Functional Damage to the Heart Caused by Monophasic and Biphasic Defibrillation Waveforms

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Although external cardiac defibrillation is generally recognized to be highly effective, repeated discharges of submaximal or maximal energy may lead to marked disturbances of myocardial contraction and relaxation [2-4], postcardioversion arrhythmias (including fibrillation) [1,9-11], and electromechanical dissociation and/or asystole [2,10], as well as to serious morphological lesions in the myocardium, including infarction [8,10]. The quoted studies, however, are mainly concerned with monophasic defibrillation waveforms widely used in the West, whereas more effective, but less well studied, biphasic waveforms are used in countries of the former USSR.

In the present study, functional damage caused to the heart by monophasic waveforms (Edmark pulses) were compared to those caused by asymmetrical quasisinusoidal biphasic waveforms (Gurvitch pulses).

MATERIALS AND METHODS

The study was conducted on three groups of nembutal-anesthesized (30 mg/kg) dogs 4-31 kg in weight artificially ventilated with room air. In group 1 (12 dogs weighing 6-13.5 kg), noncardiosynchronized Edmark and Gurvitch pulses were delivered alternately at 3-5-minute intervals to the normally contracting heart via thoracic electrodes 10 cm in diameter. In group 2 (9 dogs weighing 4-8 kg), these pulses were delivered in the same way via electrodes 4.5 cm in diameter. The criterion of functional cardiac damage was taken to be reversible ventricular asystole (VA). In both groups, asystole duration and the time when the normal sinus rhythm (NSR) reappeared were recorded in seconds. Dogs of group 3 (n=100), weighing 8-31 kg, were delivered alternating subthreshold and threshold pulses, the latter pulses resulting in the appearance on the electrocardiogram (ECG) of 1 to 3 pathological (or aberrant) ventricular complexes.

In all animals, arterial blood pressure and the ECG in three standard leads were recorded, as were the following pulse parameters: transthoracic peak voltage and current (I_1 - main positive current wave, I_2 - negative wave of the Gurvitch pulse in amperes; I_2/I_1 ratio = 0.55), and pulse duration. The released energy (E in joules) and chest resistance (R_1 in Ohms) were calculated. For the recordings, a polygraph (San Ei Instruments, Japan) and a digital C9-16 oscillograph with memory were used. The defibrillators employed were DI-03 to deliver Gurvitch pulses and a Life-Pak defibrillator/monitor/registrator (USA) to deliver Edmark pulses.

RESULTS

Transthoracic Edmark pulses of 2.0-5.8 A/kg (group 1) and 1.9-4.8 A/kg (group 2) caused, in

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TABLE 1. Functional Damage Caused to the Nonfibrillating Heart by Monophasic (Edmark Pulse) and Biphasic (Gurvitch Pulse) Waveforms in Relation to the Diameter (*d*) of Defibrillator Electrodes. The Values are Means±SEM

Waveform	I_1	I_2	I_{1+2}	E	R_1	VA	NSR
			d=10 cm (group 1; n=12)				
Biphasic	30±2	15±1	45±2	147±10.8	51±2.5	1.0±0.2	7.8±1.0
Monophasic	29±1.8	—	—	103±9.9	53±2.8	5.8±0.7	14.0±2.9
p				<0.05		<0.001	<0.05
			d=4.5 cm (group 2; n=9)				
Biphasic	17.8±2	8.6±1	26±3	94.7±14	102±9.9	2.9±1.1	9.0±3.3
Monophasic	17.2±2	—	—	87.0±12	104±6.0	14.0±5.0	31.0±6.7
p						<0.02	<0.01

all dogs, the appearance of reversible VA with preserved *P* waves. The asystole lasted for 3-11.5 sec in group 1 and 4-45 sec in group 2. The restoration of NSR occurred via an atrioventricular block of degree II_{3-1} in 5-38 sec in group 1 and 6-79 sec in group 2.

Gurvitch pulses induced VA in 8 of the 12 dogs (67%) of group 1 (I_1=2.0-5.5 A/kg) and in 5 of the 9 dogs (57%) in group 2 (I_1=2.0-4.7 A/kg). Both the duration of VA and the time taken for NSR to reappear were much shorter - 1-3 sec and 3-15 sec in group 1, respectively, and 3-6.5 sec and 6-27 sec in group 2. The coefficients of correlation between the duration of VA and the I_1/body weight ratio were 0.63-0.69 in the groups (p<0.05) and 0.82-0.93 in individual dogs (n=12) with repeated discharges (p<0.01). As seen in Table 1, VA in dogs of group 2 was induced by a peak current 1.7 times weaker than in those of group 1. This was primarily due to the use of narrower electrodes, which resulted in an increased density of the current and increased the amount of current passing directly through the heart. It should be noted that intragroup differences in the length of VA and the time when NSR reappeared increased significantly with the use of narrower electrodes and the growth of R_1; thus, they were 11 sec and 22 sec, respectively, in group 2 vs. 4.8 sec and 6.2 sec in group 1. These differences appear to have been due to a considerably longer (1.8-fold) duration of the Edmark pulse and its altered form as compared to the more stable Gurvitch pulse [5].

In group 3, the Edmark pulse (I_1=0.7-2.5 A/kg) caused the development of ventricular fibrillation in 15 cases in 13 dogs out of the 100, whereas the Gurvitch pulse did so only in 4 cases in 3 dogs.

The results of this study indicate that biphasic waveforms (Gurvitch pulses) elicit less life-threatening disturbances of cardiac function than monophasic waveforms (Edmark pulses). These results may be explained by accepting Jones' hypothesis [6,7] that the second phase of an asymmetrical biphasic waveform of particular parameters promotes a more rapid elimination of transitory microlesions in cell membranes and a faster restoration of the transmembrane potential.

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