Guidelines for Plunge Needle Recording for Effective Detection of Purkinje Activation

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Abstract-Direct recording of Purkinje fiber activity may lead to a better understanding of the role of the specialized conduction system in pathological cardiac conditions. Two studies were conducted in pigs to determine guidelines for effective plunge needle recording techniques. In the first experiment, Purkinje fiber activations were recorded at 16 KHz with 3 bipolar electrodes (2 mm spacing) on epoxy plunge needles, and were later lowpass filtered and downsampled to determine the rate required for effective identification of Purkinje activation. Purkinje spikes were identifiable at sampling rates of 4 KHz and greater, but were not easily distinguished at sampling rates of 2 KHz or less. In the second experiment, 4 plunge needles with 15 electrodes (1 mm spacing) were inserted 8 times into different locations around the left ventricle. Unipolar (15 per needle) and bipolar (14 per needle) signals were recorded simultaneously at a sampling rate of 8 KHz. Purkinje activations were identified in 13/32 plunge needle sites. Of the 13 sites with identified Purkinje activations, 10 were within 2 mm of the endocardium. Bipolar recordings demonstrated Purkinje potentials that were 13% of the amplitude of the following myocardial activation, while unipolar recordings from the same electrodes recorded Purkinje potentials that were only 5% of the amplitude of the following myocardial activations. Three guidelines were developed for effective Purkinje fiber recording: 1) use a minimum sampling rate of 4 KHz., 2) record near the endocardium, and 3) use bipolar rather than unipolar recording electrodes.

Keywords—electrophysiology, cardiac arrhythmias, Purkinje fiber activation, plunge needle recording

I. INTRODUCTION

Sudden cardiac death is typically caused by the onset of ventricular fibrillation (VF), which significantly reduces

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R. E. Ideker has appointments within the Departments of Medicine, Physiology, and Biomedical Engineering at the University of Alabama at Birmingham, 1670 University Ave, Volker Hall B140, Birmingham, AL 35294 (email: rei@crml.uab.edu). cardiac output, leading to hemodynamic collapse. If VF is not treated by electrical defibrillation, brain damage and other tissue damage occur within minutes. Approximately 450,000 Americans die each year from sudden cardiac death[1]. The role of the specialized conduction system in sudden cardiac death and VF has not been thoroughly explored, in part due to difficulty in effectively recording the activity of the Purkinje fiber system.

Plunge needle studies have demonstrated that the Purkinje fiber system may be responsible for the onset of arrhythmias during post-infarct ischemia and reperfusion[2-4]. Studies have also suggested that the specialized conduction system may be important in the maintenance of VF[5, 6].

While some studies have reported identification of Purkinje activation with plunge needles using sampling rates of 1 to 3 KHz[2-4, 7, 8], other studies have indicated that sampling rates of 15-20 KHz are required to accurately record Purkinje fiber activation morphology[9, 10]. Although higher sampling rates record Purkinje fiber activation morphology more accurately, with most recording systems there is a tradeoff between sampling rate and the number of channels available for recording. Studies requiring a large number of channels ideally would use a lower sampling rate that allows for consistent identification of Purkinje activations.

The purpose of these initial studies was to develop effective techniques for recording Purkinje activation to use in future studies that further define the role of the specialized conduction system in arrhythmia onset, maintenance, and treatment.

II. METHODOLOGY

A. Plunge needle design

Silver wire fiberglass reinforced epoxy needles were constructed as previously described[11]. Two types of plunge needles were designed. For the first experiment, 11 plunge needles with 6 electrodes positioned at 2 mm intervals were constructed. For the second experiment, 4 needles were constructed with 15 electrodes spaced at 1 mm intervals with a 5 mm gap at the proximal end of the needle where no electrodes were located. Figure 1 shows the configuration of the plunge needles used in these two studies. Since large bundles of Purkinje fibers have been shown to traverse the endocardial surface, the plunge needles were designed so electrodes would span the

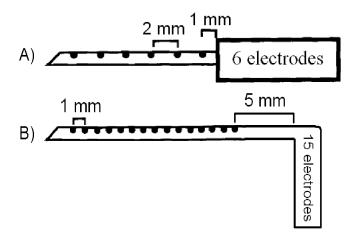


Figure 1 Plunge needles used in two studies. Needles from the first experiment are shown in A. Needles used in the second experiment are shown in B.

endocardium/blood chamber interface. Needles were chloridized prior to the experiments.

