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Transmural and Endocardial Purkinje Activation in Pigs Preceding Local Myocardial Activation after Defibrillation Shocks

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Abstract

Background—Earliest recorded post-shock myocardial activations in pigs originate in the subepicardium of the apex and lateral free wall of the left ventricle (LV) 30–90 ms after the shock.

Objective—To determine if the Purkinje system is a candidate for the source of post-shock activations, endocardial and transmural post-shock activation mapping was performed.

Methods—In 5 pigs, 32 plunge needles with 12 electrodes (1mm spacing) were inserted into the LV apex and lateral free wall. Up to 70 plunge needles with 6 electrodes (2mm spacing) were spread throughout the remainder of the LV, while 9 to 12 plunge needles with 4 electrodes (2 mm spacing) were inserted into the RV. A basket catheter with 32 bipolar recording sites was inserted into the LV. DFT level shocks were delivered during 10 episodes of electrically induced ventricular fibrillation. Electrograms of post-shock activation cycles were analyzed for Purkinje and myocardial activations.

Results—Purkinje activations were recorded prior to local myocardial activation in 9% of basket electrograms and in 15% of plunge needles during the first post-shock activation cycle. Purkinje activations were identified during the first and subsequent several post-shock activation cycles in at least 1 basket and 1 needle electrogram in 96% and 98% of defibrillation episodes, respectively.

Conclusions—The Purkinje system is active during the early post-shock activation cycles following DFT level shocks. Further studies are required to determine if activation initiates in the

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Purkinje system or whether it is activated by the myocardium or by Purkinje-myocardial junctional cells.

Keywords

Purkinje activation; defibrillation; isoelectric window

INTRODUCTION

Sudden cardiac death is frequently caused by ventricular fibrillation (VF). Untreated VF leads to permanent neurological damage within minutes, followed shortly by death. Approximately 450,000 Americans die each year from sudden cardiac death.¹ The only effective treatment for VF is electrical defibrillation.

The specialized conduction system has been implicated as a source of idiopathic VF.^{2, 3} Studies have demonstrated that the Purkinje fiber system may be responsible for the onset of arrhythmias during post-infarct ischemia and reperfusion,⁴⁻⁶ and that congestive heart failure leads to changes in the Purkinje cells that may make them more prone to arrhythmogenesis.⁷ Studies have also suggested that the specialized conduction system may be important in the maintenance of VF.^{8, 9}

The Purkinje fiber system may be more sensitive than the working myocardium to the large field potential gradients created by defibrillation shocks. Isolated preparations of canine Purkinje fibers demonstrated a dramatic increase in automaticity after defibrillation shocks, while cardiomyocytes demonstrated an increased refractory period.¹⁰ The role of the specialized conduction system in the success or failure of defibrillation shocks is not known. The possibility exists that the specialized conduction system could be active during this period, but has not previously been reported.

After near defibrillation threshold (DFT) strength shocks there is a period of 30–90 ms, called the isoelectric window, during which no activity is recorded in the working myocardium.¹¹⁻¹⁵ Previous defibrillation studies have demonstrated that in pigs first post-shock activation tends to arise closer to the epicardium than the endocardium.^{11, 16} In pigs, Purkinje fibers traverse the ventricular wall and extend nearly to the epicardial surface.¹⁷⁻¹⁹ Thus, the subepicardial location of the first recorded post-shock electrical activation in pigs raises the possibility that the earliest post-shock activation arises in the specialized conduction system.

As a prerequisite for earliest post-shock activation initiating in the specialized conduction system, we tested the hypothesis that the specialized conduction system is active during the first several post-shock activation cycles following defibrillation shocks near the DFT in strength.

METHODS

Plunge needle design

Silver wire, fiberglass reinforced epoxy needles were constructed as described elsewhere²⁰. Three types of plunge needles were constructed: 1) 12 electrode needles with 1 mm spacing, 2) 6 electrode needles with 2 mm spacing, and 3) 4 point plunge needles with 2 mm spacing. Needles were tested and chloridized prior to each experiment.

Surgical Procedure

Five pigs (31.5±2.7 Kg) were initially anesthetized with an intramuscular injection of Telazol (4.4 mg/kg), xylazine (4.4 mg/kg), and atropine (0.04 mg/kg), followed by maintenance with isoflurane in 100% oxygen by inhalation. The animals were intubated and ventilated, and ventilation was adjusted to maintain normal pCO₂ levels.

