Effects of Postshock Atrial Pacing on Atrial Defibrillation Outcome in the Isolated Sheep Heart

A.C. Skanes, MD; R.A. Gray, PhD; C.L. Zuur; J. Jalife, MD

Background—Failed atrial defibrillation shocks are associated with organization of postshock activity and a substantial postshock electrical quiescence. We investigated the ability of a train of pacing stimuli to capture or locally entrain atrial myocardium during the quiescent period after low-energy shocks and to alter defibrillation outcome.

Methods and Results—High-resolution video imaging of near-defibrillation-threshold atrial shocks was performed in 12 Langendorff-perfused sheep hearts. A train of 10 pacing stimuli (10-ms pulse width, 200-ms cycle length) was coupled to the shock at various delays in 7 hearts. Coupling intervals of 40 to 130 ms were investigated for feasibility of capture of the first pacing stimulus. The success rate of capture was 0.08±0.08, 0.43±0.13, 0.73±0.13, and 0.11±0.1 for 40-, 60-, 80-, 100-, and 120-ms coupling intervals, respectively (P<0.001). In 5 experiments, the coupling interval was fixed at 100 ms (highest success, see above), and the pacing stimulus amplitude was varied between 1.0, 2.0, and 4.0 V. Successful capture rates were 0.38±0.08, 0.75±0.08, and 0.64±0.08, respectively (P<0.003 for 1.0 versus 2.0 V, P=0.2 for 2.0 versus 4.0 V). Rates of successful defibrillation for the groups without and with pacing were 0.56±0.07 and 0.64±0.04, respectively (P=0.3). With capture of the first pacing stimulus, the rate of successful defibrillation rose to 0.75±0.05 (P<0.01); it remained unchanged without capture (0.48±0.07 versus 0.56±0.07 for no pacing).

Conclusions—Pacing during the quiescent period that follows defibrillation shocks is feasible. A pacing train whose first stimulus successfully captures during the quiescent period of near-defibrillation-threshold shocks appears to alter the outcome. (Circulation. 1998;98:64-72.)

Key Words: pacing • electrophysiology • defibrillation • atrium
areas of the atria. Hence, large areas of atrial myocardium could come under local control. We further hypothesized that should this occur, the outcome of near-threshold but unsuccessful shocks could be altered and result in a successful outcome. In essence, a low-energy shock would serve to organize the atrial activity for a pacing train to progressively entrain the atria. Some of the results of this study have been reported elsewhere in abstract form.15

Methods

Langendorff-Perfused Sheep Heart Preparation
Young sheep of either sex (18 to 25 kg) were anesthetized with sodium pentobarbital (35 mg/kg). The heart was rapidly removed through a midline sternotomy, then connected to a Langendorff apparatus. This method has been described elsewhere in detail.14,16 Briefly, the coronary arteries were continuously perfused via a cannula in the aortic root with warm (36°C to 38°C) Tyrode’s solution buffered to a pH of 7.4, under a constant flow of 115 to 140 mL/min, and bubbled with 95% O2 /5% CO2. We ensured that the heart was in sinus rhythm and contracting forcefully and rhythmically at the initiation of the experiment. Two defibrillation catheters with 6-cm2-area coil electrodes (InControl Inc) were inserted via the venae cavae to the right atrial appendage and the coronary sinus. A custom-made programmable defibrillator (InControl Inc) was used to deliver a biphasic shock (duration of each phase was 3 ms). AF was induced by burst rapid atrial pacing from the epicardial surface of either the right or left atrium after the addition of ACh in a concentration of 10−6 mol/L, to facilitate the induction of sustained AF. AF was considered sustained if it lasted 2 to 5 minutes. To stop the contraction of the heart and thus record the fluorescence associated with the electrical activity in the absence of mechanical artifacts, we added methoxyverapamil to the Tyrode’s solution at a final concentration of 2×10−6 mol/L, which, in addition, significantly reduced the sinus rate and resulted in complete atrioventricular block. After this, a bolus injection of 5 to 10 mL of the fluorescent dye di-N-ethylaminoethyl-p-phenylendiamine dihydrochloride (6NEPPS, 7.5 μg/mL) dissolved in DMSO was injected via the perfusion cannula into the coronary arteries. This enabled us to use video imaging to record the transmembrane potentials simultaneously from >20 000 sites on the epicardial surface of the right atrium in the absence of mechanical artifacts and without interference by ventricular activation.

