Endocardial catheter mapping: wire skeleton technique for representation of computed arrhythmogenic sites compared with intraoperative mapping

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ABSTRACT Guiding surgical therapy of ventricular tachycardia by preoperative endocardial catheter mapping necessitates improvement of the accuracy of localization of the arrhythmogenic site. We therefore used a new mathematical cineradiographic method during catheter mapping to compute the position of left ventricular arrhythmogenic sites relative to three anatomic reference points: the centers of aortic and mitral valve ostia and the left ventricular apex. To enable the surgeon to identify the position of the computed sites, a wire skeleton (one for each patient) representing a single or multiple arrhythmogenic site(s) relative to the anatomic reference points was constructed. This wire skeleton was inserted into the left ventricular cavity during surgery. Side branches of the device indicated preoperatively localized arrhythmogenic sites. Results in eight consecutive patients were compared with those of intraoperative simultaneous mapping of 64 endocardial sites. Sixteen morphologically distinct monomorphic ventricular tachycardias were mapped by catheter and 15 by intraoperative mapping. In 12 ventricular tachycardias an identical morphology was recorded during both techniques. The distance between arrhythmogenic sites localized with both methods was 1 cm or less in 11 of these 12 ventricular tachycardias and 2 cm in one ventricular tachycardia. These results indicate that (1) endocardial catheter mapping combined with wire skeleton representation of computed positions of arrhythmogenic sites is reliable for guiding surgical therapy of ventricular tachycardia and (2) since some of the ventricular tachycardias were inducible only during either preoperative or intraoperative mapping, both techniques have an additive value. In addition, the wire skeleton proved convenient during surgery by identifying the arrhythmogenic sites.

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INTRAOPERATIVE endocardial activation mapping during ventricular tachycardia is an established technique to localize arrhythmogenic sites (origin of ventricular tachycardia) before their surgical excision, particularly in patients with chronic ischemic heart disease.1-4 Although this technique can by very accurate, its use may be hampered by various important limitations described in previous reports.2 4-9 Additional information can be obtained from preoperative endocardial catheter mapping of electrical activation during ventricular tachycardia.5 6 10 The arrhythmogenic site may be indicated correctly by the position of a catheter electrode with accepted electrophysiologic criteria (electrophysiologic identification). However, if this information is to be used subsequently for guiding surgical excision, inaccuracy is introduced by the fact that the electrode position at the arrhythmogenic site has to be estimated from multiple fluoroscopic projections (anatomic localization). This is especially true since there are few anatomically identifiable landmarks.

In a recent study we reported a mathematical anatomic localization procedure and its validation in an experimental animal preparation.9 Accurate localiza-
tion of various left ventricular sites was confirmed. We also described a wire skeleton localization technique to identify the computed localizations during surgery. In this report we present the validation of this new technique in patients with ventricular tachycardia by comparing the results with those obtained by intraoperative mapping.

**Methods**

Mapping guided surgical excision of arrhythmogenic sites was performed in eight consecutive patients with drug-refractory ventricular tachycardia related to chronic ischemic heart disease. The arrhythmogenic sites were localized by preoperative catheter mapping as well as by intraoperative endocardial mapping.

**Endocardial catheter mapping.** Three electrode catheters were used to perform endocardial catheter mapping. A No. 6F quadripolar USCI catheter with interelectrode distances of 0.5 cm and a No. 6F tripolar USCI catheter with interelectrode distances of 1 cm were introduced into the femoral vein and advanced under fluoroscopic guidance to the right ventricular apex and the His bundle area, respectively. The distal pair of electrodes of the right ventricular apex catheter was used for programmed electrical stimulation and the proximal pair for recording a bipolar reference signal. Another No. 6F quadripolar USCI catheter (interelectrode distance 0.5 cm) was introduced into the femoral artery and advanced to the left ventricular cavity. This mapping catheter was moved along the left ventricular walls to obtain local electrical activation recordings from various sites. Recording of two bipolar electrograms derived from the distal and proximal pairs of electrodes could be obtained simultaneously with the four unipolar electrograms from these electrodes.

