SURGERY FOR ACQUIRED CARDIOVASCULAR DISEASE

CRYOABLATION OF VENTRICULAR TACHYCARDIA GUIDED BY RETURN CYCLE MAPPING AFTER ENTRAINMENT

Takashi Nitta, MD
Masataka Mitsuno, MD
Chris K. Rokkas, MD
Richard Lee, MD
Richard B. Schuessler, PhD
John P. Boineau, MD

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Background: Although the implantable cardioverter-defibrillator effectively prevents sudden cardiac death, patients are still prone to recurrence of ventricular tachyarrhythmias. Electrophysiologically guided surgery is the most effective modality in abolishing ventricular tachycardia, having a lower recurrence rate than pharmacologic therapy or catheter ablation. Return cycle mapping after entrainment has been shown to localize the central common pathway, which is the target region for ablation, without pacing at the pathway or recording the potentials from the pathway.

Methods: To determine the accuracy and usefulness of return cycle mapping in surgery for ventricular tachycardia, we cryoablated 8 morphologies of ventricular tachycardia induced in postinfarction dogs with the guidance of return cycle mapping. The ventricular tachycardia was entrained from 3 to 5 different epicardial sites at a paced cycle length 10 to 20 ms shorter than the ventricular tachycardia cycle length and the epicardium was mapped with 61 unipolar electrodes during cessation of entrainment to construct return cycle maps. The return cycle was determined by subtracting the first activation time from the second activation time after the last stimulus in each electrode location, and the maps were then displayed on a computer.

Results: The total analysis process was completed within 3 minutes by means of a computer with custom-made programs. The activation map during ventricular tachycardia did not localize the central common pathway in any morphology of ventricular tachycardia, because the pattern of activation was concentric and diastolic potentials were not recorded. Cryoablation of the region where the isopotential lines of the return cycle equal to the ventricular tachycardia cycle length intersected resulted in termination of ventricular tachycardia in all morphologies. The intersection was 26 ± 9 mm from the earliest activation site. Epicardial mapping with 253 electrodes during cryothermia showed that the region localized by return cycle mapping was the central common pathway sandwiched between the lines of conduction block and that the cryolesion connected the lines of block, blocked the rotating wave front, and resulted in termination of the ventricular tachycardia.

Conclusion: Return cycle mapping provides an accurate and rapid means of localizing the central common pathway without the need for recording potentials from the pathway or pacing at the pathway in ablation for ventricular tachycardia. (J Thorac Cardiovasc Surg 2001;121:249-58)

From the Division of Cardiothoracic Surgery, Washington University School of Medicine, St Louis, Mo.

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Address for reprints: Richard B. Schuessler, PhD, Washington University School of Medicine, Division of Cardiothoracic Surgery, Box 8234-3308 CSRB, 660 S Euclid Ave, St Louis, MO 63110.

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Although the implantable cardioverter-defibrillator effectively prevents sudden cardiac death in patients with refractory ventricular tachyarrhythmias,¹⁻⁴ the patients are still prone to recurrence of the tachyarrhythmias even with a cardioverter-defibrillator implanted. Direct surgery offers a high likelihood of cure and remains an important therapeutic option for refractory ventricular tachycardia (VT). Electrophysiologically guided surgery results in a low postoperative inducibility and recurrence of VT.⁵⁻⁷ Therefore, precise localization of the substrate for the VT is essential for a definitive surgical procedure. During the past 2 decades, intraoperative mapping has been focused on locating the earliest activation site (EAS) of VT. However, recent electrophysiologic studies in patients and dogs with infarctions revealed that the central common pathway (CCP) as the target region for ablation of VT has a significantly greater success rate.⁸⁻¹¹ The difference between the CCP ablation and the EAS ablation is illustrated in Fig 1. Reentry is abolished by creating a conduction block at a region between the lines of conduction block. This is efficiently accomplished by the CCP ablation, because it is only necessary to ablate a small amount of myocardium at the CCP to block the reentrant activation. On the other hand, the EAS ablation requires a greater amount of myocardium to be ablated to block the reentrant activation. Furthermore, insufficient ablation may allow activation conducting through a gap between the line of conduction block and the ablation scar, subsequently resulting in a recurrence of the VT.