Recording

High-Resolution Optical Mapping
The video imaging approach used for these studies has been described in detail elsewhere.14,16 Briefly, the light from a tungsten-halogen lamp was collimated and made quasi-monochromatic by the use of an interference filter (590 nm) together with a heat filter. The light was aimed directly onto the epicardial surface of the vertically hanging heart. A 50-mm objective lens was used to collect the emitted light with a depth of field of ~12 mm. The emitted light was transmitted through the emission filter (590 nm) and projected onto a CCD video camera (Cohu 6500). Our video camera was set to run in an asynchronous reset mode at an acquisition rate of 120 frames per second (sampling at 8.33-ms intervals). The video images (typically 200×100 pixels) of the epicardium of the right atrium were acquired with an A/D frame grabber (Epix) in a noninterlaced mode. The frame grabber board was mounted on a Gateway Pentium computer, which was used to process the imaged data.

Electrogram
A continuous atrial electrogram was recorded as the difference between 2 epicardial leads, 1 located on the right atrium and 1 on the left atrium. The electrodes were connected to a Gould amplification system and filtered at 0.1 and 300 Hz.

Feasibility of Pacing Immediately After the Shock

Delay of Pacing After the Shock
In the initial 7 experiments, we addressed the ability of the first paced beat in the train to capture after the shock at increasing coupling intervals. To investigate this, it was necessary that several parameters be fixed. Therefore, the pacing train consisted of 10 square-wave pacing stimuli, 10 ms in duration, at 200-ms pacing intervals. In these experiments, several coupling intervals (between shock and first paced beat) were randomly tested in a balanced approach: 0 (ie, no pacing), 40, 50, 60, 70, 80, 100, 120, and 130 ms. The pacing amplitude in these initial experiments was varied between 1.0 V (~2.5 to 3.5 times diastolic threshold at a basic cycle length of 400 ms) and 2.0 V (~5 to 6 times diastolic threshold). Pacing was performed from the epicardial surface of the free wall of the right atrium adjacent to the sulcus terminalis at its intercaval region. Successful capture of the first paced beat was assessed on the basis of the following 3 criteria via optical mapping recordings: (1) earliest activation occurring at the site of the pacing stimulus, (2) propagation of the wave front away from the pacing site to rule out the possibility of a breakthrough near or adjacent to the pacing site, and (3) timing of the propagating wave in relation to the pacing stimulus. Preliminary experiments demonstrated that the latency of successful capture pacing stimuli could be as long as 40 ms (5 frames at 8.3 ms per frame). Timing of the pacing stimulus was marked during the optical recordings by use of a red LED timed to the pacing stimulus. Those pacing stimuli that did not capture were divided into 2 types based on the mechanism of failure to capture. Type I NC occurred when the impulse did not capture secondary to postshock refractoriness. Type II NC was designated as such if external wave fronts invaded the region of the pacing stimulus before the timing of the stimulus; ie, the local tissue was refractory as a result of immediately preceding activation.

Pacing Amplitude
After the results of the initial experiments, which tested timing of the pacing stimulus to the ability to capture, pacing amplitude of the pacing stimuli was varied to study its effect on the ability to capture after the shock in 5 more experiments. These studies were performed at 100-ms delay, the optimal delay as determined in the initial experiments (see Figure 2). Pacing amplitude was randomly varied between 0 (ie, no pacing), 1.0, 2.0, and 4.0 V in a balanced approach.