Lead II surface EKG was monitored. A water-heated pad was used to maintain normal body temperature. Femoral arterial blood pressure, blood gases, pH, and electrolytes were monitored every 30 min and maintained within acceptable physiologic ranges. Throughout the experiment, normal saline was administered at a rate of 2–5 ml/kg/hr.

The chest was opened by median sternotomy, the heart was exposed, and the heart was suspended in a pericardial cradle. Defibrillation coils were inserted into the right ventricular (RV) apex and superior vena cava (SVC). The animals were heparinized with a loading dose of 300 units/kg, and maintained with a dose of 100 units/kg every hour thereafter. A 48 mm multielectrode basket (Constellation Catheter, Boston Scientific Corp., Natick, MA, USA) was introduced through a femoral artery into the left ventricle (LV). This catheter contained 8 splines with 8 electrodes spaced at 4 mm intervals along each spline. The basket was connected to a mapping system with 32 pairs of electrodes forming bipolar recording sites on the LV endocardial surface. Figure 1 shows a typical placement of the multielectrode basket and the defibrillation coils.

Since the first recorded activations in pigs tend to arise from the LV apex and lateral free wall in the subepicardial tissue for this electrode configuration,^{11, 16} needles were placed more densely in these regions than in the LV base and RV. Thirty-two 12 electrode plunge needles were inserted into the lateral LV free wall and apex with approximately 3–5 mm spacing between needles. Sixty to seventy 6 electrode plunge needles were inserted throughout the remainder of the LV with 5 – 10 mm spacing. Nine to twelve 4 electrode plunge needles were inserted evenly throughout the RV.

Mapping System Configuration

Two mapping systems were used to record activation in sinus rhythm and during the first five post-shock activation cycles after DFT level shocks. One mapping system recorded from 528 unipolar channels at 2 KHz to detect activation of the working myocardium. The signals were bandpass filtered in hardware between 0.5 Hz and 1 KHz. Unipolar electrograms were recorded from the 6 electrode and 4 electrode plunge needles, as well as from every other electrode of the 12 electrode plunge needles (or from 6 electrodes at 2 mm spacing) for recordings comparable to the 6 electrode plunge needle electrograms. A second mapping system recorded bipolar signals from the 3 most epicardial pairs of electrodes from the 12 electrode plunge needles (32 needles × 3 bipolar electrograms = 96 bipolar needle electrograms) and 32 bipolar basket electrodes at a sampling rate of 8 KHz with the signals bandpass filtered between 0.5 Hz and 4 KHz to record Purkinje as well as working myocardial activations.

Defibrillation Procedure

VF was induced with a DC shock applied to the RV. After 10 s of VF, a test shock was delivered. If the shock was not successful, a rescue shock was immediately delivered. A 3-reversal DFT search protocol, as described in a previous publication,²¹ was used to determine an initial DFT of a biphasic shock (6 ms first phase, 4 ms second phase; RV cathode, SVC anode for first phase). Briefly, leading edge voltage was successively bracketed by 80, 40, and 20 volts with the DFT being the level of the last successful shock.

Following insertion of the plunge needles, the electrodes were allowed to stabilize for at least 30 min and the DFT was again determined.

Ten times in each animal, a DFT strength shock was given 10 s after inducing VF. To assure that the shock strength was at or near the DFT level (shock strength at which 50% of the delivered shocks successfully defibrillate), the protocol required that 4, 5, or 6 of the 10 shocks be successful. If this requirement was not met, the DFT was determined again, and 10 VF episodes and shocks were repeated at the new DFT.

Localization of Needles

After completion of the study, each animal was euthanized by inducing extended VF. The heart was removed with the needles still inserted, and the heart was soaked in formalin overnight. Each plunge needle was removed, and color coded Teflon tubes were inserted into the needle locations. The needle locations were recorded with a 3D arm digitizer, and the locations of electrodes along each needle were determined, as previously described.^{11, 22}

Identification of Purkinje Activations

Electrograms from the multielectrode basket and bipolar plunge needles, during normal sinus rhythm and during the first post-shock activation cycle, were analyzed for evidence of Purkinje fiber activation using criteria similar to those used by other groups.^{2, 23, 24} Electrograms with Purkinje activations during the first post-shock activation cycle were then analyzed for Purkinje activation during post-shock activation cycles 2–5. Recorded potentials and their temporal derivatives (5-point, least squares) were examined to identify Purkinje activations. Purkinje fiber activation was identified as an initial sharp complex (1–2 ms in duration) preceding the larger and slower ventricular activation by <15 ms. Purkinje potentials were identified twice during normal sinus rhythm, once before the first mapped defibrillation episode, and once after the last defibrillation shock. The number of Purkinje spikes identified during these two normal sinus rhythm episodes was averaged. Numbers of Purkinje activations identified in the 32 bipolar electrograms from the multielectrode basket and on at least one bipolar electrogram from each of the 32 plunge needles with bipolar recording sites were determined.