B. Surgical Procedure

Two pigs (35 kg each) were initially anestitized with an intramuscular injection of Telazol (4.4 mg/kg), xylazine (2.2 mg/kg), and atropine (0.04 mg/kg), followed by maintenance with isoflurane in 100% oxygen by inhalation. Intubation was accomplished with a cuffed endotracheal tube, with mechanical ventilation (volume controlled, tidal volume = 12 ml/kg) at a rate of 10-15 cycles/min with the animal in a restrained, dorsally recumbent position.

Lead II surface EKG was monitored. A water-heated pad was used to maintain body temperature at 37 ± 1 °C. Femoral arterial blood pressure, blood gases, pH, and electrolytes were monitored every 30 minutes 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 and the heart was exposed. The heart was suspended in a pericardial cradle.

In the first experiment, 11 plunge needles with 6 electrodes were inserted into the lateral and anterior portions of left ventricular (LV) free wall. In the second animal, 4 needles with 15 electrodes were inserted in the anterior, lateral, or posterior surfaces of the LV freewall. Sinus rhythm was recorded, and the needles were removed and reinserted in a new location. This process was repeated a total of 8 times in different locations on the LV. For both experiments, the electrodes were allowed to settle for 20 minutes following insertion to permit the injury potentials to subside before continuing with recording.

C. Mapping system configuration

For experiment 1, 33 channels were recorded from 3 bipolar electrode pairs on each of 11 needles at a sampling rate of 16 KHz. The system highpass filter was set at 0.05Hz, and the lowpass filter was set at 4 KHz.

For experiment 2, 60 channels were configured to record unipolar signals from 4 plunge needles at a sampling rate of 8 KHz. An additional 56 channels recorded bipolar signals from the same electrodes (14 bipoles were recorded from the 15 electrodes on each needle) at 8 KHz. The system highpass and lowpass filters were set at 0.5 Hz and 4 KHz, respectively.

D. Data Analysis Techniques

Data from both experiments were analyzed for evidence of Purkinje fiber activation using previously published criteria of other groups[12-14]. In brief, Purkinje fiber activation was identified as an initial sharp potential (<10 ms in duration) preceding the larger and slower ventricular electrogram by <15 ms during sinus rhythm. To be considered Purkinje activations, these potentials were required to be present and qualitatively unchanged in timing or amplitude for at least three consecutive beats. Recorded potentials and their temporal derivatives were examined to identify the Purkinje activations. In the second experiment, both the unipolar and the bipolar signals were analyzed to verify the presence of Purkinje activation on a needle.

The recorded signals from the first experiment were analyzed in MatlabTM. The 16 KHz sampled data was lowpass filtered at the Nyquist rate and downsampled in MatlabTM to 8, 4, 2, and 1 KHz sampling rates.

In the second experiment, the unipolar and bipolar recordings were both examined for evidence of Purkinje fiber activation. Peak-to-peak amplitude of the myocardial activations and Purkinje activations was measured for the channels that recorded Purkinje activations. Statistical significance was determined with a paired two-tailed student t-test.

Electrodes were assumed to be in the LV blood chamber, rather than in the myocardium when the bipolar peak-topeak activation electrograms were <2 mV. Typically, the endocardial-most points of the needles displayed markedly reduced signal sizes as compared with the epicardial-most recordings, with an abrupt transition in amplitude in the vicinity of the endocardial/blood chamber interface. Since the signal amplitudes were reduced and the noise levels remained approximately the same, the autoscaled amplitude and temporal derivative signals had increased signal to noise ratios. The signals in the tissue therefore appeared cleaner than the signals in the blood cavity.

