TRANSTHORACIC ELECTRICAL IMPEDANCE DURING EXTERNAL DEFIBRILLATION: ASSESSMENT OF THE NONLINEARITY

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Abstract: *Heart fibrillation is a critical condition that requires immediate life-saving action by the application of a high-voltage pulse across the thorax of milliseconds duration. The 'classical' damped sine wave and biphasic truncated exponential pulses were applied during planned atrial cardioversion procedures in the National Centre of Cardiovascular Diseases on two groups of patients. The pulses meet a virtually pure resistance, which changes during the shock. Its nonlinearity is assessed from experimental data approximated by modelling and an equivalent circuit simulation. A characteristic time-course of the transthoracic resistance can be modelled by two exponentials, ascending and decaying respectively, with time constants and scale factor chosen to correspond to the experimental data, regardless on the defibrillation type waveform.*

This work was supported in part by the National Research Fund Grant No L-812.

1. Introduction

Transthoracic electrical defibrillation is achieved by applying a high voltage pulse on the patient chest by two electrodes. The resulting current must depolarize a large amount of myocardial cells in order to achieve defibrillation. For this reason, large electrodes are used with low-resistive conductive substance interface, placed on optimum locations on the thorax [1, 2].

The defibrillator load impedance, which includes the electrode-skin and the patient transthoracic impedance, is one of the principal defibrillation parameters. The importance of the load impedance in determining the defibrillating current amplitude and energy, therefore the defibrillation threshold, was shown by many authors [2, 3, 4]. The load impedance is virtually a pure resistance, recently reported by Tacker and Geddes [5], proven by the lack of observable phase difference between voltage and current waveforms. On the other hand, a certain nonlinearity was observed, usually explained as higher resistance met by lower current, caused by lower voltage. The transthoracic impedance was traditionally represented as a ratio of the peak shock voltage to the peak current [6, 7].

These considerations raised our interest in the transthoracic impedance characteristics and the corresponding measurement methods. The purpose of this work is to study the variations of the transthoracic impedance by continuous measurement techniques during the defibrillation shock and comparing the data with results obtained by mathematical modeling. An attempt was done for designing an equivalent circuit that could be used in development of defibrillators and studying various types of pulse waveforms.

2. Method

Two defibrillation waveforms are used – 'classical' monophasic damped waveform applied from a commercial defibrillator (Defigard 1002, Bruker Medical, France) and biphasic truncated exponential pulses, generated by an in-house developed instrument. Their corresponding output circuits are shown in fig.1(a) and (b). The voltage waveforms applied during defibrillation episodes are acquired by a 1000:1 voltage divider across the electrodes as is represented in fig.1(c). The signal is buffered and subjected to analogue-to-digital conversion with a sampling rate of 16,5 kHz and 16 bit resolution, in a highly isolated floating module, satisfying the electrical safety standards. The digitized signals are then transferred to a personal computer.

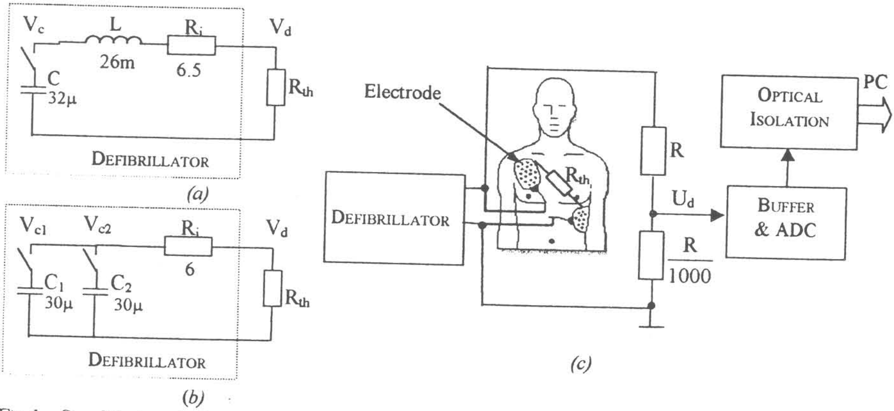

Fig.1 – Simplified equivalent circuits for (a) damped sinusoid and (b) biphasic exponential defibrillator. (c) - Block diagram for shock voltage acquisition

Mathematical modelling

The time-course of the transthoracic resistance R_{th} during the shock is obtained from the acquired voltage waveforms, approximated using equations derived from the corresponding defibrillator circuits [8].

