Return Cycle Mapping After Entrainment of Ventricular Tachycardia

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Background—The central common pathway, which is the target for ablation in reentrant ventricular tachycardia, can be localized by entrainment mapping techniques. However, localization of the pathway is not always possible because of the elevated pacing threshold and the low voltage and fractionated potentials at the pathway. We examined whether return cycle mapping after entrainment localizes the pathway without pacing at the pathway or recording the potentials from the pathway and determined the required electrode resolution to localize the pathway.

Methods and Results—Epicardial mapping was performed with 253 unipolar electrodes during and after entrainment of 13 morphologies of ventricular tachycardia that were induced in dogs 4 days after infarction. The return cycle was calculated by subtracting the first activation time from the second activation time after the last stimulus and the return cycle distribution map was constructed for each stimulation site. The return cycle isochrones equal to the ventricular tachycardia cycle length converged on the lines of conduction block irrespective of the stimulation site, and the central common pathway was localized at the region between the intersections of the return cycle isochrones after entrainment from different stimulation sites. The potentials from the central common pathway were not required to localize the pathway, and the mapping accuracy did not change with or without analysis of the potentials from the pathway. According to the correlation between the electrode resolution and the mapping accuracy, an interelectrode distance of 8.5 mm was estimated as sufficient resolution for successful tachycardia termination during radiofrequency ablation guided by return cycle mapping.

Conclusions—Return cycle mapping after entrainment localizes the central common pathway without pacing at the pathway or recording the potentials from the pathway. This new mapping technique could improve the success rate of the ablative procedures. (Circulation. 1998;97:1164-1175.)

Key Words: tachycardia • reentry • entrainment • mapping • ventricles
The return cycle after entrainment, which is the time interval between the first and second activation times after cessation of pacing, is specific to the recording site in the reentrant circuit. We have previously demonstrated that return cycle mapping after entrainment demonstrates a characteristic pattern that depends on the pacing rate and the spatial correlation between the stimulation site and the reentrant circuit and that the return cycle isochrones equal to the VT cycle length converge on the lines of conduction block of the reentrant circuit irrespective of the stimulation site. The hypothesis of this study is that the return cycle isochrones equal to the VT cycle length localize the lines of block. The objective of this study was to demonstrate that return cycle mapping after entrainment localizes the CCP without pacing at the pathway or recording potentials from the pathway. Specifically, the goals of this study were (1) to determine if the potential from the CCP was essential in localizing the lines of block by return cycle mapping and (2) to determine the required electrode resolution to localize the pathway by this mapping technique.

Methods

In 18 adult mongrel dogs of either sex, weighing 23 to 37 kg, anesthesia was induced with intravenous sodium thiopental (20 mg/kg) and was maintained with inhaled halothane (1% to 3%). The animals were intubated and ventilated with the use of a volume-limited ventilator (Harvard Apparatus Co). The heart was exposed through a left thoracotomy at the fourth intercostal space with sterile surgical technique and was suspended in a pericardial cradle. The left anterior descending coronary artery (LAD) was carefully dissected at the portion proximal to the branching of the first diagonal artery and was occluded for 2 hours. A bolus injection of intravenous lidocaine (2 mg/kg) was given 5 minutes before the coronary occlusion, and a continuous infusion of lidocaine (1 mg/kg per hour) was administered for 5 hours after the coronary occlusion. Another bolus injection of lidocaine (1 mg/kg) was given 5 minutes before the reperfusion. The chest was closed in layers, and the animals were allowed recover.

Four days after the surgery, the animals were reanesthetized with intravenous sodium pentobarbital (30 mg/kg). Supplemental doses of thiopental (10 mg/kg) were given as needed to maintain the surgical plane of anesthesia. The animals were intubated and ventilated as described above. A femoral arterial line was inserted to monitor systemic arterial pressure continuously. Arterial blood samples were drawn every 30 minutes to determine PAO2, acid-base balance, and electrolyte levels. Ringer’s lactate solution was continuously infused, and sodium bicarbonate, potassium chloride, and calcium chloride were supplemented as indicated to maintain pH and electrolyte within normal values. The heart was exposed through a median sternotomy and was suspended in a pericardial cradle. After systemic heparinization (3 mg/kg), the right atrium and femoral artery were cannulated, and normothermic cardiopulmonary bypass was instituted to maintain stable hemodynamics during sustained VT.