III. RESULTS

In the first experiment, Purkinje fiber activations were identified in two of the plunge needle recordings. Purkinje fibers could be identified consistently in recordings sampled at 16 KHz and downsampled to 8 and 4 KHz, but not consistently for signals downsampled to 2 and 1 KHz. Figure 2 shows an example from the first experiment that was recorded at 16 KHz and subsequently downsampled to 8, 4, 2, and 1 KHz.

In the second experiment, both unipolar and bipolar recordings were analyzed from the same plunge needles, and, in each case, Purkinje activations could be identified in at least one of the unipolar recordings that corresponded to a

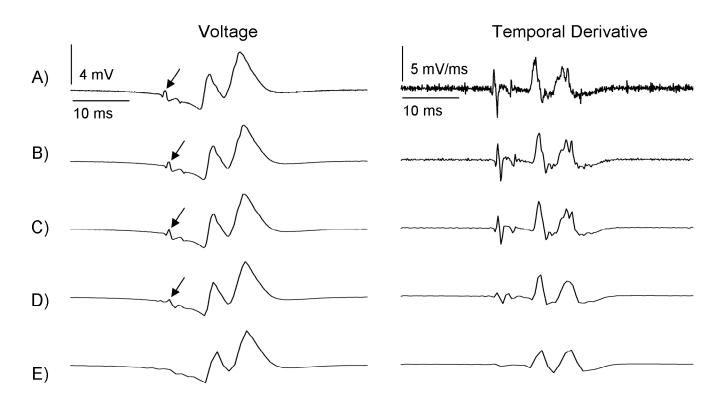


Figure 2 Bipolar recording of a Purkinje activation (shown with arrows) preceding local myocardial activation. The traces on the left are voltage, while the traces on the right are the temporal derivative of the voltage signals. A) shows an activation cycle as recorded at 16 KHz. B) through E) show the same signal after lowpass filtering below the Nyquist criterion and downsampling the signal to: B) 8 KHz, C) 4 KHz, D) 2 KHz, and E) 1 KHz. The Purkinje activation is impossible to identify at the lowest sampling rates.

Purkinje activation identified in the bipolar recordings. Purkinje fiber activations were identified in 41% (13 of 32) of plunge needles. The largest Purkinje fiber signals on these 13 needles were recorded 11.9 ± 2.9 mm (mean \pm standard deviation) from the epicardial surface. The needles protruded into the blood cavity in 11 of the 13 needles that had Purkinje spikes. The needles entered the blood cavity an average of 12.9 ± 2.6 mm from the epicardial surface. In 91% (10/ 11) of the needles with Purkinje activations that entered the blood cavity, the largest Purkinje spikes were recorded within 2 mm of the endocardial-blood interface. In a single needle, the largest Purkinje activation occurred 10 mm from the epicardial surface, while the needle entered the blood cavity 17 mm from the epicardial surface.

Measured peak-to-peak unipolar and bipolar Purkinje spikes and measured peak-to-peak myocardial activations (for the myocardial activation immediately following the Purkinje activation) as well as the ratios between the Purkinje and myocardial activations are shown in Table 1.

Table 1 Unipolar and Bipolar Amplitude Comparisons		
	Bipolar	Unipolar
	Recordings	Recordings
Purkinje Amplitude (mV)	0.44 ± 0.27	$0.54{\pm}0.28$
Myocardial Amplitude (mV)	4.88±3.26*	10.8±2.45 *
Average Purkinje/Myocardial amplitude ratio	0.13*	0.05*

*p<0.05 for bipolar compared to unipolar with paired t-test

VI. DISCUSSION

The first experiment demonstrated the importance of sampling plunge needle data at sufficiently high sampling rates in order to successfully identify Purkinje activations. In previous experiments, we were unable to explain why Purkinje spikes were only sporadically recorded when data was sampled at 2 KHz. The Purkinje activations tended to appear and disappear from beat to beat, even when the electrode location was stable. Thus, a sampling rate of at least 4 KHz is recommended to consistently record Purkinje activations.

