Optimal Small-Capacitor Biphasic Waveform for External Defibrillation

Influence of Phase-1 Tilt and Phase-2 Voltage

Yoshio Yamanouchi, MD; James E. Brewer, MS; Kent A. Mowrey, MS; Ann M. Donohoo, MS; Bruce L. Wilkoff, MD; Patrick J. Tchou, MD

Background—Biphasic waveforms have been reported to be more efficacious than monophasic waveforms for external defibrillation. This study examined the optimal phase-1 tilts and phase-2 leading-edge voltages with small capacitors (60 and 20 \( \mu \)F) for external defibrillation. We also assessed the ability of the “charge-burping” model to predict the optimal waveforms.

Methods and Results—Two groups of studies were performed. In group 1, 9 biphasic waveforms from a combination of 3 phase-1 tilt values (30%, 50%, and 70%) and 3 phase-2 leading-edge voltage values (0.5, 1.0, and 1.5 times the phase-1 leading-edge voltage, \( V_1 \)) were tested. Phase-2 pulse width was held constant at 3 ms in all waveforms. Two separate 60-\( \mu \)F capacitors were used in each phase. The energy value that would produce a 50% likelihood of successful defibrillation (\( E_{50} \)) decreased with increasing phase-1 tilt and increased with increasing phase-2 leading-edge voltage except for the 30% phase-1 tilt waveforms. In group 2, 9 waveforms were identical to the waveforms in group 1, except for a 20-\( \mu \)F capacitor for phase 2. \( E_{50} \) decreased with increasing phase-1 tilt. Phase-2 leading-edge voltage of 1.0 to 1.5 \( V_1 \) appeared to minimize \( E_{50} \) for phase-1 tilt of 50% and 70% but worsened \( E_{50} \) for phase-1 tilt of 30%. There was a significant correlation between \( E_{50} \) and residual membrane voltage at the end of phase 2, as calculated by the charge-burping model in both groups (group 1, \( R^2=0.47, P<0.001 \); group 2, \( R^2=0.42, P<0.001 \)).

Conclusions—The waveforms with 70% phase-1 tilt were more efficacious than those with 30% and 50%. The relationship of phase-2 leading-edge voltage to defibrillation efficacy depended on phase-2 capacitance. The charge-burping model predicted the optimal external biphasic waveform. (Circulation. 1998;98:2487-2493.)

Key Words: defibrillation • ventricles • death, sudden

Several internal defibrillation studies have established the superiority of biphasic shock waveforms over comparable monophasic waveforms. Exponential biphasic waveforms can also provide improved external defibrillation efficacy.

Recently, a quantitative “charge-burping” model has been proposed to explain the improved efficacy of biphasic over monophasic shocks. A recent study supported this model by demonstrating that minimum residual membrane voltage as calculated by the charge-burping model predicted the optimal internal biphasic waveform.

Defibrillation efficacy may also be improved by optimizing capacitance values. On the basis of models of internal defibrillation, optimal capacitance depends on shock impedance and the chronaxy of the strength-duration curve. Assuming a time constant of 2 to 4 ms and a mean impedance of 40 \( \Omega \), as reported in a previous swine external defibrillation study, the optimum capacitance is calculated by these theoretical models to be in the 50- to 100-\( \mu \)F range. Hence, a 60-\( \mu \)F capacitor would be within this optimal range to provide maximal external defibrillation efficacy. Recent defibrillation studies have shown that the biphasic waveform with changing capacitance at phase reversal may reduce the defibrillation threshold (DFT). However, the optimal combination of phase-1 tilt and phase-2 leading-edge voltage to maximize defibrillation efficacy has not been determined in such a changing capacitor external waveform.