An electrode patch containing 253 unipolar electrodes and 16 bipolar pacing electrodes was sutured on the epicardium of the left ventricle to cover the infarcted and surrounding area. The electrode patch was made of a silicon sheet molded to fit the convexity of the left ventricular free wall. Both unipolar and bipolar electrodes were constructed from silver balls (diameter, 1 mm) and from Teflon-insulated silver wires (diameter, 125 μm). The interelectrode distance between the unipolar electrodes was 3 to 5 mm, with higher resolution over the infarcted area and lower resolution over the remaining areas. The intralead electrode distance of the bipolar electrodes was 1 mm. The location of each unipolar and bipolar electrode is shown in the left panel of Fig 1.

Programmed electrical stimulation (DTU-101, Bloom Associates Ltd) was performed to induce VT. Each stimulation was performed through the bipolar pacing electrodes mounted on the electrode patch. 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 eight 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 to entrain the VT from various epicardial sites through bipolar electrodes. The pacing cycle length was set to 5 to 10 ms less than the VT cycle length. Surface ECGs, pacing artifacts, and reference electrograms from the unipolar electrodes were continuously monitored, and the VT cycle length was displayed digitally in a beat-by-beat fashion. After constant fusion beats in the surface ECG and constant capture of the reference electrogram at the pacing rate were demonstrated, the pacing was abruptly terminated. Several attempts to entrain the VT were repeated from different bipolar electrode locations. Entrainment of the tachycardia was directly verified by the activation maps during pacing.

A 256-channel computerized data acquisition and analysis system was used to collect, process, and display data. The mapping system was based on a VaxStation II/GPX graphics workstation connected to two 128-channel PDP 11/23+ based data acquisition subsystems. Unipolar electrograms were recorded at a gain of 250, with a frequency response of 0.05 to 500 Hz. Each channel was digitized at 1000 Hz with a 12-bit resolution. Two-hundred fifty-three unipolar electrograms, as well as surface ECGs, pacing artifacts, and reference electrograms, were recorded during and after entrainment of each VT. The data were stored on the hard disk of the VaxStation and on an optical disk. The optical disks from each experiment were replayed afterward for off-line data analysis. 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 maps of the first and second cardiac cycle after entrainment were constructed. A site of conduction block was defined as the site between any two adjacent electrodes having an activation time gradient of >10 ms/mm, associated with a different activation sequence on opposite sites of the putative block and a different morphology of the electrograms. The return cycle after entrainment was calculated by subtracting the first activation time from the second activation time after the last stimulus at each unipolar electrode location. The return cycle map was constructed as an isotemporal map for each stimulation site. The region where the isochrones equal to the VT cycle length converged and intersected was identified from the return cycle maps in each VT.

To define if analysis of the potentials from the CCP is essential for localizing the pathway by return cycle mapping after entrainment, the potentials from the electrodes located adjacent to the lines of block and the electrodes located between the lines were not analyzed (Fig 6). In addition, to determine the required electrode resolution for the mapping technique, the number of the recording electrodes was decreased from 253 to 127, 64, and 32 (Fig 7). Analysis of the potentials from every other electrode provided 127-electrode analysis. Analysis of the potentials from every fourth and every eighth electrode provided 64 and 32-electrode analyses, respectively. The accuracy of the mapping technique with and without analyzing the potentials from the CCP and with various electrode resolutions was evaluated by the distance between the mapped site demonstrated by the return cycle mapping with each technique and the lines of block during VT defined by the activation maps with 253 electrodes. The distances were measured on the electrode patch and were expressed as mean±1 SD. The accuracy of the mapping technique with and without analyzing the potentials from the CCP was compared by Student’s paired t test. Mapping accuracy with various electrode resolutions was compared with mapping accuracy using 253 electrodes by Student’s paired t test. In addition, the effect of average interelectrode distance on the mapping accuracy was examined by linear regression analysis and analysis of variance. A value of P<.05 was considered statistically significant.
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 1985). In addition, the study protocol was approved by the Washington University Animal Studies Committee.

