A14)

where \( \alpha \) is the constant of proportionality.

The measured electrode current with the field on one side of the heart unperturbed is linearly related to the measured electrode current with the field perturbed.

\[ I_E = \beta I'_E \]  
(A15)

where \( \beta \) is the constant of proportionality.

The effect of the perturbation on measured electrode current is approximately the same as the effect on the actual heart and pericardial current

\[ \beta = \alpha \]  
(A16)

On the average, current through the left side of the heart and pericardium is approximately equal to current through the right side

\[ \frac{1}{N} \sum_{i=1}^{N} I'_{Li} = \frac{1}{N} \sum_{i=1}^{N} I_{REi} \]  
(A17)

\[ \frac{1}{N} \sum_{i=1}^{N} I_{RI} = \frac{1}{N} \sum_{i=1}^{N} I_{LEi} \]  
(A18)

**Derivation.** Substituting Equations A17 and A18 into Equation A11

\[ I' = \frac{1}{2N} \sum_{i=1}^{N} (I_{REi} + I_{LEi}) \]  
But from Equation A12

\[ I' = I_E \]  
(A19)

From Equations A14, A16, and A19

\[ I = \alpha I' = \beta I' = \beta I_E \]  
(A20)

where \( \beta \) is the correction factor for the effect of a single electrode. From Equation A15, the correction factor for a pair of foil electrodes is \( \beta^2 \); that is,

\[ I = \beta^2 I_E \]  
(A21)

where \( I_E \) is the mean pericardial electrode current measured with a pair of foil electrodes during the main experimental protocol. Also from Equation A15, it is seen that \( \beta \) is the slope of the linear regression equation obtained from the calibration protocol.

**Calculation of FC From Network Elements**

Refer to Figure 2B.

Let \( I_T \) be the total transthoracic current, \( I_{CW} \) the current through \( R_{CW} \), \( I_{TC} \) the current through \( R_{TC} \), \( I_{PH} \) the current through \( R_{PH} \) and \( R_1 \) (not illustrated) the net resistance in parallel with \( R_{TC} \).

\[ R_1 = R_{CW} + \left[ R_{LP}^{-1} + (R_{LS} + R_{PH})^{-1} \right]^{-1} \]  
(A22)

\[ I_{CW} = I_T R_{TC} (R_{TC} + R_1)^{-1} \]  
(A23)

and

\[ I_{PH} = I_T R_{LC} R_{TC} (R_{TC} + R_1)^{-1} (R_{LP} + R_{LS} + R_{PH})^{-1} \]  
(A24)

Then,

\[ F_C = \frac{I_{PH}}{I_T} \]
\[ R_{TC}R_{LP}(R_{TC}+R_{1}^{-1})(R_{LP}+R_{LS}+R_{PH})^{-1} \]

(A25)

For mean-normalized voltages and currents, \( R_T = 1 \), \( R_T = V_I/I_1 = 1/1 \).

\[ R_{TC}, R_{CW}, R_{LP}, \text{ and } R_{LS} \text{ can be determined from the TC, POC, and PSC conditions. Then,} \]

\[ R_{PH} = [(1 - R_{TC}^{-1})^{-1} - R_{CW}^{-1} - R_{LP}^{-1}]^{-1} R_{LS} \]  

(A26)

and \( F_C \) can be determined from Equations A25 and A26.

**Variation in Transthoracic Threshold Current**

Let \( I_C \) and \( I_T \) be transthoracic and transthoracic threshold currents, respectively. Then,

\[ F_C = I_C/I_T \]  

and

\[ I_T = I_C/F_C \]  

(A27)

(A28)

Let

\[ \Delta I_T = \% \text{ change in } I_T \]

Then,

\[ \Delta I_T = [ (I_{T2} - I_{T1})/I_{T1} ] (100\%) \]

\[ = (I_{T2}/I_{T1} - 1) (100\%) \]

\[ = (I_{C2}F_{C1}/I_{C1}F_{C2} - 1) (100\%) \]  

(A29)

If \( I_C \) is (mathematically) held constant,

\[ I_{C2} = I_{C1} \]

then

\[ \Delta I_T = (F_{C1}/F_{C2} - 1) (100\%) \]  

(A30)

**References**


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