The purpose of this study was (1) to assess the contribution of phase-1 tilt and phase-2 leading-edge voltage in optimizing the small-capacitor (60/60-\( \mu \)F) biphasic waveform for external defibrillation, (2) to determine the optimal timing and voltage of phase reversal to maximize external defibrillation efficacy in biphasic waveform with changing capacitor at phase reversal (60/20-\( \mu \)F), and (3) to assess the ability of the charge-burping model to predict the optimal external defibrillation waveforms.
Defibrillation Equipment and Protocol

The transthoracic defibrillation electrode system consisted of adhesive pad electrodes, each with a surface area of 75 cm², applied to the right high anterior shaved thorax and to the left anterior lateral shaved thorax. Pad electrodes were connected to an external defibrillator custom-made by SurvivaLink Corp. This custom external defibrillator delivered a monophasic or biphasic, high-voltage, time-truncated, capacitative discharge pulse. The defibrillator was equipped with 2 capacitor banks. Each capacitor bank was used for a phase of a monophasic or biphasic shock, with independent phase capacitance values in the range of 10 to 300 µF, in 10-µF steps. Each capacitor bank was charged to a maximum of 4000 V and was equipped with 2 capacitor banks. Each capacitor bank was used for the test biphasic waveform, a monophasic rescue shock (450 to 900 V) was delivered via the RV electrode. A recovery period of at least 3 minutes was allowed between episodes of fibrillation. Fibrillation was not reinitiated until heart rate and blood pressure had returned to the preshock values.

Methods

Surgical Procedures

A detailed description of these procedures was published previously. Briefly, swine were premedicated with ketamine and morphine and anesthetized with intravenous sodium pentobarbital, with repeated doses of 1 to 2 mg/kg given as necessary to maintain anesthesia. Pancuronium bromide was given every 30 minutes to anesthetize the swine. Ventilation with room air supplemented with oxygen as needed to maintain normal arterial blood gases. The transthoracic defibrillation electrode system consisted of adhesive pad electrode was applied to the left high anterior shaved thorax. Pad electrodes were connected to an external defibrillator (model 4007L, Angeion Corp) was inserted into the right ventricle (RV). An adhesive pad electrode was applied to the left high anterior shaved thorax. This skin patch in combination with the transthoracic electrode was used to induce fibrillation and to deliver rescue shocks after failed test shocks.

Defibrillation Waveforms

Nine biphasic waveforms were tested in each group. The description of each waveform in groups 1 and 2 is detailed in Figures 1 and 2, respectively. The 9 waveforms in each group were tested in random order in each experiment.

Residual Membrane Voltage

Calculation of residual membrane voltage for the purpose of testing the charge-burping hypothesis was performed according to the method described by Kroll and detailed in the Appendix.

Statistical Analysis

Group data were expressed as mean ± SD. Repeated-measures 1-way ANOVA was used to compare defibrillation parameters among the 9 waveforms in each group. Pairwise comparisons were performed by the method of contrasts. ANCOVA was used between the E50 for delivered energy and the normalized absolute residual membrane voltage at the end of phase 2 in each group. The null hypothesis was rejected for P < 0.05.

Results

Group 1

Complete DFT data sets were obtained from 10 swine (33 ± 3 kg). The waveform characteristics and the DFT parameters are detailed in Table 1.
Defibrillation Energy

Figure 3 shows the E\textsubscript{50} of delivered energy in each waveform. For 30% phase-1 tilt waveforms, the E\textsubscript{50} of the 30/0.5 phase-1 leading-edge voltage (V\textsubscript{1}) waveform was the lowest compared with the 30/1.0 V\textsubscript{1} (P=0.0001), and 30/1.5 V\textsubscript{1} waveforms (P=0.0002). Similar to 30% phase-1 tilt waveforms, the 50/0.5 V\textsubscript{1} and the 70/0.5 V\textsubscript{1} waveforms generated the lowest E\textsubscript{50} within their corresponding 50% and 70% phase-1 tilt waveforms. This 3x3 design yielded 9 different waveforms. Phase-2 pulse width was held constant at 3 ms in all waveforms. Thin lines show membrane response curves to each waveform as computed by charge-burping model. Both shock voltage and membrane voltage were normalized to give a maximum possible value of 1.00 in phase 1.