**Results**

**VT Characteristics**

A total of 41 morphologies of sustained monomorphic VT were induced in 18 animals. In 7 animals, the reentrant circuit was mapped on the epicardial surface in 13 morphologies of VT, and the return cycle distribution after entrainment was examined. The VT cycle lengths ranged from 127 to 241 ms (170±37 ms). The reentrant circuits demonstrated a double loop circuit with lines of conduction block and a CCP between the lines. The lines of block were perpendicular to the LAD in 11 of 13 morphologies of VT and parallel to the LAD in 2 morphologies. The lines of block were longer and the CCP was narrower in the VTs in which the lines were perpendicular to the LAD, compared with the VTs in which the lines were parallel to the LAD. The width of the CCP ranged from 6 to 30 mm and the length of the line of block ranged from 9 to 44 mm. To evaluate conduction over the infarct, activation maps were constructed during continuous pacing from the bipolar electrode located at the anterior left ventricle, before the induction of VT in all animals. An example is shown in Fig 1. The lines of conduction block seen during VT were not evident during pacing, suggesting that the conduction block during VT was functional. These characteristics of the reentrant circuit were present also in the other VTs in this study. In the histological examination of the excised heart, extensive infarction of the anterior left ventricle was seen. The number of surviving subepicardial muscle layers varied from one third of the total ventricular wall thickness to very thin layers of muscle. A transmural extent of infarction was also found.

**Return Cycle Distribution**

The VTs were entrained from three to five different epicardial sites and the effects of the stimulation site on the return cycle distribution were studied. Sample data are illustrated in Figs 2, 3, and 4. The return cycle distribution was divided into two regions: longer than the pacing cycle length and equal to the pacing cycle length. In the region where the last stimulus caused the first activation after the cessation of pacing, the return cycle was greater than the pacing cycle length. In the region where the second to last stimulus caused the first activation after the cessation of pacing, the return cycle equaled the pacing cycle length. These return cycle distribu-
shifted and rotated around the lines of block as the spatial correlation between the stimulation site and CCP changed. Two different patterns of transition were observed between these return cycle distributions. The transition was precipitous at the region where the orthodromic activation was transposed from the last stimulated activation to the activation of the preceding stimulation (sites E and F in Fig 2, sites E and F in Fig 3, and sites G and H in Fig 4), while it

Figure 2. Activation sequence and return cycle after entrainment from a site proximal to the central common pathway (CCP). The VT shown in Fig 1 was entrained at a paced cycle length of 140 ms. ECG is shown, with pacing artifacts and electrograms from selected sites (A-J) in the maps. The vertical line indicates the time of the last paced stimulation. After cessation of pacing, the tachycardia resumed at a cycle length of 143 to 144 ms. The time intervals between each activation are shown as numbers in milliseconds. Arrows indicate the activation sequence. The activation map of the last stimulation is shown on the lower left and the return cycle map is shown on the lower right. The stimulation site is denoted by a rectangle in both maps. In the activation map, time zero indicates the time of the last stimulation. Closed arrows indicate the stimulated wave fronts; open arrows indicate the wave fronts of the preceding stimulation. The return cycle isochrones are constructed at 10-ms increments from the pacing cycle length plus 5 ms. Broken bold lines denote the lines of conduction block during VT. Note that the return cycle isochrone of 145 ms coincides with the lines of block. The configuration of the figure and the map symbols in the following figures are the same as those used in this figure. Also see abbreviations in Fig 1.
was gradual at the collision region of the antidromic and orthodromic wave fronts (sites G and H in Fig 2, sites G and H in Fig 3, and sites C through J in Fig 4). Adjacent to the collision region, there was a return cycle isochrone equal to the VT cycle length, and this isochrone represented a unique spatial correlation with the lines of conduction block. An example is shown in Fig 5. Although the isochrone shifted and changed the shape as the stimulation site changed, the isochrone always converged on the lines of block irrespective of the stimulation site. As a result, the intersections of the return cycle isochrones equal to the VT cycle length after entrainment from different pacing sites coincided with the lines of conduction block in the reentrant circuit during VT. Therefore, the CCP was localized at the region between the intersections of the return cycle isochrones equal to the VT cycle length.