Statistical Analysis

Testing for statistical significance was performed with a two-tailed, two sampled, paired Student's t-test for paired data (DFT level comparison, first post-shock activation times between needles and basket electrograms), and two-tailed, two sampled unequal variance Student's t-tests for all other data. Statistical significance was determined if $p < 0.05$. The data are expressed as the mean $\pm$ standard deviation unless otherwise stated.

RESULTS

The DFT before insertion of the plunge needles was 576 ± 155 V, while the DFT after insertion of the needles was 476 ± 41 V ($p = \text{NS}$). Of the 50 defibrillation shocks and subsequent activation sequences, 26 were failures. Of the 24 successful shocks, 22 were Type B successes, which are defined as successful defibrillation shocks with the earliest first post-shock activation recorded in the working myocardium within 130 ms of the defibrillation shock.¹⁵ Type A successes, which are defined as successful defibrillation shocks after which this interval is >130 ms, occurred twice and had an average earliest recorded post-shock activation time of 245 ms. The first five post-shock activation cycles in these two episodes were all sinus beats. None of the failed shocks were followed by any normal sinus cycles.

In all of the 48 Type B successes and failed shocks, first local myocardial post-shock activation was recorded with the plunge needles before it was recorded with the basket. First transmural myocardial post-shock activation was recorded with an LV electrode in all 48 episodes and with one of the three most epicardial unipolar electrograms in 98% (47/48) of these episodes. First post-shock local myocardial activation arose in the LV apex and lateral free wall¹¹, 16 in 88% of the instances, which is the region from which transmural bipolar electrograms were recorded.

Purkinje activations were recorded during the first post-shock activation cycle by at least one needle electrogram in 98% (47/48) of the episodes and in at least one basket electrogram in 96% (46/48) of the shock failures or Type B successes. The distance from the earliest electrode recording myocardial activation to the nearest electrode recording a Purkinje activation was 1.4 ± 0.7 cm, which is significantly closer ($p < 0.01$) than the average distance from the earliest site to all bipolar plunge needle recording sites which was 2.0 ± 1.2 cm. Purkinje spikes were identified from the same electrodes from which the earliest recorded post-shock transmural myocardial activation was recorded in 2 episodes, one of which was a shock failure and one of which was a Type B success. The earliest post-shock myocardial activation recorded on the basket was preceded by earlier Purkinje activation 8 times: once in a Type A success, 4 times in a Type B success, and 3 times in shock failures. Figure 2 shows an example of a basket recording that contained a Purkinje spike that preceded all myocardial activations recorded by the basket during the first post-shock cycle.

Purkinje activations were identified in normal sinus rhythm preceding local myocardial activation during the first post-shock activation cycle in at least one recording from 21% (6.8 ± 2.9) of the 32 plunge needles with bipolar recording sites and in 26% (8.3 ± 4.6) of the 32 basket electrograms. Purkinje activations were identified during the first post-shock activation cycle (for Type B successes and failures) in 9% (4.7 ± 2.5) of the needles and in 9% (2.9 ± 1.9) of the basket electrograms, which was less often than they were identified in normal sinus rhythm ($p < 0.05$). Of the needles and basket electrodes with identified Purkinje activations after shocks, 71% of the needles and 82% of the basket electrodes had Purkinje spikes identified in normal sinus rhythm as well as during the first post-shock activation cycle. Figures 3 and 4 show examples of plunge needle and basket electrograms with Purkinje activations in normal sinus rhythm and preceding local myocardial activation during the first five post-shock activation cycles after a successful Type B and a failed defibrillation shock. Table 1 indicates how often Purkinje activations were identified during the first post-shock activation cycle in plunge needle and basket electrograms during Type B successful shocks and failed shocks. Times for isoelectric windows for Type B successful shocks and failed shocks are also given in Table 1.