Assessment of Outcome
To examine the effect of pacing on shock outcome, the success rate of shocks with and without pacing were compared. Protocols included a defibrillation trial with no pacing for each series of defibrillation trials. This allowed direct comparison of outcomes for the pacing and no-pacing groups. Because the video imaging technique is immune to shock artifact, we were able to determine whether the first pacing stimulus successfully captured. Therefore, the pacing group was further stratified into those trials in which the first pacing stimulus successfully captured and those in which it did not capture; the success rates of these groups were also compared.

Statistical Analyses
Continuous variables are reported as mean±SD and probabilities as proportions±SD. Continuous variables were compared by use of Student’s t tests, and probabilities were compared by χ² analysis.
Results

Mapping PSA

In their original optical mapping study of the effects of atrial defibrillation on wave propagation, Gray et al. described a period of \(110\) ms after the shock during which no atrial activity was manifest. Thereafter, a short run of PSA waves could appear in succession and be followed either by quiescence and then sinus rhythm or by immediate resumption of AF. Similar results were obtained in our experiments.

The data illustrated in Figure 1 were taken from an episode of AF immediately before and after an unsuccessful defibrillation shock. Panel A shows the atrial electrogram obtained as the difference between the 2 epicardial leads, 1 located on the right atrium and the other on the left atrium. In Panel B we present 3 isochrone maps obtained from the anterior surface of the right atrial free wall, as shown by the gray region in the top left diagram. The map on the top right corresponds to activity before the shock. Notice the complex sequence of activation, with multiple activation wave fronts and epicardial breakthroughs emerging, colliding, and mutually annihilating. The other 2 maps show the activation sequence during the first (middle) and second (right) PSAs, which appeared on the right atrial free wall at \(t = 75\) ms and \(t = 175\) ms, respectively, after the shock. During the first PSA, the mapped area was activated by a broad and homogeneous wave front that emerged near the lower portion of the sulcus terminalis (see diagram of preparation) and activated the entire right atrial anterior wall within 40 ms. The second PSA also emerged from the lower portion of the sulcus terminalis but moved somewhat more slowly, following a more tortuous route. In subsequent beats, activation became increasingly disorganized, and AF was reinitiated.

In Figure 2, similar information is presented for a shock that resulted in successful defibrillation after 3 PSAs (labeled 1, 2, and 3). In the electrogram, irregular baseline activity after successful atrial defibrillation represents ongoing VF. As in Figure 2, the postshock impulses activate the right atrial free wall as a homogeneous broad wave front compared with the complex activation pattern of AF immediately preceding the shock. Similar results were obtained in all experiments surveyed. Overall, in 12 hearts, the quiescent period between the shock and the first PSA was \(97\pm27\) ms (\(n = 12\)), which was somewhat shorter than in the experiments of Gray et al. The difference is most likely due to the presence of ACh in this series of experiments, whereas it was absent in the previous study.

Feasibility of Pacing After the Shock

Mapping the Pacing-Induced Wavefronts

In Figure 3A, we present an electrogram obtained from an experiment during the application of a defibrillating shock that was followed by pacing at the lower left border of the tissue, near the sulcus terminalis (see asterisks in maps of panel B). Clearly, the first as well as all subsequent pacing stimuli (10 total) captured the atrium. This resulted in
successful defibrillation. As in Figure 2, VF was seen after successful atrial defibrillation. Eight-millisecond isochrone maps of the first 2 paced beats after the shock are shown in Figure 3B. The asterisks designate the site of pacing. These stimuli resulted in broad wave fronts that activated the entire right atrial free wall in 66 and 58 ms, respectively. Pacing was not followed by resumption of fibrillation, and none of the stimuli gave rise to unidirectional block or initiated reentry over the right atrial free wall even though the first paced beat occurred 100 ms after an 80-V (0.25-J) shock and subsequent ones occurred at relatively brief cycle lengths (200 ms). In fact, in all experiments, despite successful capture of pacing stimuli of various amplitudes between 60 and 130 ms after shocks that ranged from 0.03 to 3.17 J (see below), in no case did the wave front propagating from a successfully captured pacing stimulus result in reentry within the imaging area. Although it is difficult to ensure that propagation of these beats did not induce reentry elsewhere in the atria, beyond our field of view, it seems unlikely, because successful capture of the first paced beat was associated with successful outcome and therefore was unlikely to have been proarrhythmic in nature.