Endocardial resection or cryoablation of the broad area around the EAS may destroy a considerable amount of normal viable myocardium that does not participate in reentry. This may increase the risk of postoperative cardiac dysfunction. Therefore, precise localization of the CCP of the reentrant circuit is required both for eliminating VT and for avoiding the unnecessary resection or ablation of normal myocardium. The CCP can be localized by high-resolution activation mapping using hundreds of electrodes.^{10,11} However, this method is frequently hampered by fractionated and low-voltage electrograms at the CCP (Fig 1). Extensive editing is generally required to determine the activation time at the CCP, and this process prolongs the intraoperative mapping. Entrainment mapping has been widely used in catheter ablation, as well as in intraoperative mapping. However, this technique is basically a site-by-site mapping method. The CCP is never localized if the electrode is not located at the pathway. In addition, the pacing threshold is elevated and the potentials are usually of low voltage and frag-

mented at the pathway. Therefore, a new intraoperative mapping technique that is capable of localizing the CCP without the need for analyzing potentials from the pathway would provide rapid and accurate mapping of the critical region of VT.

In dogs subjected to infarction, we¹² have previously shown that return cycle mapping after entrainment localizes the CCP without pacing at the pathway or recording the potentials from the pathway. The mechanism of return cycle mapping is illustrated in Fig 2. Entrainment is a continuous resetting of a tachycardia by pacing. The return cycle is the time interval between the first and second activations after cessation of entrainment. As shown in *panels 1* and *2* of Fig 2, the return cycle distribution represents a characteristic pattern that depends on the spatial correlation between the stimulation site and the CCP. At *region A*, where the last stimulus causes the first activation after entrainment, the return cycle is longer than the VT cycle length. At *region B*, which is on the CCP, the return cycle equals the VT cycle length, because the revolution time around the conduction block is the cycle length of the tachycardia. At *region C*, where the preceding stimulus causes the first activation, the return cycle equals the pacing cycle length. Adjacent to the region where the antidromic activation of the stimulus and the orthodromic activation of the preceding stimulus collide, there is another region where the return cycle equals the VT cycle length. This isochrone is connected to the lines of conduction block. As the site of stimulation shifts, the return cycle distribution changes, and the return cycle isochrone equal to the VT cycle length shifts (*panels 1-3* and *1-4*). However, the isochrone always converges on the lines of block irrespective of the stimulation site. Moreover, potentials from the CCP are not necessarily required to have the return cycle isochrones converge on the lines of block. This is because the degree of shift of the return cycle isochrone is more gradual at the region closest to the reentrant circuit than at the region most distant from the circuit. The presence of slow conduction at the edge of the lines of block allows the collision region to rotate slowly at this region as the stimulation site shifts.¹³ As a result, the return cycle isochrones converge on the lines of block nearest the entrance of the CCP even without the potentials from the pathway. We have shown that an interelectrode distance of 8.5 mm provides sufficient resolution for successful tachycardia termination guided by return cycle mapping.

The objective of this study was to determine whether return cycle mapping after entrainment is a feasible method for precise localization of the target region to

achieve successful ablation during intraoperative mapping of VT. Specifically, cryothermia was applied at the region localized by 61-channel return cycle mapping to determine whether the accuracy of the mapping method is sufficient in a canine infarction model of VT. Then, the location of the mapped site was verified by 253-channel high-resolution activation mapping.