Basket electrodes and bipolar needle electrodes with identified Purkinje activations in the first post-shock activation cycle were analyzed during the second through fifth post-shock activation cycles for Purkinje activations. In the majority of the cases, when Purkinje activations were identified preceding cycle 1 on an electrode, Purkinje activations were identified during cycles 2–5. Table 2 shows the incidence of identified Purkinje activations during each cycle for the electrodes with identified Purkinje activations in cycle 1. Figure 5 shows how many ectopic and sinus cycles followed successful and failed shocks. While most of the first cycles after successful shocks were ectopic (in 22 of 24 recordings), by the 5th cycle, all of the activations after successful shocks were sinus beats.

First myocardial activation was recorded approximately 25 ms earlier on average in the plunge needle electrograms than with the basket electrograms. First activation initiated near the epicardium in almost every case, and then spread transmurally and laterally to the

surrounding tissue. Examples of the location of the earliest post-shock activation and Purkinje activations are shown for a Type B Success and a failed shock in Figure 6.

DISCUSSION

Activation of the Purkinje fiber system during the first several post-shock activation cycles in almost every episode demonstrates that the specialized conduction system is active shortly after near DFT level shocks. While Purkinje activation preceded first recorded myocardial activation in 8 basket electrograms, these activations did not precede the first recorded transmural myocardial activations. In two episodes, transmural myocardial activation was preceded by Purkinje activations. However, our spatial resolution did not permit us to determine precisely the location of the first myocardial activation. Purkinje activation at sites away from the site of origin could have initiated in the Purkinje system or the myocardium. If the Purkinje system was activated retrogradely from the myocardium, the faster conduction velocity in the Purkinje system could still permit the Purkinje spike to precede the local myocardial activation at a distance from the site of origin. The lack of Purkinje activations identified near the site of earliest recorded post-shock myocardial activation may be because first activation originates in the myocardium and spreads to surrounding tissue before activating the Purkinje system. On the other hand, Purkinje activation could have occurred near the sites of first post-shock myocardial activation but not have been identified due to the lack of temporal separation in the Purkinje and myocardial activations. This could have happened if earliest post-shock activation occurred in Purkinje tissue near the Purkinje-myocardial junction (PMJ), from which it would have quickly spread into the working myocardium so that the Purkinje and myocardial activation complexes would have been superimposed.

The majority of electrodes from which Purkinje activations were recorded during cycle 1 after near threshold shocks also had identified Purkinje activations in normal sinus rhythm. Also, the majority of the electrodes that had identified Purkinje activations during post-shock activation cycle 1 continued to record identifiable Purkinje activations in cycles 2–5. The consistency of electrode sites with identifiable Purkinje activity suggests that these electrodes were likely in close proximity to Purkinje bundles. This finding also suggests that either 1) the Purkinje system was the site of earliest post-shock activation which then spread to activate the working myocardium, or 2) these Purkinje sites were sufficiently far from the PVJ sites for the Purkinje activation to arrive at the electrode location before the local myocardial activation wavefront due to the faster conduction velocity in the specialized conduction system.

Additional recording sites to further map the Purkinje system may aid in determining the initial site of activation. This is the first reported direct recording of the Purkinje system immediately following defibrillation shocks in an intact heart model. One difficulty in increasing the number of channels for mapping Purkinje activity is the tradeoff between sampling rate and channels available with any mapping system. While some studies have reported identification of Purkinje activation with plunge needles using sampling rates of 1 to 3 KHz,^{4-6, 25, 26} other studies have indicated that sampling rates of 15–20 KHz are required to accurately record Purkinje fiber activation morphology.^{27, 28} Therefore, in this study a compromise of 8 KHz was used.

Direct recording of transmural Purkinje activation in pigs has not previously been reported. This may be due to the difficulty in recording from the small Purkinje bundles near the epicardium. Large Purkinje fiber bundles can be visualized on the endocardium of the pig, but these fibers branch into smaller bundles as they approach the epicardium, branching to

1- or 2-cell columns before connecting to even smaller PMJ cells which activate the cardiomyocytes.^{19, 29, 30}

The small bundles of Purkinje fibers in the wall may produce relatively small signals as compared to the local myocardial action potentials.