Membrane Voltage

The residual membrane voltages at the end of phase 2, based on the charge-burping model for the test waveforms, are shown in Table 1. The theoretical cell membrane response curves are illustrated as the thin lines in Figure 1. As predicted by this model and illustrated in Figure 1, the residual membrane voltage at the end of phase 2 for each waveform was again no significant difference between the E\textsubscript{50} of the 50/1.0 V\textsubscript{1} and the 70/1.0 V\textsubscript{1} waveforms (P=0.3836). For waveforms with phase-2 leading-edge voltage of 1.5 V\textsubscript{1}, the E\textsubscript{50} in the 30/1.5 V\textsubscript{1} waveform was higher than the 70/1.5 V\textsubscript{1} waveform (P=0.0019). Thus, the waveform with phase-1 tilt of 50% or 70% had lower E\textsubscript{50} of delivered energy than the waveform with the phase-1 tilt of 30% when phase-2 leading-edge voltage was constant.
fixed phase-1 tilt increased with increasing phase-2 leading-edge voltage. According to the charge-burping hypothesis, optimal DFT energies are obtained when phase 2 of the shock minimizes any residual cell membrane voltage. Figure 4 shows the relationship between E50 of delivered energy and residual membrane voltage at the end of phase 2. There is a significant correlation between the residual membrane voltage at the end of phase 2 and the measured E50 of delivered energy.

**Group 2**

Complete DFT data sets were obtained from 10 swine (35±6 kg). The waveform characteristics and the DFT parameters are detailed in Table 2.

**Defibrillation Energy**

Figure 5 shows the E50 of delivered energy in each waveform. For 30% phase-1 tilt waveforms, the E50 in the 30/0.5 V1 waveform was lower than in the 30/1.5 V1 waveform. For 50% and 70% phase-1 tilt waveforms, the E50 at phase-2 leading-edge voltages of 1.0 V1 (50/0.5 V1 versus 50/1.0 V1, P=0.0391; 70/0.5 V1 versus 70/1.0 V1, P=0.0414). However, the differences in E50 between the 50/0.5 V1 and 50/1.5 V1 waveforms (P=0.071) and between the 70/0.5 V1 and 70/1.5 V1 waveforms (P=0.087) did not reach statistical significance. Thus, the lowest E50 of delivered energy appeared to be associated with waveforms having longer phase-1 durations (50% and 70%) and larger phase-2 leading-edge voltages (1.0 V1).

When one compares the waveforms of differing tilts but the same 0.5 V1 phase-2 leading-edge voltage, the E50 for the 30/0.5 V1 waveform was higher than the corresponding E50 of the 70/0.5 V1 waveform (P=0.0095) but not different from that of the 50/0.5 V1 waveform (P=0.1373). There was no significant difference in E50 between the 50/0.5 V1 and the 70/0.5 V1 waveforms (P=0.2496). For waveforms with phase-2 leading-edge voltage of 1.0 V1, the E50 in the 30/1.0

For P values, repeated-measures one-way ANOVA was used. Pairwise comparisons of each defibrillation parameter were performed by the method of contrasts.

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**TABLE 2. DFT Parameters and Waveform Characteristics in Group 2**