Surface electrocardiograms and intracavitary electrograms were recorded directly on paper with a Siemens eight-channel ink-jet recorder and stored on magnetic tape with a Honeywell recorder. Paper speed was 50 to 250 mm/sec. Two different channel programs could be applied during catheter mapping: (1) surface standard electrocardiographic leads I, II, III, and V1 and intracavitary bipolar electrograms derived from the His bundle area, the right ventricular apex, and the two electrode pairs of the left ventricular mapping catheter and (2) the surface standard electrocardiographic lead I, bipolar electrograms derived from the right ventricular apex and the two electrode pairs of the left ventricular mapping catheter, as well as the four unipolar electrograms derived from this catheter. These programs were rapidly interchangeable with a pushbutton. Filters were set at 50 to 1000 Hz for bipolar and at 0.1 to 1000 Hz for unipolar recordings.

Programmed electrical stimulation with 2 msec pulse width and at twice diastolic threshold current was performed with a Janssen programmable stimulator. Sustained monomorphic ventricular tachycardia was induced with one or two premature ventricular stimuli during sinus rhythm or ventricular pacing. We tried to identify the arrhythmogenic site of each morphologically distinct ventricular tachycardia, although special attention was paid to ventricular tachycardia morphologies similar to those occurring spontaneously. Ventricular tachycardias were defined as morphologically different (pleomorphism) if one or more of the following criteria were present: (1) different mean polarity in V1 (right or left bundle branch block morphology); (2) difference in the frontal QRS axis of 90 degrees or more; (3) remarkable difference in QRS configuration in at least one of the leads I, II, III, and V1 combined with a reproducible stable and for each tachycardia different cycle length. When the patient was hemodynamically intolerant to the induced ventricular tachycardia because of a fast ventricular rate, procainamide 300 to 800 mg (50 mg/min) followed by a 5 mg/min infusion was given intravenously.

The following criteria were used for electrophysiologic identification of the arrhythmogenic site: (1) during extensive activation mapping of ventricular tachycardia, reproducible recording of the earliest obtained local distinct diastolic potential (index potential) in each consecutive cardiac cycle; (2) a fixed relationship of this index potential to the subsequent QRS complex of ventricular tachycardia, during spontaneous or pacing-induced change of the interval between consecutive index potentials (figure 1, A); (3) absence of the index potential during normal sinus rhythm and the sudden appearance of this potential preceding the first QRS complex of a morphologically distinct ventricular tachycardia; (4) sudden disappearance or a change in morphology of the index potential after the last QRS complex of this ventricular tachycardia configuration (figure 1, B). The first criterion together with at least one of the other criteria were considered obligatory to define the arrhythmogenic site. When an initial negative deflection of the diastolic potential in the unipolar electrogram during ventricular tachycardia or an identical QRS morphology during ventricular tachycardia and during pacing at the presumed arrhythmogenic site were recorded, these were considered as additional criteria only.

**Mathematical anatomic localization of arrhythmogenic sites.** A mathematical cineradiographic method for anatomic localization of any left ventricular site in terms of cylinder coordinates has been described extensively in a previous article. This method was used in the present study to compute the electrophysiologically identified arrhythmogenic site or sites relative to three anatomic reference points: the center of aortic and mitral valve ostia and the left ventricular apex. Anatomic localization was derived from spatial localizations of the arrhythmogenic site and the anatomic reference points relative to the same origin of a cartesian (x,y,z) coordinate system.

Instead of biplane frontal and lateral x-ray projections (animal study), 45 degree right (RAO) and 45 degree left anterior oblique (LAO) projections were used to improve localization of the mitral valve ostium center and to avoid overprojections of the arms. A rotating x-ray system was used for sequential projections. A monoplane radiolucent frame was attached to the x-ray focus, and calibration was performed by inserting small radiopaque pegs into the frame at distances of multiples of 1 cm. In addition, these pegs were used to define x and y axes in the RAO and a z axis in the LAO projection. The origin of the coordinate system was defined by the point of intersection of the three axes.