Activation mapping of the Purkinje system after defibrillation shocks in an intact heart provides an opportunity to study the role the specialized conduction system plays in defibrillation. However, effective transmural Purkinje mapping of large areas is difficult. Isolated papillary muscle studies have shown that Purkinje fibers are more sensitive to large shocks than is the working myocardium, and that they fire rapidly after exposure to a large shock.¹⁰ This rapid firing may lead to multiple wavefronts on the heart at one time, which may degenerate back into VF.¹¹ While mapping studies have demonstrated that first myocardial activation emerges from the low field gradient region after near DFT shocks,^{31, 32} activation could originate in the conduction system in high gradient areas from rapidly firing Purkinje fibers and then spread to the low gradient areas. The rapid activation rate of the Purkinje fibers in the high gradient regions of the heart may be spreading through the specialized conduction system, but may not spread through the myocardial tissue because it is still refractory from the defibrillation shock. The earliest post-shock myocardial activation may emerge from these low gradient areas because they return to an excitable state before myocardium in the high gradient regions,^{33, 34} and are excited by activity in the specialized conduction system. Purkinje fiber activation during the interval between the shock and earliest post-shock myocardial activation is a prerequisite for the Purkinje fiber system causing the earliest post-shock activation.²⁹

An additional possible source for the first activation, which is consistent with our findings, could be the specialized PMJ cells between the Purkinje system and the myocardial cells. These cells have been implicated in arrhythmogenesis under certain conditions.³⁵ Initiation of activation in PMJ cells would lead to simultaneous activation of the myocardium and the Purkinje system. Purkinje fiber activations at these sites would be difficult to distinguish since the Purkinje potential would likely be lost in the larger myocardial action potential. PMJ initiation of first activation would be consistent with the lack of Purkinje fiber activation at the sites of earliest post-shock myocardial activation.

Secondary sources created by inhomogeneities in cardiac tissue have been shown play an important role in defibrillation.^{36, 37} The magnitude of the secondary sources created by an obstacle that interrupts the intracellular space depends upon the size of the obstacle and its distance from other obstacles.^{38, 39} The smaller is the obstacle, the smaller are the magnitudes of the secondary sources adjacent to it. Thus, the large, broad connective tissue septae throughout the myocardium would be expected to create much larger secondary sources than the much smaller obstacles created by the plunge needles. Therefore, even though the plunge needles did not create secondary sources large enough to significantly alter the DFT, the connective tissue septa may have been large enough to create secondary sources of sufficient magnitude to contribute to defibrillation. While the connective tissue septae are separated by approximately 80 microns,⁴⁰ the Purkinje fibers at their narrowest point where they are still insulated from the working myocardium near the Purkinje-myocardial junctions are only approximately 25 microns wide.²⁹ Therefore, for a particular shock electric field across these narrowest dimensions, the secondary sources created in the working myocardium by the connective tissue septae should have been larger than the secondary sources created within the insulated Purkinje fibers near their terminations. Thus, shocks near the DFT in strength may create secondary sources large enough to defibrillate the working myocardium but not create secondary sources large enough to defibrillate the Purkinje fibers so that activation appears there first after the shock.

Study Limitations

While the Purkinje fibers in pigs traverse the ventricular wall and extend nearly to the epicardial surface,^{17-19, 29} in humans the Purkinje fiber system is limited primarily to the endocardial surface^{41, 42}. Therefore, transmural Purkinje activation would not be expected in humans. While the anatomical location of PMJ cells may differ between pigs and humans, the involvement of the specialized conduction system in the initiation of activation immediately following near threshold defibrillation shocks may still be similar between the species. If so, earliest Purkinje activation would be expected to arise near the endocardium in human.

Purkinje potentials were identified preceding local myocardial activation in normal sinus rhythm and immediately after defibrillation shocks. Purkinje fiber activations were identified consistently during the diastolic interval or while there was little other electrical activity. However, identification of Purkinje fiber activations occurring at the same time as local myocardial activation is much more difficult. The reduction in identifiable Purkinje spikes after defibrillation may be due to Purkinje activations that occurred coincidentally with local myocardial activations, rather than preceding local myocardial activation, as would be expected in normal sinus rhythm. Due to the method used to identify Purkinje activations, Purkinje potentials occurring after the start of the myocardial activation could not be identified. Since the sources for the Purkinje potentials are relatively small signals, these potentials are lost in the much larger myocardial activation. While Purkinje activation precedes myocardial activation in normal sinus rhythm and at least some of the time before myocardial activation after near DFT strength shocks, it is possible that some Purkinje activations after shocks occur at the same time as or after myocardial activations.