**Figure 3.** Activation sequence and return cycle after entrainment from the site in the CCP. The VT is the same VT as shown in Fig 1. The pacing cycle length was 140 ms, and the return tachycardia cycle length ranged from 146 to 148 ms. The ECG and electrograms from selected sites (A–J) are shown. The activation map of the last stimulation and the return cycle map are shown on the lower left and right. Note that the antidromic activation is confined to a limited region within the CCP and that the activation sequence outside the CCP is similar to the sequence during VT (Fig 1). Also see Fig 2.
The effect of recording the CCP potentials on localizing the pathway by return cycle mapping was examined. An example is shown in Fig 6. In the activation map during VT with analysis of all the potentials recorded, two lines of block and a CCP between them are evident. The return cycle map demonstrated the characteristic pattern as described above and the return cycle isochrone equal to the VT cycle length (153 ms) converged on the line of conduction block during VT. In the activation map in which the potentials from the CCP were not analyzed, a time gap of approximately 30 ms was seen between the latest activation and the earliest activation, and the lines of block and the CCP were not localized. However, the return cycle isochrones converged on a region that coincided with the line of block nearest the entrance of the CCP even when the potentials from the CCP were not analyzed.

**Figure 4.** Activation sequence and return cycle after entrainment from a site distal to the CCP. The VT is the same VT as shown in Fig 1. The pacing cycle length was 140 ms, and the return tachycardia cycle length was 145 to 146 ms. The ECG and electrograms from selected sites (A-J) are shown. The activation map of the last stimulation and the return cycle map are shown on the lower left and right. Also see Figs 1 and 2.
The number of electrodes not analyzed was 56±24 (22±9.5% of 253 electrodes), and the corresponding area was 7.0±3.5 cm² (19±9.5% of the total mapped area). The accuracy of mapping with and without analysis of the potentials from the CCP was evaluated by the distance between the intersection of the return cycle isochrones equal to the VT cycle length and the line of block localized by the activation map during VT. The distance was 1.0±1.4 mm with analysis of the potentials from the CCP and 1.5±1.3 mm without analysis of the potentials. No statistical differences were found between the two analyses with or without analysis of the potentials from the CCP.

Mapping Resolution and Accuracy of Localizing the CCP

The effect of electrode resolution on return cycle mapping was examined. Fig 7 illustrates how the mapping resolution affected the activation maps during VT and the localization of the lines of block by return cycle mapping. In the activation map with 253 electrodes (left map of panel A), almost all of the entire sequence of activation during VT was elucidated. As the number of electrodes decreased, the isochrones of activation became smooth and simple, and the location, shape, and length of the lines of block changed. As a result, the location and extent of the CCP became ambiguous with fewer electrodes (left maps of panels B, C, and D). In the right map of panel A, the return cycle isochrones equal to the VT cycle length converged on the lines of block irrespective of the stimulation site.

The effect of the interelectrode distance on the mapping accuracy of this technique is shown in Fig 8. The mapping accuracy was evaluated by the distance between the lines of block localized by the activation maps with 253 electrodes and the intersections of the return cycle isochrones equal to the VT cycle length in each mapping resolution. The distance was 1.0±1.4, 1.9±1.5, 4.0±2.6, and 6.8±3.0 mm for each analysis group of 253, 127, 64, and 32 electrodes, respectively. The average interelectrode distance was 4.3, 6.0, 8.5, and 12.0 mm for each analysis group of 253, 127, 64, and 32 electrodes. As the interelectrode distance increased, the mapping accuracy decreased. However, there was no statistical difference in the mapping accuracy between the 127- or 64-electrode analysis groups and the baseline analysis (253-electrode analysis). The mapping accuracy with 32 electrodes was significantly lower than the mapping accuracy with 253 electrodes. There was a significant linear correlation between the average interelectrode distance and the mapping accuracy (r=0.72, P<0.001).