Materials and methods

Experimental procedures. Myocardial infarction was induced in the anterior left ventricle by occlusion and reperfusion of the left anterior descending coronary artery (LAD) in 17 adult mongrel dogs of either sex, weighing 23 to 37 kg. The experimental procedures have been described previously.¹¹ In brief, through a left anterolateral thoracotomy, the LAD was occluded at the portion proximal to the branching of the first diagonal artery for 2 hours, followed by reperfusion. Intravenous lidocaine (Xylocaine) was given during and after LAD occlusion. Three animals died within 24 hours after LAD occlusion. In the remaining 14 animals, the mapping study was performed 4 days after LAD occlusion. Each animal was reanesthetized, intubated, and ventilated as described before. The heart was exposed through a median sternotomy and the right atrium and femoral artery were cannulated. Normothermic cardiopulmonary bypass was instituted to maintain stable hemodynamics during sustained VT. After the electrophysiologic study and the cryoablation of the induced VT were completed, the animal was killed. The heart was excised and the extent of the myocardial infarction was determined microscopically. All animals received humane care in compliance with the "Principles of Laboratory Animal Care" formulated by the National Society of Medical Research and the "Guide for the Care and Use of Laboratory Animals" prepared by the National Academy of Science and published by the National Institutes of Health (NIH publication No. 86-23, revised 1996). In addition, the study protocol was approved by the Washington University Animal Studies Committee.

Electrophysiologic study and cryoablation of VT. The ventricles were covered with an electrode patch carrying 61 unipolar electrodes. The electrode patch was made of a silicone sheet molded to fit the convexity of the ventricles. The patch had holes to allow epicardial pacing and recording of epicardial electrograms simultaneously. The periphery of the patch was slit to provide continuous contact between the electrodes and the epicardial surface during a whole cardiac cycle. The patch was fitted with 61 custom-manufactured, silver-plated unipolar electrodes. The diameter of the electrode tip was 2 mm. The interelectrode distance between the unipolar electrodes was 5 to 10 mm. The location of unipolar electrodes is shown in Fig 3 as a polar view of the ventricles.

While the animals were maintained on normothermic cardiopulmonary bypass, programmed electrical stimulation (Bloom model DTV 101, Reading, Pa) was performed to induce VT. Each stimulation was performed through the

Fig 1. Difference between the CCP ablation and the EAS ablation. The *upper panel* illustrates the figure-of-8 reentry model. The CCP is a narrow isthmus between the lines of conduction block that are depicted as *bold lines*. The EAS locates at the exit from the pathway. The *arrows* indicate activation wave fronts. Each *shaded region* indicates the region to be ablated required to terminate reentry. The *lower panel* shows the electrocardiogram and the electrograms recorded at the CCP and at the EAS during VT in an animal in this study. Note that the electrograms at the CCP are low voltage and polyphasic, whereas the electrograms at the EAS have monophasic potentials with higher voltage and steep deflection. See text for detailed explanation. CCP, Central common pathway; EAS, earliest activation site.

bipolar electrodes sutured on the right ventricular outflow tract. A pacing threshold was determined, and all stimulation was performed at a pulse width of 2 ms and at twice the diastolic threshold. After a train of 8 paced beats (S1) at a paced cycle length of 300 ms, single or double extrastimuli (S2 and S3) were delivered at varying coupling intervals until VT was induced. Once a stable sustained VT was induced, continuous pacing was performed by means of handheld bipolar electrodes through a hole of the electrode patch to entrain the VT from multiple ventricular sites. The pacing cycle length was set 5 to 10 ms less than the VT cycle length. Surface electrocardiograms, pacing artifacts, and reference electrograms from the unipolar electrodes were continuously monitored on an oscilloscope, and the VT cycle length was displayed digitally in a beat-by-beat fashion. After constant fusion beats in the surface electrocardiogram and constant capture of the ref-

Fig 2. Mechanism of return cycle mapping after entrainment. *Panel 1* illustrates the figure-of-8 reentry circuit and the activation pattern during entrainment from a region proximal to the CCP. The pacing site is denoted as a rectangle and the lines of block are denoted as bold lines. Arrows indicate the activation sequence. The antidromic activation of the Nth pacing collides with the orthodromic activation of the (N-1)th pacing. The fine lines connected to the lines of block are the lines of collision and the transition from Nth to (N-1)th activation. *Panel 2* represents the activation sequence and the return cycle after entrainment at sites A, B, and C in panel 1. *Panel 3* illustrates that the return cycle isochrone equal to the VTCL shifts as the stimulation site changes, but always converges on the lines of block irrespective of the stimulation site. *Panel 4* shows that the potentials from the CCP are not necessarily required to have the return cycle isochrones converge on the lines of block. See text for detailed explanation. CCP, Central common pathway; VTCL, ventricular tachycardia cycle length; PCL, pacing cycle length.

reference electrogram at the pacing rate were demonstrated, the pacing was abruptly terminated. The VT was repeatedly entrained from 3 to 5 epicardial locations at the same pacing cycle length.