In the second experiment, identification of Purkinje fibers in 41% of the needles is similar to reported results of others in dogs[2, 4, 15]. Studies have demonstrated that the Purkinje system in pigs permeates transmurally from the endocardium to extend nearly to the epicardium[16, 17]. The largest bundles are found near the endocardium, which is consistent with our finding that in 10 of the 13 plunge needles with identified Purkinje activations, the largest Purkinje potentials were within 2 mm of the endocardium. The three needles with Purkinje spikes at a distance of >2mm from the endocardium were presumably adjacent to smaller Purkinje bundles in the myocardium. The Purkinje fiber distribution may also explain why we were only able to identify Purkinje potentials in 2 of 11 needles in the myocardial tissue in the first experiment.

In general, Purkinje fiber potentials were easier to identify with bipolar rather than unipolar recordings. While the average peak-to-peak amplitudes of the recorded Purkinje potentials were not significantly different, the myocardial activations recorded by unipolar electrodes were significantly larger than those recorded by bipolar electrodes. The Purkinje potentials were on average 13% of the amplitude of the following myocardial action potential for bipolar recordings, versus 5% of the amplitude of the unipolar myocardial action potential. The increased ratio of Purkinje to myocardial amplitude for bipolar versus unipolar recordings facilitated the identification of these potentials.

To be able to use the rules established by other researchers for identifying Purkinje potentials, Purkinje potentials were identified in sinus rhythm. Purkinje fiber activations could be identified during the diastolic interval while there is little other electrical activity. However, identification of Purkinje fiber activations during VF will be essential to establish the role of the conduction system in VF. We recommend that sinus rhythm be used to identify plunge needles that are in close proximity to Purkinje fibers, and that these needles then be examined in VF for evidence of Purkinje activation. While the same rules of timing of the Purkinje potentials to local myocardial activation may not apply during VF, the guidelines of sampling rate, endocardial recording sites, and bipolar recording techniques should be useful for identifying Purkinje spikes in VF as well as sinus rhythm.

V. CONCLUSIONS

Recording Purkinje fiber activations directly may lead to greater understanding of the role of the specialized conduction system in arrhythmogenesis and the maintenance of VF. To facilitate direct recording and identification of Purkinje fiber activation, the following recommendations are offered: 1) a sampling rate of at least 4 KHz should be used, 2) plunge needle electrodes that are near the endocardium are more likely to record Purkinje activity, and 3) bipolar recording provides larger Purkinje to myocardial activation amplitude ratios. Future experiments following these guidelines may lead to a greater understanding of the specialized conduction system in sudden cardiac death.