Discussion

Mapping During VT Ablation

The most important finding in the present study is that return cycle mapping localizes the CCP without pacing at the pathway or recording potentials from the pathway. The CCP is the region where the activation wave fronts are confined to a narrow isthmus. This causes the diastolic phase in the
Figure 6. The effect of analyzing the potentials from the CCP on localizing the line of block during VT by return cycle mapping. ECG during VT induced in an infarcted heart is shown. The VT cycle length is 153 ms. The activation map during VT is shown on the middle left. The boxed area on the ECG represents the data window analyzed to construct the activation map. Entrainment of the VT was performed from three epicardial sites (A, B, and C in the lower right) at the same paced cycle length of 140 ms. The return cycle map after entrainment from the anterior left ventricular free wall (denoted as a rectangle, site A) is shown on the middle right. On the lower left, the activation map when the potentials from the CCP and the adjacent region (shaded area) were not analyzed. The asterisk shows the earliest activation site. The activation was discontinuous from 120 ms to the earliest activation. On the lower right, the return cycle isochrones equal to the VT cycle length (153 ms), corresponding to each stimulation site are illustrated. The return cycle isochrones, corresponding to each stimulation site (A, B, and C), are illustrated as broken, dotted, and solid lines, respectively. The shaded area indicates the region where the potentials were not analyzed and the bold lines denote the lines of block of the reentrant circuit. Even without the analysis of the potentials from the CCP, the return cycle isochrones equal to the VT cycle length converged on the lines of block. Also see abbreviations in Figs 1 and 2.
Figure 7. Effects of the mapping resolution on the activation maps and on the localization of the lines of block by return cycle mapping. The activation times during a ventricular tachycardia (VT) were analyzed with four degrees of mapping resolution. The VT cycle length was 140 ms. The VT was entrained from three different epicardial sites (A, B, and C) at a paced cycle length of 130 ms. Each stimulation site is denoted as a rectangle. The activation map in each mapping resolution is illustrated in the left maps and the return cycle isochrones equal to the VT cycle length (140 ms) are illustrated on the right. The numbers of channels analyzed were 253, 127, 64, and 32 for the maps in each row of A through D. The numbers in the activation maps denote the activation times during VT at each electrode location. The bold lines show the lines of block and the isochrones are drawn in 10-ms increments. On the right maps, the return cycle isochrones equal to the VT cycle length (140 ms), corresponding to each stimulation site (A, B, and C), are illustrated as broken, dotted, and solid lines, respectively. Overlap of more than two lines is expressed as a solid line.
tachycardia cycle, because of the small volume of tissue that is activated as the wave front traverses the CCP. Therefore ablation of a small amount of myocardium at the CCP is necessary to eradicate reentry and successful elimination of the VT largely depends on mapping the pathway. Unfortunately, mapping the CCP is not always feasible during surgery or catheter ablation. Therefore, this new mapping technique would help in localizing the CCP and improve the success rate of the ablative procedures. During surgery for VT, high-resolution mapping with hundreds of electrodes can exhibit the entire reentrant circuit and localize the CCP. Although computers are used to process multiple simultaneous recordings, determination of the activation times at the CCP frequently requires careful analysis and extensive manual editing of the complex and fractionated electrograms. This process significantly lengthens the duration of mapping. During catheter ablation of VT, precise localization of the CCP is essential because the ablation lesion created by the delivered energy through a catheter is small. The entrainment mapping technique is helpful for localizing the CCP with fewer electrodes. As demonstrated in Fig 3, during entrainment from a site in the CCP, the antidromic activation is confined to a limited area and the activation sequence outside the CCP is similar to the sequence during VT. As a result, the QRS morphology during entrainment is identical to the QRS morphology during VT (concealed entrainment). Unfortunately, the predictive value of concealed entrainment alone for localizing the successful ablation site is 50%. Because the concealed entrainment can be demonstrable during pacing from a bystander pathway or inner loop of the reentrant circuit, the other criteria, such as the presence of isolated mid-diastolic potentials or electrogram−QRS interval=stimulus−QRS interval, have been shown to enhance the predictive value of the concealed entrainment for successful ablation. The postspacing interval (the time interval from the last stimulus to the return cycle potential) has also been shown to predict VT termination during radiofrequency current application. Combining these criteria with the demonstration of concealed entrainment has been shown to correlate with a high likelihood of VT termination by radiofrequency energy. On the other hand, in the case in which concealed entrainment or other criteria are not demonstrated, the success rate for VT ablation is low. The present technique is a site-by-site mapping method, yet requires pacing at the CCP or recording potentials from the pathway to demonstrate that the electrode is positioned at the pathway. The pacing threshold is frequently elevated and the potentials are complex and fractionated at the CCP because the pathway usually consists of islets of surviving myocardium in the scar tissue. Return cycle mapping localizes the CCP by intersecting the return cycle isochrones equal to the VT cycle length after entrainment from the sites outside the CCP. Moreover, this technique does not necessarily require the potentials from the CCP to localize the pathway. Therefore, return cycle mapping after entrainment localizes the CCP even when no electrodes are positioned at the pathway.