Unipolar electrograms, as well as surface electrocardiograms, pacing artifacts, and reference electrograms were recorded during and after entrainment of each VT by means of a 256-channel computerized data acquisition and analysis system. The mapping system was based on a VaxStation II/GPX graphics workstation connected to two 128-channel PDP 11/23+ based data acquisition subsystems (Compaq Computer Corporation, Houston, Tex). Unipolar electro-

grams were recorded at a gain of 250, with a frequency response of 0.5 to 500 Hz. Each channel was digitized at 1000 Hz with a 12-bit resolution. Local activation times were determined at the time of the maximum negative derivative in each unipolar electrogram. All electrograms were edited visually to verify accuracy of the computer-picked activation times. Activation times during the first and second cardiac cycles after cessation of pacing were analyzed. The custom-made program calculated the return cycle, which is the time interval between the first and the second activation after entrainment, by subtracting the first activation time from the second activation time at each unipolar electrode location. The return cycle map was constructed as an isochrone map for each stimulation. Then the region where the return cycle isochrone equal to the VT cycle length converged and intersected was identified. The duration of the mapping process to construct the return cycle maps was determined.

Cryoablation of the VTs was attempted guided by return cycle mapping with the use of 61 epicardial electrodes. The intersection of the return cycle isochrone equal to the VT cycle length was identified on the computer display. Then cryothermia was applied to the intersection. In cases of multiple intersections, the epicardium between the intersections was cryoablated. To examine where in the reentrant circuit the region localized by the return cycle mapping was located and how the cryothermia terminated the VTs, the anterior left ventricle was mapped with 253 electrodes during the application of cryothermia. The electrodes were made of silver balls (diameter, 1 mm) mounted on a silicone sheet with an inter-electrode distance of 3 to 5 mm. Epicardial cryothermia was performed at a temperature of -60°C by means of a cryoprobe with a tip diameter of 3 mm (Frigitronics, Inc, Coopersurgical, Shelton, Conn) that was introduced through a hole on the electrode patch. The effect of the cryothermia on VT was verified by applying cryothermia during VT. After the VT was terminated, programmed electrical stimulation was performed again. Elimination of VT was determined as inability to induce the same VT despite the whole stimulation protocol. Each VT was characterized by the cycle length, axis, and configuration of the QRS during VT.

After each experiment, the data from 253 electrodes were analyzed and the distance between the EAS and the region where the return cycle isochrone equal to the VT cycle length intersected was determined on the electrode patch for all the VTs. The EAS was defined as the site where the local activation occurred earliest referenced to the QRS onset. Correlation between the distance and the VT cycle length was examined statistically by linear regression analysis. The distances were expressed as mean $\pm$ 1 standard deviation.

Results

Characteristics of VTs. A total of 24 morphologies of monomorphic VT were induced. Eight of the 24 morphologies of VT were stable and sustained for more than 3 minutes. These morphologies of VT were subjected to the study. The cycle length of the VTs ranged

from 142 to 244 ms (175 ± 36 ms). Epicardial mapping with 61 electrodes showed that the EAS of VTs was distributed over the anterior left ventricle and was located at the border zone between the infarct and intact myocardium in most VTs. The activation maps during VT did not always localize the CCP, because the pattern of activation was concentric and diastolic potentials were recorded at only 2 to 4 electrode locations.