The catheterization table was kept in the same position relative to the x-ray system in both projections during the entire mapping procedure and subsequent left ventricular cineangiography. At the beginning of the mapping, the right ventricular apex catheter, the His catheter, the patient’s diaphragm, and part of the cardiac silhouette were depicted in both oblique projections on the fluoroscopic screen, at a similar level of inspiration. These data were used as references to ensure that the heart was kept in the same position relative to the x-ray system during consecutive localization steps. Whenever one of the mapping catheter-electrodes was positioned at an electrophysiologically identified arrhythmogenic site, cineradiograms were generated in both oblique projections. At the end of the entire procedure, the mapping catheter was removed and a No. 8F NIH catheter was advanced to the left ventricle to perform cineangiography (Isoopaque Coronar 42 ml, flow rate 14 ml/sec) in order to obtain images of the anatomic reference points in the same oblique projections.
FIGURE 1. For legend see opposite page.
FIGURE 2. A, Cylinder coordinates for anatomic localization of the arrhythmogenic site (AS). A left ventricular quadripolar catheter pointing to an arrhythmogenic site is shown. The line connecting the left ventricular apex (LVA) and the center of the aortic valve ostium (CA) constitutes the cylinder axis. The cylinder coordinates are (1) the distance $z$ between LVA and the projection of AS on the cylinder axis, (2) the distance $\rho$ between AS and this axis, (3) the angle $\phi$ between two planes, of which A is determined by the cylinder axis and AS and B by this axis and the center of the mitral valve ostium (CM). When looking from the apex to the base of the heart, $\phi$ is defined as positive when plane A is rotated clockwise with respect to plane B and as negative when plane A is rotated counterclockwise. B, Wire skeleton representation of the anatomic location of AS, based on cylinder coordinates. The long axis corresponds to the cylinder axis. The ring corresponds to the aortic valve ostial contour. The angle between the long axis and the mitral side branch (MSB) is 30 degrees. The tip of the other side branch corresponds to AS. This side branch with the length $\rho$ is constructed perpendicularly to the long axis at a distance $z$ from the point corresponding to LVA. The direction of this side branch is determined by the angle $\phi$. The mitral side branch can be shifted into the long axis (arrows). C, After left ventricular incision, the wire skeleton is inserted into the left ventricular cavity in such a way that the long axis connects the apex and the center of the aortic valve ostium and the mitral side branch points to the center of the mitral valve. The other side branch then points to AS.

FIGURE 1. A, Recordings of a ventricular tachycardia during catheter mapping. Shown are the electrocardiographic lead V1, the bipolar His bundle electrogram (HBE), the bipolar right ventricular apex electrogram (RVA), and two bipolar electrograms (LV1, 2 and LV3, 4) derived from the distal and proximal pairs of electrodes of the left ventricular mapping catheter, which is positioned at the presumed arrhythmogenic site. Multiple distinct diastolic potentials are recorded in LV1, 2 and LV3, 4. The most prominent diastolic potential $V$ in LV3, 4 is recorded 85 msec in front of the RVA reference signal, or 75 msec before the onset of the QRS complex, and these intervals remain constant in spite of the irregularity induced by an extrastimulus (S). Although the most prominent very early diastolic potential in LV1, 2 has apparently a fixed relationship to the subsequent QRS complex, its importance for the perpetuation of the tachycardia is questionable because of the findings described in panel B. B, Termination of the same ventricular tachycardia as in panel A by an extrastimulus (S). The mapping catheter remained at the arrhythmogenic site. Note the diastolic potentials $V$ in LV1, 2 and LV3, 4. The extrastimulus is followed by a similar diastolic potential in LV1, 2. The diastolic potential in LV3, 4 disappears, coinciding with the termination of the tachycardia. This suggests a causal relationship between this potential and the perpetuation of the arrhythmia. The diastolic potential in LV1, 2 may be due to very early conduction at the arrhythmogenic site, which is concealed after the extrastimulus. However, conduction in a dead-end pathway, at the distal pair of electrodes, cannot be excluded. Also note the late fragmented simultaneous activity recorded during sinus rhythm in LV1, 2 and LV3, 4. The morphology of the potentials has been changed as well.
Measurements needed for the computations were performed off line from end-diastolic frames at the onset of the QRS complex of a simultaneously recorded electrocardiogram. Cylindrical coordinates of all arrhythmogenic sites and the three reference points were computed off line with a computer system (figure 2, A).

