714 Cardiac Defibrillation: Physiologic Observations

Monday, March 20, 1995, 2:00 p.m.–3:30 p.m.
Ernest N. Morial Convention Center, Room 26

2:00

714-1 Interaction Between Strong Electrical Stimulation and Reentrant Wavefronts in Canine Ventricular Fibrillation

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To test the hypothesis that the effects of a strong electrical stimulus on reentrant wavefronts (RW) in ventricular fibrillation (VF) is dependent upon the timing of the stimulus, we studied 6 open-chest dogs with computerized mapping techniques. A plaque electrode array with up to 509 bipolar electrodes was placed on the RV epicardium. The interelectrode distance was 1.6 mm and the interpolar distance was 0.5 mm. Following 8 baseline stimuli (S_1) at 300 ms cycle length, a strong premature stimulus (S_2) was given to the center of mapped tissue to induce VF. In subsequent episodes, a second premature stimulus (S_3) of the same strength as S_2 was given to the same site at progressively longer S_2–S_3 intervals with 20 ms increments. *Results:* At baseline, S_2 consistently induced figure 8 reentry and VF. The VF cycle length immediately after S_2 averaged 108 ± 17 ms. The S_3 resulted in one of the following responses: (I) termination of RW and VF, (II) induction of different RW, or a focal pattern of repetitive activation, and (III) persistence of the same figure 8 RW. The intervals between S_3 and the immediate preceding activation at the site near S_3 were 41 ± 18 ms, 60 ± 18 ms and 99 ± 14 ms ($p < 0.001$), for response patterns I, II and III, respectively. We conclude that the effects of strong electrical stimulation on the RW in VF is dependent upon the recovery interval since the previous local activation. A protective zone occurred first, during which a strong electrical stimulation can terminate RW and abort VF. This zone was followed by a vulnerable period during which new wavefronts could be induced. If the S_3 was given after the end of the vulnerable period, there was no change of the RW.

2:15

714-2 Diffuse Cardiac Sympathetic Dysfunction Follows Implantable Defibrillator Shocks Delivered Through Epicardial Patch Electrodes

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Decreased efferent sympathetic neural function has been demonstrated following >10J DC shocks delivered through epicardial patch electrodes in dogs. To evaluate the effect of DC shocks on cardiac sympathetic innervation in humans, we performed I-123-metaiodobenzylguanidine (MIBG) scintigraphy in 5 patients (age 46–73 years, mean 64) prior to and after receiving shocks from implanted cardiac defibrillators (ICD). Four patients had coronary artery disease (CAD) with prior myocardial infarction and one had an idiopathic dilated cardiomyopathy (IDCM). All patients had spontaneous and inducible ventricular tachycardia. One patient was receiving amiodarone at the time of this study. One patient had had prior coronary artery bypass surgery (CABG), and two had concurrent CABG at the time of ICD implantation. This study was performed during an ICD generator change in 2 patients and at the time of initial ICD implantation in 3 patients. Four patients (3 CAD, 1 IDCM) received epicardial patch electrodes, and one patient had a transvenous lead system. Baseline MIBG and thallium-201 scintigraphy performed >1 week preoperatively and remote from any cardioversions or shocks revealed focal areas of reduced MIBG uptake in areas of thallium perfusion defects in all patients. Repeat MIBG scans were performed <four hours after shocks delivered during either the surgical implantation or follow-up EPS. All patients had received at least one 24J shock (mean 2.4 shocks, range 1–4). In all patients with epicardial patch electrodes, there was minimal or no cardiac uptake of MIBG post-shock, consistent with diffuse sympathetic dysfunction. In the patient with a transvenous lead system the post-shock MIBG scan was unchanged from baseline. This study demonstrates that following DC shocks delivered through epicardial patches there is diffuse cardiac sympathetic neural dysfunction, which may contribute to the arrhythmic "storm" that has been reported following some ICD implantations.