Return cycle maps after entrainment. Transient entrainment was demonstrated in all morphologies of VT. Rapid ventricular pacing during VT was performed from 3 to 5 different sites at the same pacing cycle length. The pacing cycle length ranged from 130 to 230 ms and was 89% to 95% ($93\% \pm 2\%$) of the VT cycle length. Return cycle maps after entrainment were constructed from 61 epicardial electrodes for each pacing site in all the VTs. There was no need for extensive editing in constructing the return cycle maps. This is because the electrograms were monophasic and high voltage at the normal myocardium, so that the activation times were determined mostly by computerized analysis. Each map was displayed on a computer within 3 minutes. The return cycle maps demonstrated the characteristic pattern that was related to the spatial correlation between the stimulation site and the reentrant circuit as described above. The site where the return cycle isochrones equal to the VT cycle length intersect was identified in all the VTs. An example is illustrated in Fig 4. The cycle length of the VT was 168 ms. The earliest activation occurred at the left ventricular apex, and the activation spread toward the base of the ventricles. Diastolic potentials were recorded at 3 electrode locations at the anterior left ventricle. However, the reentrant circuit of the VT was not determined. The VT was entrained from the right ventricular outflow epicardium at a paced cycle length of 160 ms. The return cycles after entrainment were longest near the pacing site and shortened moving away from the pacing site. The VT was entrained from two more different ventricular sites at the same paced cycle length. The intersections of the return cycle isochrones equal to the VT cycle length (168 ms) were identified at the region 20 mm from the EAS of the VT.

Cryoablation guided by return cycle mapping. All the VTs were terminated by no more than three applications of cryothermia guided by return cycle mapping that used 61 epicardial electrodes. Activation mapping using 253 electrodes during application of cryothermia revealed that the region localized by return cycle mapping was the CCP of the reentrant circuit between the lines of conduction block, whereas the EAS was located at the exit of the pathway. The VT cycle length was

Fig 3. Location of 61 unipolar electrodes over the epicardium. The schema represents the polar view of the ventricles as if they are observed from the apex. Nineteen electrodes are distributed over the right ventricular epicardium, and 31 over the left. Eleven electrodes are located at the interventricular groove along the LAD. AO, Aorta; PA, pulmonary artery; RA, right atrium; LAA, left atrial appendage; IVC, inferior vena cava.

always prolonged before the termination of VT. An example is illustrated in Figs 5 and 6. The cycle length of the VT was 153 ms and the EAS was at the anterior left ventricle adjacent to the LAD. Return cycle mapping with 61 electrodes suggested that the CCP was located at the inferior left ventricle between the apex and the LAD. The VT was terminated by a cryothermia applied to the left ventricular epicardium between the intersections of the return cycle isochrones equal to the VT cycle length. Extensive editing was required in off-line analysis of the data from 253 electrodes. The electrograms were low voltage and fractionated at the CCP, as shown in Fig 1. The activation maps using 253 electrodes during application of cryothermia are illustrated in Fig 6. The region localized by return cycle mapping was the entrance of the CCP between the lines of conduction block. The baseline VT cycle length was 153 ms. After application of cryothermia, and as the diameter of the cryolesion became larger, the cycle length of the VT gradually prolonged. The cryolesion served as an anatomic block that elongated the reentrant circuit and slowed the VT. After 20 seconds of cryothermia, the VT was terminated and sinus rhythm resumed. The cryolesion connected the lines of block, blocked the rotating wave front, and resulted in termination of the VT.