Wire skeleton. The coordinates of the arrhythmogenic and reference sites were used to construct a stainless-steel wire skeleton® consisting of a fixed long axis equal to the distance between the left ventricular apex and the center of the aortic valve ostium and flexible side branches, one pointing to the center of the mitral valve ostium (mitral side branch) and the other(s) to one or more arrhythmogenic sites (figure 2, B). The site corresponding to the center of the aortic valve ostium was constructed as the center of a tilting ring, with a diameter equal to that of the aortic valve ostium as calculated from the left ventricular cineangiogram. The flexible mitral side branch could be shifted into the long axis. The device could be constructed and sterilized within 2 hr.

Procedures during surgery. After cannulation for cardiopulmonary bypass during surgery for ventricular tachycardia, the infarcted area was incised during normothermic perfusion. The minimal length of this incision is equal to the diameter of the tilting aortic ring. Before insertion of the wire skeleton, the mitral side branch was withdrawn into the long axis. Subsequently, the wire skeleton was inserted into the left ventricular cavity so that the long axis connected the left ventricular apex and the center of the aortic valve ostium. The mitral side branch was pushed out carefully in the direction of the center of the mitral valve ostium. When positioned in this way, the other side branch(es) point to the preoperatively localized arrhythmogenic site(s) (figure 2, C) enabling the surgeon to identify these sites of interest.

Subsequently, intraoperative endocardial mapping of the left ventricle was performed with a previously described intracavitary balloon covered with 64 electrodes on its surface and connected to a computer system for simultaneous unipolar recording of electrical activation during ventricular tachycardia. The sequence of electrical activation for each induced morphologically distinct ventricular tachycardia was depicted in isochrones on an activation map. Morphologic interpretation was performed from electrocardiographic leads I, II, and III. The arrhythmogenic site was defined as the earliest site of endocardial activation. From the balloon position in the left ventricular cavity and the position of the balloon-electrode from which the earliest electrical activity during ventricular tachycardia was recorded, the localization of the arrhythmogenic area relative to surrounding anatomic structures was determined by the surgeon. Intraoperative mapping was followed by resection of all arrhythmogenic sites, irrespective of the method by which they were localized.

Preoperative endocardial catheter mapping combined with the wire skeleton localization technique and intraoperative endocardial mapping were compared by estimating the distance in centimeters between the arrhythmogenic sites localized with both methods. Comparison was performed only when identical ventricular tachycardia morphologies in the electrocardiographic leads I, II, and III were recorded during both procedures.

Results

Electrophysiologic identification of arrhythmogenic sites during endocardial catheter mapping. Catheter mapping was performed in eight consecutive patients with previously documented myocardial infarction (six anteroseptal, one [patient 6] inferior, and one [patient 4] in both the anteroseptal and inferior walls). Sixteen morphologically distinct ventricular tachycardias were induced and mapped. Before the mapping, nine of these ventricular tachycardia morphologies had occurred

<table>
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<th>Patient</th>
<th>Mapped VT</th>
<th>LV sites (n)</th>
<th>DA-QRS (msec)</th>
<th>z (cm)</th>
<th>ρ (cm)</th>
<th>Φ (degrees)</th>
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VT = ventricular tachycardia; LV sites = total number of reached left ventricular sites; DA-QRS = interval between index potential and onset of subsequent QRS complex; AO-LVA = distance between center of aortic valve ostium and left ventricular apex.
spontaneously and were documented as clinical arrhythmias. During each ventricular tachycardia, electro-
rical activity was recorded from 10 to 70 (mean 27) left ventricular endocardial sites and the arrhythmogenic site could be identified according to the abovementioned criteria. Diastolic activation derived from the arrhythmogenic site was recorded between 5 and 115 msec before the onset of the QRS complex during ventricular tachycardia. The results are summarized in table 1.

Mathematical anatomic localization. The anatomic localization of the arrhythmogenic sites was expressed in cylinder coordinates. Results are shown in table 1. The computed distance between the center of the aortic valve ostium and the left ventricular apex is also shown. As can be seen from their cylinder coordinates, arrhythmogenic sites were localized at various parts of the left ventricular endocardium.

Wire skeleton. For each patient a different wire skeleton was constructed representing arrhythmogenic sites and anatomic reference points. Insertion into the left ventricular cavity and positioning was quite easy and could be performed within 10 sec in each patient. The position in situ of the wire skeleton of patient 1 is shown in figure 3.