Return Cycle After Entrainment

The return cycle was defined in the present study as the time interval between the first and second activation times after cessation of pacing. As shown in Figs 2 to 4, the return cycle equals the pacing cycle length at the region activated by the preceding stimulation orthodromically, because the return cycle is the time interval between the last two stimulated activation times in this region. In the rest of the region, the return cycle is the time interval between the last stimulated activation and the first tachycardia activation. This definition of the return cycle helps the distribution map of the return cycle to localize the lines of conduction block, because the computer can automatically measure and calculate the return cycle, which is merely the first time interval after the last pacing artifact. In contrast, determination of the time interval between the last stimulated activation and the first tachycardia activation after entrainment can be ambiguous at the region where the antidromic and orthodromic wave fronts collide (site H in Fig 2, site G in Fig 3, and site J in Fig 4). This is because the first tachycardia activation after entrainment is the continuation of the last paced stimulation, and it is extremely difficult to determine by the potential morphology whether the activation is caused by the last paced stimulation or the preceding stimulation at the region.

The primary mechanism for localizing the CCP by return cycle mapping is in that the return cycle isochrones equal to the VT cycle length converge on the lines of conduction block of the reentrant circuit. This is because the revolution time around the line of block after cessation of pacing is the cycle length of the tachycardia as long as an excitable gap exists along the lines of block. The present study also showed that the potentials from the CCP were not necessarily required to have the return cycle isochrones converge on the lines of block, and that the mapping accuracy did not change with or without analysis of the potentials from the CCP. The reason for this is that the return cycle isochrones equal to the VT cycle length radiate from the CCP. In addition, as shown in Figs 5 to 7, the return cycle isochrone was inclined to converge at the line of block nearest the entrance of the CCP. The mechanism for this is illustrated in Fig 9. As the
Figure 9. Mechanism for converging the return cycle isochrones equal to the ventricular tachycardia (VT) cycle length on the lines of functional block. Schematic illustrates the shift of the return cycle isochrones equal to the VT cycle length corresponding to each stimulation site (A and B). Each arrow indicates the shift of the isochrone for each stimulation site. CCP indicates central common pathway; N, the region activated by the last stimulus; and N-1, the region activated by the preceding stimulus.

stimulation site shifts from site A to site B, the collision region of the antidromic and orthodromic wave fronts shifts, the return cycle isochrone equal to the VT cycle length also shifts. The degree of shift of the return cycle isochrones depends on the conduction time difference from each stimulation site to the reentry circuit and to the collision region. The presence of slow conduction at the edge of the lines of block and in the CCP allows the collision region to rotate slowly at the region around the end of the lines as the stimulation site shifts. As a result, the degree of shift of the return cycle isochrone is more gradual at the region close to the reentrant circuit than at the region distant from the reentrant circuit. This allows the return cycle isochrones converge at the lines of block nearest the entrance of the CCP even without the potentials from the pathway.