Fig 4. An example of return cycle mapping. The *upper panel* shows the electrocardiogram during cessation of entrainment of a VT induced in a 4-day-old canine infarct. The activation maps during the first and second activation after cessation of entrainment are shown in the *middle panels*. *Boxed areas* on the electrocardiogram are the data window analyzed to construct the activation maps. The VT was entrained from the right ventricular outflow tract (denoted as a *rectangle*) at a pacing cycle length of 160 ms. After cessation of pacing, the VT resumed at a cycle length of 168 ms with the EAS located at the inferior left ventricle near the apex (denoted as an *asterisk*). The activation times are represented as color codes with 20-ms increments. The *left lower panel* shows the return cycle map constructed from these activation maps, by subtracting the first activation time from the second activation time after cessation pacing. The return cycle was also represented as color codes with 10-ms increments. The return cycle was longest at the region around the pacing site and was shorter at the region near the earliest activation site. The *blue* indicates the region where the return cycle equaled the pacing cycle length, suggesting orthodromic activation by the preceding stimulus. The *red line* indicates the return cycle isochrone equal to the VTCL. As we changed the pacing site from the red to the blue rectangle, this isochrone shifted from the red to the blue line as shown in the *right lower panel*. These lines converged on a region 20 mm away from the earliest activation site and formed intersections. The VT was terminated by a cryothermia to the region between the intersections. ECG, Electrocardiogram; AO, aorta; PA, pulmonary artery; RA, right atrium; LAA, left atrial appendage; IVC, inferior vena cava; VTCL, ventricular tachycardia cycle length.

Fig 5. Another example of return cycle mapping. The cycle length of the VT was 153 ms and the earliest activation site was at the anterior left ventricle adjacent to the LAD (denoted as an *asterisk*). The VT was entrained at a pacing cycle length of 140 ms. The *left panel* represents the return cycle map after entrainment from a site at the anterior left ventricle. The site of stimulation is denoted as a *rectangle*. The return cycle is represented as color codes with 10-ms increments, and the *red line* indicates the return cycle isochrone equal to the VTCL. The *right lower panel* shows the return cycle isochrone equal to the VTCL in the return cycle maps after entrainment from two different pacing sites that were denoted as *blue and red rectangles*. The VT was terminated by a cryothermia applied to the region between the intersections of these isochrones. For abbreviations, see Fig 4.

Distances between the EAS and the sites where cryothermia successfully terminated the VTs were plotted as a function of the cycle length of the VT in Fig 7. The distances ranged from 20 to 45 mm (26 ± 9 mm). The longer the cycle length of VT was, the larger the distance was. There was a significant linear correlation between the distance and the cycle length of VT.

Discussion

Return cycle mapping. The most important finding in the present study was that cryoablation guided by return cycle mapping with 61 electrodes terminated all the VTs that were induced in postinfarction dogs. This suggests that return cycle mapping localizes the critical region of the reentrant circuit accurately without the use of high-resolution mapping electrodes. Furthermore, high-resolution activation mapping confirmed that the region localized by 61-channel return cycle mapping was the CCP sandwiched between the lines of conduction block. Accurate mapping is important both to direct the definitive surgical procedure and to preserve viable myocardium and prevent postoperative cardiac dysfunction by avoiding unnecessary myocardial excision. This study also showed that the regions where cryothermia terminated VTs were separated from the EASs by 26 ± 9 mm, and the distance between the two regions was larger in the VT with longer cycle

length. This suggests that a VT with slow heart rate has a larger reentrant circuit and the patients may be prone to failure or recurrence of the VT after an ablation at the EAS.

Localization of the target region for cryoablation was accomplished within 3 minutes by return cycle mapping with 61 electrodes. Computerized processing of the data for constructing the return cycle maps allowed rapid localization of the critical region in the reentrant circuit. Moreover, the CCP was localized with only a few potentials from the CCP by return cycle mapping. Localization of the CCP by high-resolution activation mapping with hundreds of electrodes requires careful analysis and extensive editing of the data, because the potentials at the CCP are frequently low voltage and fractionated. This process is time consuming and not suitable for intraoperative mapping. In contrast, return cycle mapping allows accurate and rapid localization of the CCP. Thus, return cycle mapping might reduce the time for intraoperative mapping and increase the success rate in surgery for VT.