**Success of Capture for Each Coupling Interval**

<table>
<thead>
<tr>
<th>Delay (ms)</th>
<th>V</th>
<th>n</th>
<th>Capture</th>
<th>% Capture ± SD</th>
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<tr>
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<td>8</td>
<td>0</td>
<td>0</td>
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<tr>
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<td>2</td>
<td>14</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>1 and 2</td>
<td>22</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>60 and 70</td>
<td>1</td>
<td>9</td>
<td>2</td>
<td>0.22±0.14</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>12</td>
<td>1</td>
<td>0.08±0.08</td>
</tr>
<tr>
<td></td>
<td>1 and 2</td>
<td>21</td>
<td>3</td>
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<tr>
<td>80</td>
<td>1</td>
<td>13</td>
<td>3</td>
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<tr>
<td></td>
<td>2</td>
<td>14</td>
<td>6</td>
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</tr>
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<td>9</td>
<td>0.33±0.09</td>
</tr>
<tr>
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<td>10</td>
<td>4</td>
<td>0.40±0.15</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>11</td>
<td>8</td>
<td>0.73±0.13</td>
</tr>
<tr>
<td></td>
<td>1 and 2</td>
<td>21</td>
<td>12</td>
<td>0.57±0.11</td>
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<td>1</td>
<td>7</td>
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</tr>
<tr>
<td></td>
<td>2</td>
<td>9</td>
<td>1</td>
<td>0.11±0.10</td>
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<td></td>
<td>1 and 2</td>
<td>16</td>
<td>1</td>
<td>0.06±0.06</td>
</tr>
</tbody>
</table>

**Coupling Interval**

In the first group of 5 experiments, we analyzed the ability of the first paced beat to capture after the shock as a function of the coupling interval. The first stimulus of the pacing train was coupled to the shock at intervals of 40, 50, 60, 70, 80, 100, 120, and 130 ms. Success of capture for each coupling interval is tabulated in the Table and represented graphically in Figure 4A. From these data, it is evident that as the coupling interval of the first pacing stimulus increased, the ability to successfully capture increased, until the success rate peaked at 100 ms. The success rate subsequently decreased dramatically at 120 ms. At these pacing amplitudes, the maximal capture rate was 0.73±0.13 at a delay of 100 ms and
pacing amplitude of 2.0 V. The data suggested 2 different mechanisms by which pacing stimuli at each coupling interval failed to capture, depending on the coupling interval. We labeled these mechanisms “type I” and “type II.” In type I, the first pacing stimulus failed to capture at short coupling intervals because its occurrence was too early after the shock. In type II, at long coupling intervals, interference by postshock activity reduced the ability of the first pacing stimulus to capture. We compared the rate of type I versus type II NC at each coupling interval. This is represented in Figure 4B, in which we have plotted the number of each type of NC as a percentage of the total number. The percentages at each coupling interval therefore add to 100%. At short coupling intervals, the pacing stimulus was unable to capture because of postshock refractoriness; at 40 ms, 94.9±3.5% of the failures to capture were type I. As the coupling interval was increased, the mechanism of NC shifted to a greater proportion of type II; ie, at 100 ms, 64±6.6% of the failures to capture were due to encroachment by a postshock wave front. At longer coupling intervals, the relative frequencies of these phenomena did not appear to change. The crossover point of these curves (see Figure 4B) occurred between 80 and 100 ms.