<table>
<thead>
<tr>
<th>Waveform</th>
<th>Stored Energy, J</th>
<th>Delivered Energy, J</th>
<th>Phase-1 Leading-Edge Voltage, V</th>
<th>Phase-2 Leading-Edge Voltage, V</th>
<th>Phase-1 Impedance, Ω</th>
<th>Phase-2 Impedance, Ω</th>
<th>Residual Membrane Voltage, V</th>
<th>Phase-1 Pulse Width, ms</th>
<th>Phase-2 Tilt, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>30/0.5 V1</td>
<td>149±54§</td>
<td>82±29†</td>
<td>2112±392§</td>
<td>1056±196§</td>
<td>35±3†</td>
<td>56±7†</td>
<td>0.08</td>
<td>0.8±0.1§</td>
<td>94±3†</td>
</tr>
<tr>
<td>30/1.0 V1</td>
<td>135±43§</td>
<td>85±27§</td>
<td>1813±305§</td>
<td>1813±305§</td>
<td>37±3</td>
<td>55±6</td>
<td>0.12</td>
<td>0.8±0.1§</td>
<td>94±3</td>
</tr>
<tr>
<td>30/1.5 V1</td>
<td>140±58§</td>
<td>101±42§</td>
<td>1597±357§</td>
<td>2396±536§</td>
<td>37±3</td>
<td>49±5</td>
<td>0.32</td>
<td>0.8±0.1§</td>
<td>96±2</td>
</tr>
<tr>
<td>70/0.5 V1</td>
<td>91±31†</td>
<td>70±24*</td>
<td>1646±297†</td>
<td>823±148†</td>
<td>36±3†</td>
<td>56±8</td>
<td>0.21</td>
<td>1.5±0.1</td>
<td>95±2</td>
</tr>
<tr>
<td>70/1.0 V1</td>
<td>64±26</td>
<td>52±21</td>
<td>1247±255</td>
<td>1247±255</td>
<td>39±5</td>
<td>55±8</td>
<td>0.01</td>
<td>1.6±0.2</td>
<td>94±3</td>
</tr>
<tr>
<td>70/1.5 V1</td>
<td>64±18</td>
<td>55±15</td>
<td>1094±158</td>
<td>1641±236</td>
<td>41±7</td>
<td>53±7</td>
<td>0.19</td>
<td>1.7±0.3</td>
<td>95±3</td>
</tr>
<tr>
<td>70/0.5 V1</td>
<td>66±28</td>
<td>60±26*</td>
<td>1393±301†</td>
<td>696±151†</td>
<td>40±4</td>
<td>62±7†</td>
<td>0.27</td>
<td>2.8±0.3†</td>
<td>92±4‡</td>
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<tr>
<td>70/1.0 V1</td>
<td>46±16</td>
<td>43±15</td>
<td>1062±198</td>
<td>1062±198</td>
<td>40±4</td>
<td>55±8</td>
<td>0.07</td>
<td>2.9±0.3</td>
<td>95±2</td>
</tr>
<tr>
<td>70/1.5 V1</td>
<td>48±21</td>
<td>46±19</td>
<td>938±214</td>
<td>1407±321</td>
<td>43±9</td>
<td>55±9</td>
<td>0.13</td>
<td>3.1±0.7</td>
<td>94±3</td>
</tr>
<tr>
<td>30/1.5 V1</td>
<td>0.0001</td>
<td>0.0001</td>
<td>0.0001</td>
<td>0.0001</td>
<td>0.0013</td>
<td>0.0021</td>
<td>...</td>
<td>0.0001</td>
<td>0.0006</td>
</tr>
</tbody>
</table>

For P values, repeated-measures one-way ANOVA was used. Pairwise comparisons of each defibrillation parameter were performed by the method of contrasts.

*P<0.05 vs corresponding 1.0 V1 phase-2 leading-edge voltage waveforms.
†P<0.05 vs corresponding phase-2 leading-edge voltage 1.5 V1 waveforms.
‡P<0.05 vs corresponding 1.0 V1 and 1.5 V1 phase-2 leading-edge voltage waveforms.
§P<0.05 vs corresponding 50% and 70% phase-1 tilt waveforms.
||P<0.05 vs corresponding phase-1 70% tilt waveforms.
V₁ waveform was higher than that of the 50/1.0 V₁ (P=0.0001) and the 70/1.0 V₁ waveforms (P=0.0001). There was again no significant difference between the E₅₀ of the 50/1.0 V₁ and the 70/1.0 V₁ waveforms (P=0.2596). In a similar manner, for waveforms with phase-2 leading-edge voltage of 1.5 V₁, the E₅₀ in the 30/1.5 V₁ waveform was higher than the 50/1.5 V₁ waveform (P=0.0001) and the 70/1.5 V₁ waveform (P=0.0001). Thus, the waveform with the phase-1 tilt of 50% or 70% had lower E₅₀ of delivered energy than the waveforms with the phase-1 tilt of 30% for each phase-2 leading-edge voltage.