Comparison with intraoperative mapping. Fifteen morphologically distinct ventricular tachycardias were induced and mapped during surgery and six of these had been documented as clinical arrhythmias. In 12 of the 15 ventricular tachycardias, an identical morphology had been recorded during catheter mapping (table 2). The distance between arrhythmogenic sites localized with both techniques was 1 cm or less in 11 ventricular tachycardias and 2 cm in one ventricular tachycardia. Wire skeletons and intraoperative maps in patients 2 and 6 are shown in figures 4 to 7.

Discussion

Methodologic differences between experimental and clinical study. In our previous animal study we could demonstrate that the position of any left ventricular site can be accurately calculated and anatomically local-

![FIGURE 3. The exposed heart with the wire skeleton in situ (patient 1). The left ventricular wall is incised at the apex and a part of the correctly positioned wire skeleton is visible. The white arrow points to the apical end of the long axis. The black arrow and the white star indicate side branches pointing to the arrhythmogenic sites of two ventricular tachycardias, VT1 and VT2, with cylinder coordinates 1.7, 0.6, −159 degrees (VT1) and 1.9, 0.9, −46 degrees (VT2). These correspond to a septal and a lateral wall site near the apex, respectively. VT2 was not induced during intraoperative mapping. LAD = left anterior descending artery.]

<table>
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<th>Clinical VT</th>
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AS = arrhythmogenic site; VT = ventricular tachycardia; CM = catheter mapping; IM = intraoperative mapping.
FIGURE 4. The wire skeleton constructed for patient 2. The shiftable mitral side branch is clearly visible. The tip of the short side branch near the apical end of the long axis corresponds to the anterosetal origin of VT1 (cylinder coordinates 2.1, 2.7, -137 degrees). The tip of the long side branch (cylinder coordinates 4.5, 3.9, -97 degrees) corresponds to a potential arrhythmogenic area in the anterior wall where continuous electrical activity was recorded.

FIGURE 5. The sequence of electrical activation during ventricular tachycardia in isochrones on an activation map, based on computer-assisted, simultaneous intraoperative endocardial mapping. The endocardial surface is depicted as if the left ventricle were folded out after incising the ventricular wall along the left anterior descending artery, from base to apex. The surface is divided in three parts corresponding to the septal, the posterior, and lateral and anterior areas. Shown is the activation map of VT1 in patient 2. The earliest activation is recorded in a large anterosetal area near the apex. The star indicates the arrhythmogenic site pointed out by the short side branch of the wire skeleton shown in figure 4. APM = anterior papillary muscle; PPM = posterior papillary muscle.

FIGURE 6. Example of a complicated wire skeleton (patient 6, with inferior wall infarction). The side branches constructed to point to arrhythmogenic sites are pointing downward and somewhat to the left. Going from the aortic end of the long axis, the tip of the first of these side branches corresponds to the origin of VT2 (cylinder coordinates 5.7, 3.9, +58 degrees), the tip of the second to the origin of VT1 (cylinder coordinates 4.9, 3.9, +69 degrees), and the tips of the third and fourth branches (some millimeters apart only) to the origin of VT3 (cylinder coordinates fourth tip 3.7, 3.2, +51 degrees).

ized by means of the wire skeleton technique. The uncertainty of the calculated position in one direction was maximally 7 mm in normal beagle hearts.

Although mathematically the same method was used in the present study, its application was different in three ways: (1) The frontal and lateral x-ray projections used in the animal study were abandoned. Perpendicular 45 degree LAO and RAO projections are superior to identify the center of the mitral valve ostium and overprojection of the arms is avoided. (2) Since oblique projections could not be obtained simultaneouely in our institution, a sequential procedure was used in this study. (3) Cineradiography for localization of markers simulating arrhythmogenic sites in the experimental study was immediately followed by cineangiography for identification of anatomic reference points. However, in the practice of catheter mapping, much time elapses between cineradiograms of electrode positions at various arrhythmogenic sites and cineangiography at the end of the study. Evidently, the accuracy of our method could have been influenced by these three changes. Better identification of the mitral reference point might have led to more accurate anatomic localization, whereas the increase in the number of and a longer delay between consecutive localization steps might have had an adverse effect. To minimize this problem, the heart was kept in the same position relative to the x-ray system during the different steps and measurements were always performed from end-diastolic frames at the onset of the QRS complex.