**Pacing Amplitude**

In these initial experiments, at a pacing amplitude of 2.0 V and a coupling interval of 100 ms, ≈73% of the pacing stimuli captured. Therefore, in 5 additional experiments, we attempted to increase this probability by increasing the pacing stimulus amplitude. At a fixed coupling interval of 100 ms, we compared 1.0-, 2.0-, and 4.0-V pacing amplitudes (n=34, 32, and 33, respectively). These data are represented graphically in Figure 5A. The ability of the first pacing stimulus to capture increased sharply when the pacing amplitude was increased from 1.0 to 2.0 V (0.38±0.08 to 0.75±0.08, P<0.003). However, no incremental improvement in successful capture rate occurred with a subsequent increase in pacing amplitude to 4.0 V compared with 2.0 V (0.75±0.08 versus 0.64±0.08, P=0.2). The decrease in capture rate from 2.0 to 4.0 V was not statistically different. To further analyze this difference, we compared the types of NC in both groups. The 4.0-V pacing group had a larger rate of type II NC (9 of 12, versus 5 of 8 for 2.0 V). This difference presumably occurred by chance alone. When the rate of successful capture was compared after these events were removed (see Figure 5B), the successful capture rates were similar (0.89±0.06 at 2.0 V versus 0.88±0.07 for 4.0 V, P=NS). Furthermore, we compared the rate of type I NCs in the 3 groups. We felt this to be appropriate because the optimal pacing strategy would limit the number of type I NCs; type II NCs presumably could not be altered by the pacing stimuli. The rates of type I NCs for pacing amplitudes of 1.0, 2.0, and 4.0 V were 0.43±0.11, 0.38±0.17, and 0.25±0.13, respectively (Figure 5C).

**Outcome**

To determine whether pacing as an adjunct to defibrillation shocks could alter outcome, we compared the rate of successful defibrillation for the no-pacing and pacing groups (see Figure 6). The rate of successful defibrillation in the no-
after the shock. Note that the electrogram of the first PSA was different from the remainder of the recorded pacing electrograms. All subsequent pacing stimuli captured, and the result was successful defibrillation. An 8-ms isochrone map of the collision is shown in Figure 7B. In Figure 8, we demonstrate that a complex interplay can occur between the pacing-induced wave fronts and postshock wave fronts. Immediately after the shock, the first pacing stimulus captures and the resulting wave front collides with and results in the annihilation of the first PSA wave front. A second PSA wave propagated from the superior edge of the preparation to invade the pacing region before the second pacing stimulus. Hence, the second pacing stimulus was unable to capture. However, the third pacing stimulus and all subsequent pacing stimuli did capture.

**Discussion**

**Feasibility of Pacing Immediately After the Shock**

The most important result of this study is the demonstration that successful capture of the atria during pacing after a shock increases the effectiveness of atrial defibrillation. The video imaging approach used in this study allowed us to record the transmembrane potentials simultaneously from >20,000 sites from the epicardial surface of the right atrium during AF and defibrillation as well as during subsequent pacing. In contrast to multiple-electrode mapping, video imaging is immune to shock-induced signal distortion and is able to distinguish propagating fronts that are initiated by pacing stimuli from wave fronts that result from postshock activity. Indeed, our results show that it is possible to successfully capture atrial myocardium by pacing as early as 60 ms after a defibrillation shock.

The success rate of capture was dependent on the amplitude of the pacing stimulus and the coupling interval of the pacing stimulus to the shock. The optimal coupling interval appeared to be between 80 and 100 ms. At coupling intervals shorter than this window, postshock refractoriness limited the ability to capture. At longer coupling intervals, the encroachment of postshock activity into the vicinity of the pacing site limited the success rate. With 1.0- and 2.0-V stimuli, success rates were limited to <75%. Even though the pacing stimuli were increased to 4.0 V specifically at the optimized coupling interval, 100 ms, rates of successful capture remained unchanged. Therefore, these data suggest a limitation of rates of successful capture of <75% despite optimization of both coupling interval and pacing amplitude.