**Membrane Voltage**

The absolute residual membrane voltage at the end of phase 2 based on the charge-burping model for the test waveforms are shown in Table 2. The theoretical cell membrane response curves are illustrated as the thin lines in Figure 2. Figure 6 shows the relationship between E₅₀ of delivered energy and absolute residual membrane voltage at the end of phase 2. Similar to group 1, there is a significant correlation between the absolute residual membrane voltage at the end of phase 2 and the measured E₅₀ of delivered energy.

**Discussion**

This study investigated the effect of phase-1 tilt and phase-2 leading-edge voltage on external defibrillation efficacy with a 60-μF capacitor for phase 1 and either a 60- or 20-μF capacitor for phase 2. It appears that larger phase-1 tilts, at least up to 70%, were more efficacious than the smaller tilts of 30% and 50%. The relationship of phase-2 leading-edge voltage to defibrillation efficacy appeared more complex and depended on phase-2 capacitance. When the same capacitance was used for both phases (60-μF), a lower phase-2 leading-edge voltage (0.5 V₁) appeared to be the most efficient. However, when a smaller phase-2 capacitance of 20 μF was used, a higher phase-2 leading-edge voltage appeared to be best. In addition, the results confirmed that the charge-burping model can predict the optimal biphasic waveform for external defibrillation.

**Residual Membrane Voltage**

Recently, a quantitative charge-burping model has been proposed. In this model, the optimal phase 1 of the biphasic waveform should be identical to the optimal monophasic waveform, while the optimal phase 2 discharges the residual charges left on the cell membranes by phase 1. A study by Swerdlow et al supported this model by demonstrating that the optimal ratio of phase-1 pulse width to phase-2 pulse width depended on the relationship between the time constant of the shocking system and the time constant of myocardial cell membranes. This result was predicted by the charge-burping model. In this study, we calculated the membrane response curves in each waveform using the same charge-burping model. There was a significant correlation between the absolute residual membrane voltage at the end of phase 2 and the E₅₀ of delivered energy in both groups (Figure 4 and 6). This result suggests that minimizing the residual membrane voltage at the end of phase 2 as calculated by the charge-burping model correlates with the optimal phase 2 for generating the lowest delivered energy. The original charge-burping model postulated that biphasic waveforms may have little advantage over monophasic ones for external defibrillation. This hypothesis was based on the concept that internal defibrillation creates large voltage gradients in the heart, especially near the shocking electrodes, whereas external defibrillation would create relatively homogeneous voltage gradients within the heart. A few early experimental studies supported this hypothesis. Recent external defibrillation studies, however, have found that biphasic waveforms do indeed improve defibrillation efficacy. Furthermore, our study suggests that the charge-burping model reasonably predicted the defibrillation efficacy of a particular phase-1 waveform in combination with various phase-2 pulses. Thus, it would appear that discharging the residual membrane voltage at the end of phase 1 may still be important even with external defibrillation.

**Phase-2 Leading-Edge Voltage**

One recent internal defibrillation study compared defibrillation efficacy among 3 biphasic waveforms with equal phase-1 tilt at 65% but shorter phase-2 pulse width or smaller phase-2 leading-edge voltage. Defibrillation energy requirements were significantly increased for the waveform with a smaller phase-2 leading-edge voltage, whereas a short phase-2 pulse width did not influence defibrillation efficacy. This result suggested that the amplitude of phase-2 leading-edge voltage may be a more critical determinant than the phase-2 pulse width for defibrillation success of biphasic waveforms in humans.