In the case of the damped sine wave (C=32μF, L=26mH, R_i=6.5Ω), the voltage waveform is obtained from the equation:

$$V_d(t) = \frac{V_c R_{th}}{L(\alpha_1 - \alpha_2)}\left(e^{\alpha_1 t} - e^{\alpha_2 t}\right), \qquad (1)$$

where t is time , V_c is the initial voltage stored in the defibrillator capacitor, $\alpha_{1,2} = -\delta \pm \sqrt{\delta^2 - \omega_0^2}$, $\delta = (R_{th} + R_i)/2L$, $\omega_0^2 = 1/LC$, $\delta > \omega_0$ as $(R_{th} + R_i > 2\sqrt{L/C})$.

The biphasic truncated exponential pulses are obtained with capacitors $C_1 = C_2 = C = 30$ μF, with C_1 charged positive and C_2 - with negative polarity respectively. Here:

$$V_d(t) = V_c \frac{R_{th}}{R_{th} + R_i} e^{-t/\tau_d}, \qquad (2)$$

where $\tau_d = (R_i + R_{th})C$ - is the time constant of the defibrillator.

R_{th} is proven to change during the shock and is modelled by the following equation:

$$R_{th} = R_o + 2R_o e^{-t/\tau_1} + R_o(1 - e^{-t/\tau_2})/20\,, \qquad (3)$$

Here the resistance R_o is determined experimentally for each patient applying a fitting procedure. It consisted of iteratively varying the value of R_o, τ_1 and τ_2 in eqn (3), substituting after in eqn (1) and (2), until an acceptable coincidence (within an average difference of less than 50 V) with the acquired shock waveforms is reached. In the case of biphasic pulses the fitting is applied by varying R_{th} in eqn (3) for both phases independently.

Examples of fitting computed to experimental waveforms are shown in figure 3a and 4a, for damped sinusoid and truncated exponential pulses respectively.

Design Center simulation modelling

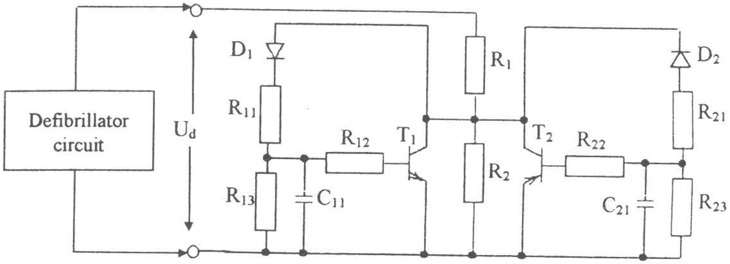

Fig.2. Equivalent circuit of the transthoracic resistance between the defibrillating electrodes.

This equivalent circuit was composed with the aim of reproducing the nonlinear behaviour of the transthoracic resistance. It corresponds adequately to both the damped sine wave pulse and to the biphasic exponential waveform.

3. Patients

Thoracic impedance measurements are performed on two groups of patients during planned application of transchest atrial cardioversion for treatment of sustained atrial fibrillation or flutter. Cardioversion is a procedure consisting in application of QRS-synchronised shocks, thus avoiding to disturb the ventricular beats and attempting to defibrillate the atria.

Group 1 included 9 patients: age 42 - 57 (Mean/SD=53/5), weight 56 - 150 (Mean/SD=86/29), height 162 - 200 (Mean/SD=176/13). Fourteen shocks were recorded with monophasic damped sinusoid pulses (in some of the patients it was necessary to apply more than one shock for achieving correct rhythm restoration).

Group 2 was of 14 patients: age 38 to 68 (Mean/SD = 61/10), weight 54 to 90 (Mean/SD = 80/15, height 155 to 180 (Mean/SD = 172/9). They were subjected to cardioversion using biphasic truncated exponential pulses. Thirty two shocks were recorded.