Clinical implications and limitations. There would be some limitations and issues to be considered and solved in applying the return cycle mapping in intraoperative mapping of human VT. Determination of the activation times and calculation of the return cycle can be easily performed with currently available mapping

Fig 6. Electrocardiograms and activation maps of the anterior left ventricle during cryoablation guided by return cycle mapping. A total of 253 unipolar electrodes were distributed over the anterior left ventricle. The electrode patch was located from the basal left ventricle 1 to 2 cm away from the left atrioventricular groove to the inferior apex of the left ventricle. The left margin of the electrode array was adjacent to the LAD. The right margin was located at the obtuse margin. *Boxed areas* on the electrocardiogram are the data window analyzed to construct the activation maps. *Panel 1* shows the baseline activation map during VT before the application of cryothermia. The cycle length of the VT was 153 ms. Cryothermia was applied at the region localized by return cycle mapping with 61 electrodes as shown in Fig 5. The mapped region was the entrance of the CCP between the lines of conduction block. *Panels 2 and 3* show the activation maps 5 and 10 seconds after the start of cryothermia, respectively. As the diameter of the cryolesion became larger, the cycle length of the VT prolonged by degrees. *Panel 4* shows the activation map during the last VT cycle before its termination after 20 seconds of cryothermia. The cryolesion connected the lines of block, blocked the rotating wave front, and resulted in termination of the VT. For abbreviations, see Fig 4.

systems. Once a stable VT is induced, entrainment of the VT is performed from a ventricular site at a paced cycle length slightly shorter than the cycle length of the VT. Then, the electrograms are recorded during cessation of pacing. The first two consecutive activation times after the last pacing stimulus are determined and the time interval between the activation times (return cycle) is calculated in each electrode location. The distribution of the return cycle is displayed on a computer as an isochronal contour. An important point in the return cycle mapping is to locate each electrode on the computer accurately on a 2- or 3-dimensionally dis-

played ventricular model, because the CCP is localized by the intersections of the isochronal lines of the return cycle equal to the cycle length of VT. It is also essential to refer to the location of electrodes in positioning an ablative device at the target ventricular region that is determined by the return cycle mapping.

In the present study, only the epicardium was mapped, because the reentrant circuit of the induced VT was located in the thin epicardial tissue overlying a subendocardial infarct in this animal model. However, the patients with ischemic VT may have a more complex electrophysiologic basis for their reentrant VT

Fig 7. Distance between the earliest activation site and the sites where cryothermia successfully terminated the VT guided by return cycle mapping. The distance was plotted as a function of the cycle length of the VT (VTCL).

than the canine model. Septal involvement may be an important electrophysiology of VT in patients with extensive anterior left ventricular myocardial infarction.¹⁴ We may need to map ventricles endocardially and construct the return cycle isochrones on the endocardial surfaces displayed on a computer in these patients. Intramural reentry may also be the mechanism of the VT in some patients.¹⁵⁻¹⁷ Since this mapping technique does not require the potentials from the CCP to localize the pathway, it could be possible to extend the technique for 3-dimensional return cycle mapping, which may be helpful to map the intramural reentrant circuit. However, further studies are necessary to determine whether a CCP located intramurally can be localized by interpreting return cycles mapped endocardially or epicardially.

This mapping technique greatly relies on ability to entrain a VT. Entrainment of a VT requires an excitable gap in the VT cycle to be activated. VT with rapid heart rate may have a narrow excitable gap and thus may not be entrained continuously at a faster pacing rate.¹⁸ Location and size of the reentrant circuit can be different during and after entrainment of a fast VT, and this may impair the accuracy of the mapping. Therefore, the return cycle mapping would be useful in VTs with a moderate to slow heart rate. In fact, the present study showed that ablation at the EAS may be prone to failure or recurrence of the VT, particularly in patients with a slow VT, because the distance between the EAS and the region where cryothermia terminated VT was large in the VT with slower heart rate. Furthermore, the

return cycle mapping requires repeated times of entrainment of a VT by changing the stimulation site. Therefore, the QRS morphology and cycle length of the VT have to be stable and the VT should be reproducibly inducible by programmed stimulation.

This is a preliminary short-term study of a newly developed mapping method. The advantages and limitations in this technique should be determined in a long-term study. It is interesting to determine whether the inducibility of VT is lower after ablation of CCP guided by the return cycle mapping than after ablation of the EAS. Amount of myocardium that must be ablated to eliminate VT should also be compared between the CCP ablation and the EAS ablation.

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