Although the 0.5 V₁ phase-2 leading-edge voltage always had the lowest DFT in the 60/60-μF shocks (group 1), this relationship did not persist in the 60/20-μF waveforms (group 2). For the optimal phase-1 tilts in group 2, the best phase-2 leading-edge voltages were higher in the 1.0 to 1.5 V₁ range. Thus, when smaller phase-2 capacitors are used, the optimal phase-2 leading-edge voltage is higher than those waveforms when the same phase-1 and phase-2 capacitors are used.
Figure 7. Circuit model of charge-burping theory for external defibrillation. See text for abbreviations.

Limitations
The characteristics of the defibrillation waveform in this study depended on shock impedance. The typical patient impedance for external shock is 60 to 80 Ω, but the shock impedance in our study was ~40 Ω. Although the optimal phase-1 tilt value is 70% with a phase-1 pulse width of 3.0 ms in this study, this phase-1 pulse width will be longer in a clinical setting because of the higher impedance. Thus, these results may need to be verified in humans because of this difference impedance.

Conclusions
The major findings of the present study are as follows: (1) the 60/60-μF biphasic waveform with combination of phase-1 70% tilt and phase-2 leading-edge voltage of 0.5 times phase-1 leading-edge voltage provided the maximal defibrillation efficacy, (2) the 60/20-μF capacitor biphasic waveform with combination of phase-1 50% and 70% tilt and phase-2 leading-edge voltage of 1.0 and 1.5 times phase-1 leading-edge voltage provided the optimal defibrillation efficacy, and (3) the residual membrane voltage as calculated by the charge-burping model predicted the optimal small-capacitor external defibrillation biphasic waveform.

Appendix

Generalized Charge-Burping Theory: \( \phi_2 \) Independent of \( \phi_1 \)

Charge-burping theory and its associated equations describe the time course of the membrane potential of a cell during a biphasic shock pulse. Charge-burping theory hypothesizes that an optimal pulse duration for phase 2 (\( \phi_2 \)) is the duration that leaves as little residual membrane potential remaining on a cell affected by phase 1 (\( \phi_1 \)) as possible. Ideally, affected cells are set back to “relative ground” with \( \phi_2 \).

Generalized charge-burping theory for external defibrillation is based on the Kroll cardiac cell response model illustrated in Figure 7. The Kroll design equations are extended to account for the transthoracic resistance and a charge-burping theory and its associated equations describe the time course of the membrane potential of a cell during a biphasic shock pulse. Charge-burping theory hypothesizes that an optimal pulse duration for phase 2 (\( \phi_2 \)) is the duration that leaves as little residual membrane potential remaining on a cell affected by phase 1 (\( \phi_1 \)) as possible. Ideally, affected cells are set back to “relative ground” with \( \phi_2 \).

In the transthoracic extension of the Kroll model, the resistance variables are \( R_s \) =resistance of the defibrillator (including the electrode-electrolyte interface), \( R_{cw} \) =resistance of the thoracic cage, \( R_{cw} \) =resistance of the chest wall, \( R_{lp} \) =resistance of the lungs in parallel, \( R_{ls} \) =resistance of the lungs in series, and \( R_h \) =resistance of the heart. \( R_R \) represents the combined parallel and series resistances of the patient’s body. Also, \( C_1 \) represents the \( \phi_1 \) capacitor system, \( C_2 \) represents the \( \phi_2 \) capacitor system, and the pair \( C_a \) and \( R_h \) represent the membrane series capacitance and resistance of a single cell. The node \( V_n \) represents the voltage between the electrodes, and \( V_m \) denotes the voltage across the cell membrane.