The left ventricular apex was chosen as one of the anatomic reference points. However, whereas the hearts in the experimental study were normal, the hearts of the patients in this study were extensively damaged by myocardial infarctions. Since the apical area was always involved, resulting in apical aneurysms in most of the patients, the most inferior part of the left ventricular apex was taken as the reference point to minimize the risk of identifying different sites in both oblique projections. Nevertheless, accurate identification of the left ventricular apex can be difficult in patients with apical aneurysms.

The wire skeleton proved convenient during surgery, including those with more than one side branch.
as in patients 5 and 6. Making the mitral side branch shiftable into the long axis of the device was clearly an improvement as compared to the flexible but fixed construction used in the animal study.9 Positioning proved easier and hence minimized the chance of damaging the chordae.

Comparison with intraoperative mapping. Although during catheter mapping 16 distinct ventricular tachycardia morphologies were mapped as opposed to 15 during intraoperative mapping, a similar configuration was recorded in only 12 of these. The actual number of these pre- and intraoperatively induced identical ventricular tachycardias might have been lower, since only the electrocardiographic leads I, II, and III were available for comparison. Differences might have been obscured by the absence of more leads and especially of lead V1 during surgery, i.e., absence of the preoperatively used V1 polarity criterion for morphologic difference (see Methods). Nevertheless, the surgeon identified in most instances the same location as the arrhythmic site when both techniques were compared in these 12 ventricular tachycardias. The maximal distance between comparable arrhythmic sites was 1 cm in 11 ventricular tachycardias and 2 cm in one. This suggests that the accuracy of the localization in patients is similar to that described in the experimental study, in spite of the limitations described above.

Clinical implications. Endocardial catheter mapping combined with the wire skeleton localization technique enables the surgeon to identify correctly and easily left ventricular arrhythmogenic sites. The wire skeleton technique can localize the arrhythmogenic site within an area of 3 cm² and this compares favorably with the 4 to 8 cm² when estimation from multiple fluoroscopic projections is used.5 Surgeons often resect more than the areas of localized origins of ventricular tachycardia in patients with myocardial infarction in an effort to excise all potential arrhythmogenic sites.12,13 Therefore, one may question the need for so precise a method to define the site of origin of ventricular tachycardia. However, since the arrhythmogenic site is mostly localized at the border zone between infarcted and normal tissue,1,14 the more accurate the localization the more viable myocardium may be spared. Accurate localization is even more important in areas near or at the papillary muscles. Both problems were encountered in patient 1. The side branch of the wire skeleton corresponding to the arrhythmogenic site of the clinical VT3 (not induced during surgery) in this patient pointed to apparently normal myocardium at the base of the posterior papillary muscle. Only after endocardial resection guided by the results of catheter mapping did small fibrotic offshoots of the infarction become visible at this area. The papillary muscle would have been unnecessarily threatened when catheter mapping results were used without the wire skeleton.

Catheter mapping combined with wire skeleton localization and intraoperative mapping should be considered complementary techniques. This is clearly shown in table 2. Catheter mapping was superior in patients 1 and 5, since arrhythmogenic sites of spontaneously occurring clinical ventricular tachycardias (VT3 in patient 1 and VT1 and VT3 in patient 5) were localized with this technique only. The origin of VT1 in patient 1 was localized at the lateral base of the posteri-
or papillary muscle, whereas the nonclinical VT1 originated from the septum and thus from a different site (figure 3). Without catheter mapping, only this septal site would have been excised. A similar problem was encountered in patient 5. VT1, VT2, and VT3 originated from different sites, whereas VT4 had presumably the same origin as VT1 (see cylinder coordinates in table 1). Only the arrhythmogenic site of the nonclinical VT2 would have been localized if intraoperative mapping alone had been used. However, in patients 2 and 6 more arrhythmogenic sites were localized with intraoperative mapping. None of these sites was localized preoperatively during mapping of different tachycardia morphologies. Therefore, at this time both techniques should be used in each patient.

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