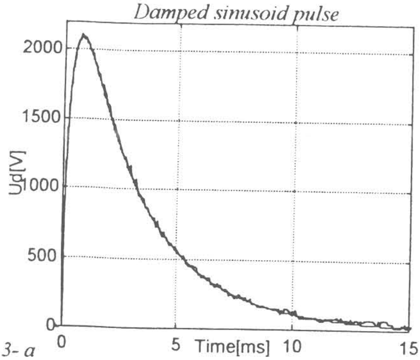

Fig 3- a

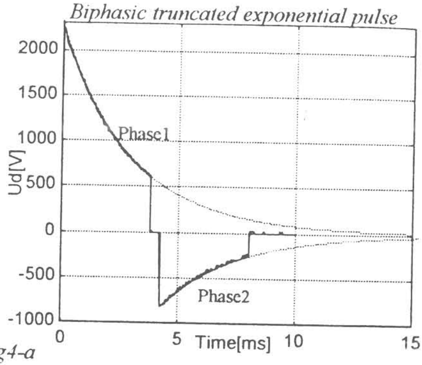

Fig4-a

Examples for fitting the acquired voltage to the mathematical model and the waveform derived from Design-Center simulation of the proposed equivalent patient circuit

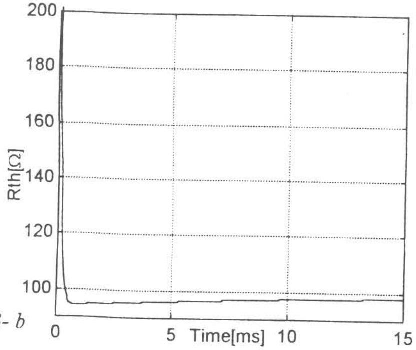

Fig 3- b

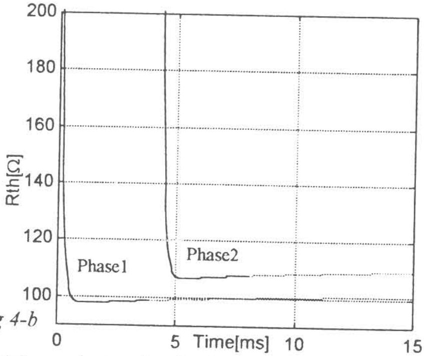

Fig 4-b

Time course of the transthoracic resistance during the shock

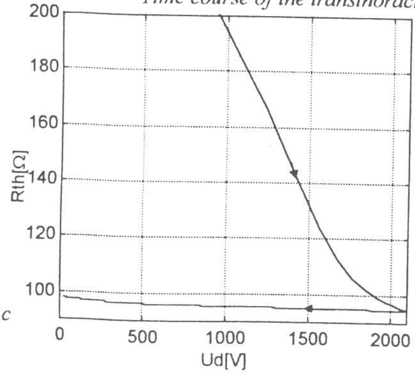

Fig3- c

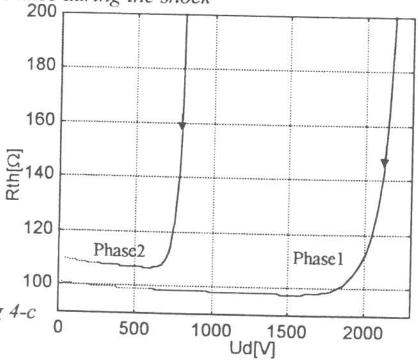

Fig 4-c

Dependence of the transthoracic resistance from the applied voltage

4. Results

Examples of mathematical and equivalent circuit approximation of the acquired damped sinusoid and truncated exponential pulses from patients defibrillated with energy of 100J are shown in Fig 3a and 4a. This energy corresponds to 2100 V and 2300/-800 V peak voltages applied to the patient for the respective defibrillation pulses. After applying mathematical modelling, the parameters in equation (3) were obtained as follows: R_o=94 Ω, τ_1=0.11 ms and τ_2=8 ms for the damped sinusoid pulse and $R_{ophase1}$ = 97 Ω, $R_{ophase2}$ = 106 Ω, τ_1=0.14 ms, τ_2=8 ms for both phases of the truncated exponential pulse. The time course of the transthoracic resistance during the shocks is presented in fig.3b and 4b. The dependence between the applied defibrillating voltage and the transthoracic resistance for each type waveform is shown in fig. 3c and 4c. The behavior of the approximated waveform and its resistance during full capacitor discharge is presented with dotted lines for the truncated exponential pulse. The proposed mathematical fitting procedure applied to all patients yields: R_o=(88.5± 11) Ω - damped sinusoid pulse, $R_{ophase1}$=(91.7± 14.9) Ω and $R_{ophase2}$=(97.8± 18.7) Ω - biphasic truncated exponential pulse, τ_1=(0.136±0.03) ms and τ_2=8 ms. The values of R_o, $R_{ophase1}$ and $R_{ophase2}$ are not statistically different ($p>0.05$).

The fitting of the acquired voltages with the Design Center simulation of the circuit in fig.2 is also presented in fig.3a and 4a. The circuit parameters obtained after iterative approximation are: $R_{11}=R_{21}$=800Ω, $R_{12}=R_{22}$=1kΩ, $R_{13}=R_{23}$=400Ω, R_2=280Ω, $C_{11}=C_{21}$=2μF, R_1=70Ω for the sine wave and R_1=68Ω for the biphasic exponential pulse.

5. Discussion

The transthoracic resistance nonlinearity has been observed by many researchers [6, 7]. However, the underlying mechanisms were not investigated and revealed. Our attempt at modelling the time-course of the resistance changes was undertaken with the aim of suggesting some basic relationships. The transthoracic resistance time course was modelled by two exponentials – ascending and decaying, regardless of the used waveform. The influence of the first time