2:30

714-3 Defibrillation Using a Series of High-Frequency Monophasic Current Pulses

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Chopping a defibrillation waveform into a series of high-frequency pulses may be a method to alter its effective shape. In seven pentobarbital anesthetized dogs, we measured defibrillation current (I50) using a 10 msec long series of high-frequency (HF) pulses. HF waveforms consisted of monophasic rectangular current pulses at 1 KHz to 20 KHz with 50% "on-time" duty cycle. Control I50s were measured for 5 and 10 msec continuous pulses. Data were normalized by the current (2.61 ± 0.51 amps) required for the 10 msec controls. Resistance was the same for HF (71Ω) and continuous (75Ω) pulses. I50 did not change with frequency (anova, $p = 0.55$) but all HF required twice (1.96 ± 0.17) the current of the 10 msec control. The 5 msec control required 1.27 ± 0.16 times the 10 msec control current.

These data demonstrate that defibrillation was governed by the time-averaged current since HF and 10 msec control waveforms required equal time-averaged currents but HF required twice the actual current and energy. Waveform duration was also important since HF waveforms were "on" for 5 msec but required 1.55 ± 0.16 times the 5 msec control current. These data also suggest that the heart defibrillates as though the current pulses were "filtered" with a time constant greater than 1 msec although no actual filtering was observed. Such filtering might help explain the role of waveshape on defibrillation efficacy and could be utilized in HF series to create arbitrary effective waveform shapes.

We conclude that HF defibrillation is possible; that time-averaged current governs I50 for HF waveforms ≥1 KHz; and that HF chopping may permit waveform shapes to be effectively altered.

2:45

714-4 What is the Optimal Pulse Duration for Defibrillation? Insights from a Porcine Animal Model

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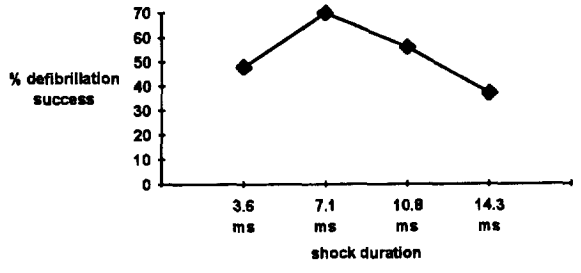
We investigated the effect of modifying total biphasic shock duration (SDur) in 7 anesthesized pigs while keeping pulse 1/pulse 2 duration ratio constant (60%/40%; pulse 1 = first part, pulse 2 = second part of biphasic shock). Different SDurs of 10.8 ms (pulse 1: 6 ms/interpulse delay: 0.8 ms/pulse 2: 4 ms), 7.1 ms (3.8/0.8/2.5 ms) and 3.6 ms (1.7/0.8/1.1 ms) generated by an external capacitor (Medtronic 2349®) were tested in a randomized protocol against a SDur of 14.3 ms (8.1/0.8/5.4 ms) used by a currently available defibrillator system (CPI ECD®). 5 shocks were delivered at each energy level between 5 and 40 Joules (J) in 5 J steps through a transvenous/subcutaneous lead system (CPI Endotak C®/SubQ®). Voltage and current were recorded on an oscilloscope and impedance calculated as voltage divided by current. *Results:* Cumulative success at all energy levels was higher with 7.1 ms SDur (70%) than with 10.8 ms (56%, $p < 0.01$), 14.3 ms (37%, $p < 0.001$) and 3.6 ms (48%, $p < 0.001$). SDur of 10.8 ms were significantly more successful than 14.3 ms ($p < 0.001$; χ^2-test). Shocks of 14.3 ms had a significantly lower impedance at the trailing edge of pulse 2 compared to shorter SDur (31.6 ± 9.3Ω vs. 37 ± 7.9Ω/10.8 ms, 36.2 ± 7Ω/7.1 ms and 35 ± 9Ω/3.6 ms).



Conclusions: 1) In analogy to chronaxy in electrostimulation there is an optimal SDur for defibrillation. 2) In this animal model the optimal SDur for biphasic shocks is considerably shorter than the one used with currently available defibrillator systems. 3) This may be due to a significantly lower impedance at the trailing edge of pulse 2 for longer SDur possibly leading to refibrillation. 4) Shorter SDurs may allow the use of smaller capacitors in future clinical devices.

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