References

- M. H. Lehmann and S. Saksena, "Implantable cardioverter defibrillators in cardiovascular practice: report of the Policy Conference of the North American Society of Pacing and Electrophysiology. NASPE Policy Conference Committee," *Pacing Clin Electrophysiol*, vol. 14, pp. 969-79, 1991.
- [2] D. O. Arnar, J. R. Bullinga, and J. B. Martins, "Role of the Purkinje system in spontaneous ventricular tachycardia during acute ischemia in a canine model," *CIRC*, vol. 96, pp. 2421-9, 1997.
- [3] D. Xing and J. B. Martins, "Triggered activity due to delayed afterdepolarizations in sites of focal origin of ischemic ventricular tachycardia," *Am J Physiol Heart Circ Physiol*, vol. 287, pp. H2078-84, 2004.
- [4] D. O. Arnar and J. B. Martins, "Purkinje involvement in arrhythmias after coronary artery reperfusion," *Am J Physiol Heart Circ Physiol*, vol. 282, pp. H1189-H1196., 2002.
- [5] S. J. Worley, J. L. Swain, P. G. Colavita, W. M. Smith, and R. E. Ideker, "Development of an endocardial-epicardial gradient of activation rate during electrically induced, sustained ventricular fibrillation in the dog," *AJC*, vol. 55, pp. 813-820, 1985.
- [6] Y.-M. Cha, T. Uchida, P. L. Wolf, B. B. Peters, M. C. Fishbein, H. S. Karagueuzian, and P.-S. Chen, "Effects of chemical subendocardial ablation on activation rate gradient during ventricular fibrillation," *AJP*, vol. 269 (Heart Circ. Physiol.), pp. H1998-H2009, 1995.
- [7] D. J. Wendt and J. B. Martins, "Autonomic neural regulation of intact Purkinje system of dogs," *Am J Physiol*, vol. 258, pp. H1420-6, 1990.
- [8] S. A. Ben-Haim, D. G. Cable, T. E. Rath, L. Carmen, and J. B. Martins, "Impulse propagation in the Purkinje system and myocardium of intact dogs," *Am J Physiol*, vol. 265, pp. H1588-95, 1993.
- [9] R. C. Barr and M. S. Spach, "Sampling rates required for digital recording of intracellular and extracellular cardiac potentials," *CIRC*, vol. 55, pp. 40-48, 1977.
- [10] A. W. Cates, W. M. SMith, R. E. Ideker, and A. E. Pollard, "Purkinje and ventricular contributions to endocardial activation sequence in perfused rabbit right ventricle," *Am J Physiol Heart Circ Physiol*, vol. 281, pp. H490-H505, 2001.
- [11] J. M. Rogers, S. B. Melnick, and J. Huang, "Fiberglass needle electrodes for transmural cardiac mapping," *IEEE Trans Biomed Eng*, vol. 49, pp. 1639-41, 2002.
- [12] M. Haïssaguerre, M. Shoda, P. Jaïs, A. Nogami, D. C. Shah, J. Kautzner, T. Arentz, D. Kalushe, D. Lamaison, M. Griffith, F. Cruz, A. de Paola, F. Gaïta, M. Hocini, S. Garrigue, L. Macle, R. Weerasooriya, and J. Clémenty, "Mapping and ablation of idiopathic ventricular fibrillation," *Circulation*, vol. 106, pp. 962-967., 2002.
- [13] M. Haissaguerre, D. C. Shah, P. Jais, M. Shoda, J. Kautzner, T. Arentz, D. Kalushe, A. Kadish, M. Griffith, F. Gaita, T. Yamane, S. Garrigue, M. Hocini, and J. Clementy, "Role of Purkinje conducting system in triggering of idiopathic ventricular fibrillation," *Lancet*, vol. 359, pp. 677-8, 2002.
- [14] M. Haissaguerre, F. Extramiana, M. Hocini, B. Cauchemez, P. Jais, J. A. Cabrera, J. Farre, A. Leenhardt, P. Sanders, C. Scavee, L. F. Hsu, R. Weerasooriya, D. C. Shah, R. Frank, P. Maury, M. Delay, S. Garrigue, and J. Clementy, "Mapping and ablation of ventricular fibrillation associated with long-QT and Brugada syndromes," *Circulation*, vol. 108, pp. 925-8, 2003.
- [15] D. O. Arnar, D. Xing, H. Lee, and J. B. Martins, "Prevention of ischemic ventricular tachycardia of Purkinje origin: role for alpha²-adrenoceptors in Purkinje?," *Am J Physiol Heart Circ Physiol*, vol. 280, pp. H1182-H1190., 2001.
- [16] R. P. Holland and H. Brooks, "The QRS complex during myocardial ischemia. An experimental analysis in the porcine heart," *J Clin Invest*, vol. 57, pp. 541-550, 1976.
- [17] R. L. Hamlin, R. R. Burton, S. D. Leverett, and J. W. Burns, "Ventricular activation process in minipigs," *J Electrocardiol*, vol. 8, pp. 113-116, 1975.