constant is observed in the initial part of the waveform with duration of about 500μs, when the transthoracic resistance changes very sharply and with a ratio of about three, as shown in fig.3b and 4b. For the remaining pulse duration the thransthoracic resistance increases slightly. The resistance strongly decreases during the smooth initial part of the sine wave defibrillation waveform until the peak is reached (fig. 3c). After that it changes negligibly, during the decaying part of the voltage. This phenomenon is observable as a hysteresis in the voltage-resistance curve of fig.3c. In the case of direct capacitor discharge, the hysteresis cannot be observed because the applied voltage front edge is very sharp. It can be seen in fig. 4c, that the transthoracic resistance decreases as the voltage decays. It could be speculated that the physiological processes concerning the tissue resistance changes do not depend on the applied defibrillation waveform. The time constant τ_1 represents the physiological response of the cell membrane electroporation after applying a strong electrical shock. This time constant, with a mean value of about 136 μs, slightly varies among patients, regardless of the defibrillation waveform. As the applied voltage decreases, the resistance slightly increases possibly because of the recovery processes in the cell membrane. They are described with the time constant τ_2=8 ms, multiplied by a factor equal to 1/20 of the minimum transthoracic resistance.

The proposed in fig. 2 equivalent circuit reproduces well the nonlinearity in the transhoracic resistance, as it can be seen in fig. 3a and 4a. It consists of a constant resistance, designated by R_1 and a voltage dependent resistance – R_2, shunted by the collector-emitter junction. Its resistance is controlled by the capacitor $C_{11}(C_{21})$ charge and discharge voltage, with time constants chosen to correspond to the experimental data. The circuit is applicable for biphasic pulses. The diodes D_1 and D_2, connected in opposite polarity, control one of the two transistors – NPN (T_1) with positive and PNP (T_2) with negative defibrillation pulse. This configuration corresponds to the tissue two-way conductive structures. The lower voltage of the second phase results in higher resistance, as the transistor control voltage is lower and decaying.

6. Conclusion

The investigation of the time-course of the transthoracic resistance changes by comparing experimental to model data showed that it can be represented by two exponential functions, a decaying and an ascending. The modelling allowed to design an electrical equivalent circuit, which adequately reproduces the nonlinearity and is envisaged for use in designing defibrillators with various pulse waveforms.

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