Limitations
Localization of the CCP by entrainment mapping is based on the assumption that the reentrant circuit has a fully excitable gap that allows a pacing impulse to reset the tachycardia without decremental conduction in the circuit. Almendral and colleagues and Gottlieb et al demonstrated, in human ischemic VT using the resetting response pattern, that there were some patients whose VT showed an increasing pattern in the resetting response that suggested decremental conduction in the reentrant circuit or in the region between the stimulation site and the reentrant circuit. Decremental conduction in the reentrant circuit as a response to rapid pacing may impair the accuracy of the entrainment mapping technique. The return cycle mapping shares the same limitation. In this study, we examined the return cycle distribution after entrainment at a paced cycle length close to the VT cycle length, therefore the decremental conduction was not significant. Entrainment at a shorter pacing cycle length can cause a change in the location and shape of functional block or result in its acceleration or termination. Therefore pacing should be performed with a cycle length long enough to capture all the myocardium and avoid decremental conduction in the reentrant circuit in this mapping technique.

In the present study, the reentrant circuit of the induced VT was located in the thin epicardial tissue overlying a subendocardial infarct, so that it was only necessary to map the epicardium. However, intramural reentry may be the mechanism of the VT in patients. Although this mapping technique does not require the potentials from the CCP to localize the pathway and could be easily extended for three-dimensional return cycle mapping, further studies are necessary to determine whether a CCP located intramurally can be also localized by interpreting return cycles mapped endocardially or epicardially.

Clinical Implications
This new mapping technique can be easily applied intraoperatively during surgery for VT. Determination of the activation times and calculation of the return cycle can be performed using currently available mapping systems. Our mapping system takes <3 minutes to display the return cycle map for 253 electrode locations. To obtain the intersections of the return cycle isochrones equal to the VT cycle length, VT should be entrained from more than two different ventricular sites. As shown in the present study, the pattern of the return cycle distribution depended on the spatial correlation between the stimulation site and the CCP. Entrainment from sites outside and distant from the CCP may demonstrate an expedient distribution of the return cycle to localize the CCP, because the antidromic and orthodromic wave fronts collide outside the CCP. In consequence, the return cycle isochrones equal to the VT cycle length also distribute outside the CCP. When the diastolic potentials are recorded from the CCP, the return cycle isochrones equal to the VT cycle length intersect at two regions that coincide with the ends of the lines of block nearest the entrance of the CCP. The distance between the two intersections gives the width of the pathway; thus ablation of the tissue between the intersections would interrupt the CCP completely. When no potentials are recorded from the CCP, the return cycle isochrones equal to the VT cycle length intersect at a single region at the entrance of the CCP. Ablation between the earliest activation site and the intersection of the return cycle isochrones would interrupt the CCP. Demonstration of a return cycle equal or close to the VT cycle length at most areas except for the region around the stimulation site suggests that the VT is entrained from inside or close to the CCP. The location of the return cycle isochrone equal to the VT cycle length can be ambiguous, because the isochrone forms a small circle in the CCP and the difference in return cycles between the neighboring electrode positions is small. The stimulation site should be changed until the return cycle map demonstrates the proper distribution pattern as described above.

Application of this mapping technique for catheter mapping requires simultaneous recording of multipoint potentials. Recently, a basket-shaped mapping catheter carrying 64 electrodes was developed, tested, and applied in patients with recurrent sustained VT. There is a limit to the number of electrodes that a catheter can carry. In the present study, we estimated the sufficient interelectrode distance for catheter ablation guided by this mapping method. The size of the lesion created by radiofrequency energy is determined by the
power and exposure duration of the energy and can be large as 8 mm in diameter.18 If mapping error is less than half of the diameter of the ablation lesion, the delivered energy can terminate the tachycardia. According to the correlation between the interelectrode distance and the mapping accuracy shown in Fig 8, the required interelectrode distance is estimated to be 8.5 mm for the radiofrequency energy to terminate VT guided by return cycle mapping. The basket-shaped catheter described above provides enough mapping resolution. Specifically, the whole endocardial surface of the multichannel catheter mapping technique with return cycle mapping. A radiofrequency catheter then will be directed to the mapped site to ablate the VT, or the mapping electrodes will be replaced with a different type of multielectrode catheter to localize the CCP more precisely. Combining the multichannel catheter mapping technique with return cycle mapping would provide a rapid and systematic means of localizing the CCP during catheter ablation of VT.

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References
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