**Outcome**

We tested the hypothesis that successful capture of a significant portion of the atrium with a pacing train might alter the outcome of near-threshold shocks. During the course of investigation of the feasibility of successful capture after the shock, we compared the outcome of shocks in the no-pacing and pacing groups. Although overall, the success rate of
defibrillation by use of the pacing strategy was higher than the no-pacing strategy, this difference was not statistically significant (see Results section and Figure 6). However, from the investigation of feasibility of capture, only 75% of first paced stimuli captured, despite optimal conditions. Our optical technique allowed us to further divide the pacing group into those that successfully captured during the quiescent period and those that did not. When this analysis was performed, the success rate rose to 75%, which was statistically larger than the no-pacing group. It is important to note that despite pacing and successful capture with pacing stimuli at short coupling intervals after the shock, no paced wave front initiated reentry during propagation over the right atrial free wall.

**Possible Mechanisms for Alteration in Outcome: Entrainment**

Recently, it was shown that AF has a partially if not fully excitable gap by the demonstration that rapid pacing could locally entrain a portion of the atrium during AF. In an open-chest canine model of AF, pacing at cycle lengths slightly shorter or longer than the median AF interval and at pacing amplitudes 6 times diastolic threshold could repeatedly capture and thus entrain an area of approximately 4-cm diameter in the left atrium. Theoretically, regional entrainment of AF at a pacing cycle length slightly shorter than the median AF interval should result in progressively enlarging areas of entrainment and possible pacing-mediated termination of AF. However, in the above-mentioned studies, the area of entrainment was limited by the block of pacing-induced wave fronts as they propagated away from the pacing site or by collision of the pacing-induced wave fronts with fibrillation wave fronts. Regional control was lost by pacing either too slowly, in which case fibrillatory wave fronts invaded the pacing region, or too rapidly, in which case local reentry circuits were induced that reentered the pacing region before the next pacing stimulus. The window of cycle lengths during which entrainment occurred was approximately 16.5 ms. Pacing termination of AF did not occur in any of those experiments. Although never demonstrated, it is at least theoretically possible that AF could be pacing-terminated if a sufficiently large area of the atria was entrained by increasing either the number of pacing sites or the area of entrainment for each pacing site. Previous work in our laboratory has shown that failed defibrillation shocks are followed by a 110-ms quiescent period and by organized activity on the right atrial free wall. The results from this study suggest that a pacing train whose first pacing stimulus successfully captures during the quiescent period has the ability to alter outcome. The wave front induced by capture of the first pacing stimulus propagated in a homogeneous, broad front over the entire mapped surface. As such, the right atrial free wall was effectively “entrained” in an organized manner for the 2-second duration of the pacing train. It is therefore possible that the remaining atrial tissue was insufficient to allow the reinitiation of AF.

**Impact on Postshock Activity**

The source of the postshock activity remains unclear. There are 3 likely possibilities: (1) The shock induces reentrant activity in an area of critical potential gradient in a manner similar to the critical-point hypothesis for ventricular shocks; (2) the shock triggers focal activity from areas in the atria with pacemaker activity, either from the right atrium or elsewhere; and (3) the shock fails to completely terminate AF, and a remaining wavelet lingers at some distant point from the recording area. In the first and third cases, it is possible that the paced wave fronts propagate toward and collide with postshock activity, resulting in the mutual annihilation of both wave fronts. This phenomenon has been documented to occur at least over the right atrial free wall (see Figure 8). Continued pacing would potentially result in the progressive invasion of the source of activity and its possible termination either directly or by driving the source of rotating activity to a boundary. It has been demonstrated that externally induced wave fronts can collide with and terminate rotating sources of activity (spirals) in both isolated atrial and ventricular preparations. This can occur through collision and mutual annihilation of activity or by shifting the spiral core close to a boundary, which results in termination. Given the current limitations of our experimental design, it is impossible to confirm or disprove this hypothesis.

It is also possible that the source of the postshock activity is focal in origin from an automatic source induced by the shock. Studies of ventricular defibrillation have suggested that shocks may induce ectopic activity. In the atria, where
a greater abundance of “pacemaker”-type tissue resides, this mechanism may be even more tenable. If, indeed, the postshock activity is automatic in nature and induced by the shock, it is reasonable to believe that paced wave fronts could invade and suppress these sources. The present study does not address the nature of induction of postshock activity or its relation to failed defibrillation. However, regardless of the mechanism, it is theoretically possible that externally induced wave fronts have the potential to result in annihilation or suppression of this activity.