Although the possibility for secondary sources to be created around the plunge needles exists,^{39, 43} the change in DFT before and after insertion of the needles failed to reach statistical significance and the macroscopic post-shock activation patterns and isoelectric windows seem to be unaffected by insertion of the plunge needles. Similar results with regard to the isoelectric window and first activation location and patterns have been reported with plunge needles,¹¹ epicardial electrical mapping,^{14, 15, 44} and optical mapping.^{12, 13} A study conducted in pigs with fiberglass plunge needles and an epicardial plaque demonstrated that a row of closely spaced fiberglass plunge needles did not substantially disrupt short duration fibrillation patterns.²⁰ A study in dogs showed that there were no acute or chronic changes in the slope of electrogram recordings, activation time, hemodynamic function, or an increase in occurrence of tachycardia when 66 plunge needles were inserted.⁴⁵

CONCLUSION

Plunge needle electrograms with identified transmural Purkinje activation and multielectrode basket electrograms with endocardial Purkinje activation demonstrate that the specialized conduction system is active during the first several post-shock cycles following near DFT level shocks. Whether activation initiates in sub-epicardial cardiomyocytes, transitional cells, or the Purkinje system, and then spreads to surrounding tissue has yet to be determined.

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Glossary of Abbreviations (Each defined at first use.)

DFT	(Defibrillation Threshold)
LV	(Left Ventricle)
(PMJ)	Purkinje-myocardial junctions
RV	(Right Ventricle)
VF	(Ventricular Fibrillation)

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Figure 1.
Typical placement of the RV and SVC shocking coils and the multielectrode basket in the LV shown in an anterior-posterior fluoroscopic image.

Figure 2. Four bipolar traces from a single spline of a multielectrode basket during the first post-shock activation cycle. Voltage electrograms (left) and the first temporal derivative of the voltage electrograms (right) are shown. Purkinje activation (arrows) was identified prior to earliest recorded myocardial activation (*) recorded by any electrode on the basket.

Figure 3. Plunge needle electrograms and temporal derivatives from a single bipolar electrode. The top trace in each panel is a voltage recording with the temporal derivatives of the numbered activations shown below. Purkinje activations are indicated with arrows. Electrograms from normal sinus rhythm (A), after a Type B successful shock (B), and after a failed shock (C) are shown. In this example, Purkinje activations were identified in each sinus cycle, preceding each of the first 5 activations after a successful shock, and preceding activations 1, 2, and 5 after a failed defibrillation shock.

Figure 4. Multielectrode basket electrograms and temporal derivatives from a single bipolar electrode during the same episodes as shown in Figure 3. See Figure 3 for a description of each panel. Purkinje activations were identified in the same activations as shown in Figure 3.

Figure 5.

Post-shock cycle characteristics and number of electrodes with Purkinje activations. Each post-shock activation cycle was categorized as an ectopic cycle after a successful shock, an ectopic cycle after a failed shock, or a sinus cycle after a successful shock (A). The number of electrodes that recorded Purkinje activations during the first post-shock cycle was quantified, as well as the number of those same electrodes that recorded Purkinje activations in cycles 2–5 for both plunge needle recordings (B) and basket recordings (C).

Figure 6. Location of earliest post-shock myocardial and Purkinje activations for a Type B success (A) and a failure (B). Plunge needles with unipolar recording sites (black circles) were distributed through the LV and RV, while combined unipolar and bipolar recording sites (all other colored markers) covered the LV apex and lateral free wall. The locations of the earliest post-shock myocardial activation (green squares designated by the number 1 and with accompanying temporal derivative of the electrograms on the right) and all identified Purkinje activations (red diamonds designated by numbers 2–8 (A) and 2–5 (B) with accompanying electrograms on right) are shown for a Type B successful shock (A) and a failed shock (B). Arrows denote identified Purkinje activations in the temporal derivatives of electrograms on the right.

Table 1

of Purkinje Activation and Earliest Recorded Activation Times

	Bipolar Plunge Needles			Basket Electrodes		
	Type B Successes	Failures	Total	Type B Successes	Failures	Total
Identified in cycle *	17% (5.3±2.5)	13% (4.1±2.4)	15% (4.7±2.5)	10% (3.3±2.0)	8% (2.5±1.7)	9% (2.9±1.9)
Window (ms)	56.5±22.0	43.3±8.4	49.4±17.3	80.1±17.8	70.0±14.6	74.8±16.6

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plunge needles or of 32 basket sites. Brackets indicate p<0.05 between compared values.

Table 2

Incidence of Purkinje Activations During Postshock Cycles in Electrograms with Purkinje Activations in the First Post-shock Cycle

Post-shock activation cycle	2	3	4	5
Plunge needle electrodes with Purkinje activations*	71%	60%	60%	59%
Basket electrodes with Purkinje activations†	70%	57%	51%	53%

Purkinje activations identified in the first post-shock activation cycle in 277 plunge needle electrograms(*) and 146 basket electrograms(†)