The discharge of a capacitor system is modeled by \( V = V_ie^{-t/\tau} \) for an initial \( C_i \) capacitor system with a leading-edge voltage of \( V_i \).

Using the abstract circuit of Figure 7, an equation for \( V_m \) may be determined to be

\[
\frac{dV_m}{dt} + \frac{V_m}{\tau_m} \left( 1 - \frac{1}{\Omega_m} \right) = \frac{V_i}{\tau_m} e^{-t/\tau_1}.
\]

where \( \Omega_m \) and \( \Omega_s \) are non-linear resistance representations of the transthoracic resistive elements. \( \tau_m = R_m C_m \) represents the time constant of the myocardial cell in the circuit model, and \( \tau_1 = (R_s + R_h) \cdot C_1 \) represents the time constant of \( \phi_1 \). The ordinary differential equation (ODE) in Equation 1 models the effects of a transthoracic, monophasic, time-truncated, capacitor-discharged shock pulse on the myocardium. Equation 1 is an initial-value, first-order, linear differential equation. Assuming that the initial value for \( V_{m_i} \), is \( V_m(0) = V_m(0) \) (‘‘cell ground’’) and applying the initial condition to this equation, the solution to the equation for \( \phi_1 \), in terms of phase-1 cell potential \( V_{m_i} \), is

\[
2 V_{m_i}(t) = V_i \left( \frac{\tau_1}{\tau_m} \right) \left[ \frac{\tau_1}{\tau_m (1 - \frac{1}{\Omega_m}) - \tau_m} \right] e^{-t/\tau_1}.
\]

For \( \phi_2 \), an analysis identical to Equations 1 and 2 is derived. The differences are 2-fold. First, a biphasic waveform reverses the flow of current through the myocardium during \( \phi_2 \). Reversing the flow of current in the circuit model changes the sign on the current. The sign changes on the right hand side of Equation 1. Second, the \( \phi_2 \) portion of the waveform is assumed to be independent of \( \phi_1 \). Therefore, the \( \phi_2 \) ODE incorporates an independent leading-edge voltage, \( V_n \), for the \( \phi_2 \) portion of the pulse. Let \( \tau_2 \) represent the \( \phi_2 \) time constant. With these considerations, the \( \phi_2 \) ODE becomes

\[
\frac{dV_m}{dt} + \frac{V_m}{\tau_m} \left( 1 - \frac{1}{\Omega_m} \right) = \frac{-V_i}{\tau_m \Omega_m} e^{-t/\tau_2}.
\]

where \( \Omega_{m_i} = (R_m C_m) \) is the overall time of discharge for \( \phi_2 \) and \( V_{m_i} \) is the voltage left on the cell at the end of \( \phi_2 \). Applying the initial condition to equation 3, the solution for phase-2 cell potential \( V_{m_i} \) is

\[
V_{m_i}(t) = V_i \left( \frac{\tau_2}{\tau_m} \right) \left[ \frac{\tau_2}{\tau_m (1 - \frac{1}{\Omega_m}) - \tau_m} \right] e^{-t/\tau_2}.
\]

Equation 4 provides a means to calculate the residual membrane potential at the end of \( \phi_2 \) for those cells that were not depolarized by \( \phi_1 \). Equation 4 is set equal to zero and is solved for \( t \). The solution for \( t \) is the optimal charge-burping pulse duration for \( \phi_2 \) denoted by \( \Delta_t \). Arranging the exponential functions to 1 side and taking the logarithm of both sides, we solve for \( \Delta_t \) to get

\[
\Delta_t = \left( \frac{\tau_2 \tau_m}{\tau_m (1 - \frac{1}{\Omega_m}) - \tau_m} \right) \ln \left( 1 + \frac{(\Omega_m V_{m_i})}{\tau_2} \right).
\]

Acknowledgment
This work was supported by a grant from the SurVivaLink Corporation.

References


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Circulation. 1998;98:2487-2493
doi: 10.1161/01.CIR.98.22.2487

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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