Advantages of Video Imaging
The video imaging technique used in these studies offered several unique advantages. First, video imaging allowed the recording of transmembrane signals during high-voltage defibrillation shocks because there was no contamination by electrical artifact. In addition, methoxyverapamil removed the mechanical contraction associated with shocks, such that recordings were not corrupted by motion artifact. Second, video imaging provides high spatial resolution with simultaneous recordings from 10,000 to 30,000 sites. This is 2 orders of magnitude greater than other cardiac mapping systems and allowed us to determine with a high degree of accuracy whether the wave fronts that appeared on the epicardium immediately after the shock were indeed from the pacing site or spontaneous activity from elsewhere. Moreover, the interaction of pacing-induced wave fronts with postshock activity could be studied with high spatial resolution.

Limitations of the Approach
The experiments in which optical recordings were used to study the feasibility of pacing were performed in the presence of ACh (10⁻⁶ mol/L) to facilitate the induction of AF. The absolute values of the coupling intervals for pacing after the shock and pacing amplitudes may not be applicable to experiments without ACh. It is likely that pacing after the shock without ACh is feasible; however, this will need documentation.

Our model is that of AF induced by burst pacing in the Langendorff-perfused isolated sheep heart after infusion of ACh. As such, ours is a model of acute AF in a normal heart. Hence, extrapolation of these data to the human condition in which atrial disease causes dilatation and/or patchy fibrosis is made with caution. We limited our pacing site to the optical field of view on the right atrial free wall to investigate the ability to capture. Hence, other pacing sites were not investigated. Other pacing sites, especially on the left atrium, may be more or less successful in altering outcome. The effects of pacing at other sites, especially those in the left atrium, after defibrillation shocks warrants further study, ideally studies in which the effects of pacing can be recorded simultaneously from both atria.

The optical recordings were made exclusively from the epicardial surface of the right atrium. There is considerable evidence that transmural propagation occurs during AF. It is unlikely that transmural propagation continued during the quiescent period for 95 ms without propagation to the epicardium. Furthermore, when epicardial activity did reappear after the shock, it did not occur as an epicardial breakthrough but rather from the edges of the field of view. However, video imaging was performed exclusively from the right atrial free wall. Therefore, the dynamics of wave propagation and interaction of paced wave fronts with other postshock wave fronts could only be studied in the field of view of the video camera. In an attempt to understand our results, we extrapolated our observations of the dynamics of wave propagation of paced stimuli and the interaction of pacing-induced wave fronts with postshock wave fronts to the entire surface of the atria. This obviously is less than ideal. Currently, however, no cardiac mapping systems exist that can record atrial defibrillation simultaneously from all surfaces of the atria with sufficient resolution and without shock artifact.

Clinical Implications
The impact of adjuvant approaches to atrial defibrillation shocks is currently under investigation. This study provides evidence that it is feasible to deliver a pacing train immediately after an atrial defibrillation shock with a reasonable expectation that the first pacing stimulus will capture. Furthermore, we have presented evidence that pacing during the quiescent period that follows atrial defibrillation shocks alters outcome of near-defibrillation threshold shocks. We introduce a new method by which pacing may be used in conjunction with defibrillation shocks. However, further studies are required to determine whether this “hybrid” therapy is a reasonable approach in conjunction with implanted atrial defibrillators to alter outcomes in humans.

Acknowledgments
This work was supported in part by Grant PO1-HL39707 from the National Heart, Lung, and Blood Institute, NIH and a grant from InControl Inc. We would like to thank Jiang Jiang, Megan Flanagan, Jianguo Chen, and Laverne Gilbert for their technical assistance.

References


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Circulation. 1998;98:64-72
doi: 10.1161/01.CIR.98.1.64

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