Ventricular Tachycardia After In Vivo DC Shock Ablation in Dogs
Electrophysiologic and Histologic Correlation

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Background. DC shock catheter ablation for the treatment of ventricular tachycardia (VT) may induce VT episodes that disappear within days.

Methods and Results. A 30-J cathodal shock was delivered to the endocardial left ventricular wall in 15 closed-chest dogs. All dogs had VT during the first day after ablation. Eleven of these dogs were studied on the first day. Extensive epicardial and endocardial activation mapping in vivo, in Langendorff-perfused hearts, and in tissue blocks in a tissue bath localized the site of origin of VT to subendocardial Purkinje fibers in a border zone surrounding the central necrotic ablation lesion. Intracellular recording showed that this zone consisted of a subendocardial superficial layer (SSL) of cells with abnormal characteristics, a resting membrane potential (RMP) of −58±11 mV (mean±SD), and an action potential amplitude (APA) of 61±20 mV. In addition, the steepness of phase 0 of the action potential was markedly reduced. In three dogs abnormal automaticity was found in a very small area. Immediately below the SSL, cells were normal with an RMP of −78±5 mV and an APA of 107±8 mV. Histology confirmed a thin SSL with edematous and necrotic cells, hemorrhage, and infiltration. The other four dogs were studied at 1 week after ablation when VT was absent. Microelectrode impalement in the SSL was either impossible or showed nearly normal action potential characteristics. Histologic examination showed a markedly thickened fibrotic subendocardial layer at places where impalement was impossible. Normal subendocardium was found in other areas of the border zone.

Conclusions. Our results indicate that VT after DC shock ablation originates from cells with abnormal automaticity in the superficial subendocardial border zone around the central ablation lesion. Within 1 week edematous and necrotic cells in this border zone are replaced by a fibrotic layer, and this transition is associated with the disappearance of VT. (Circulation 1991;84:267-278)

Clinical studies have shown that DC shock catheter ablation for the treatment of ventricular tachycardia may occasionally be complicated by ventricular tachyarrhythmias.1-6 This proarrhythmic effect is very common in dogs.7-10 In a previous report we described the high incidence of episodes of monomorphic ventricular tachycardia after a single 30–250-J shock delivered to the canine ventricular endocardium.11 Arrhythmogenicity rapidly declined, however, since at 1 week after the shock arrhythmias either occurred very rarely and were brief, or were completely absent.

Unipolar electrograms derived from the site of shock delivery during postablation tachycardia indicate that the arrhythmias originate close to the ablation lesion, but careful activation mapping has never been performed.11 The mechanism of postablation tachycardia is not known either. Levine et al12 studied cellular electrophysiological changes induced by DC shock ablation and suggested the potential role of reentry and early afterdepolarizations. However, tachycardias did not occur in their in vitro model. In addition, some characteristics of the tachycardias, such as a similar appearance of all complexes of an episode including the first and last complexes com-
bined with a marked variability of the cycle length and coupling interval and the inability to induce these tachycardias by programmed electrical stimulation, are not in favor of a reentrant mechanism.\textsuperscript{11}

The present series of experiments were performed to determine the site of origin and the mechanism of ventricular tachycardia after in vivo DC shock ablation. The findings were related to observations on the histological substrate. Mapping of electrical activity during tachycardia was performed in vivo, in Langendorff-perfused hearts, and in excised superfused tissue blocks in a tissue bath. Intracellular recording and histological examination were performed at either day 1 after the shock or at day 7, when ventricular tachycardia was absent, to elucidate time-dependent changes.

Methods

Experimental Model

Closed-chest DC shock ablation was performed in 15 beagles weighing 13–18 kg. The dogs were anesthetized with methadone and droperidol, as previously described.\textsuperscript{10} Under sterile conditions, a new standard 6F USCI bipolar electrode catheter (inter-electrode distance 1 cm) was introduced into the left femoral artery and advanced to the left ventricular cavity under fluoroscopic guidance. The electrode catheter tip (distal electrode, surface area 11 mm\textsuperscript{2}) was positioned against the inferoposterior left ventricular wall. In each dog a single 30-J cathodal shock was delivered to the endocardium between the distal catheter electrode and an anodal external chest paddle. Details of the technique have been described previously.\textsuperscript{10}

During the first hour after the shock, the heart rhythm was monitored in the electrophysiology laboratory. In each dog, episodes of monomorphic nonsustained and sustained ventricular tachycardia started spontaneously within 10 minutes after the shock. Endocardial catheter mapping was then performed in four dogs. The catheter was removed at the end of the monitoring period, and the dog was allowed to recover.

The dogs were anesthetized again; 11 dogs on day 1 (next day) and the other four dogs on day 7 after the shock. Eight of the 11 dogs studied on day 1 showed episodes of ventricular tachycardia. In the other three dogs, which had sinus rhythm at a high rate, electrical stimulation of the left vagal nerve unmasked episodes of ventricular tachycardia. Ventricular tachycardia was absent in the dogs studied on day 7. Thoracotomy was performed and the heart was excised in all dogs. Before excision, epicardial mapping of the in situ heart fixed in a pericardial cradle was performed in five dogs.

In the hearts of three dogs studied on day 1 endocardial mapping was carried out with the heart connected to a Langendorff perfusion setup.\textsuperscript{13}

Parts of the hearts of all 15 dogs were studied in a tissue bath. Tissue blocks with a volume of about 9 cm\textsuperscript{3} (length 3 cm, width 3 cm, depth 1 cm) enclosing the clearly visible ablation lesion were cut from the ventricular wall. From three hearts that were studied

\begin{table}[h]
\centering
\caption{Action Potential Characteristics in Border Zone}
\begin{tabular}{|l|c|c|c|}
\hline
 & \multicolumn{2}{c|}{Day 1 postablation} & \multicolumn{1}{c|}{Day 7 postablation} \\
 & Superficial cells & Deeper cells & Superficial cells \\
\hline
Action potential characteristics & \(n=75\) & \(n=75\) & \(n=97\) \\
\hline
RMP (mV) & \(-58\pm11\) & \(-78\pm5\) & \(-81\pm8\) \\
APA (mV) & \(61\pm20\) & \(107\pm8\) & \(105\pm18\) \\
APD 80\% (msec) & \(235\pm22\) & \(209\pm18\) & \(160\pm29\) \\
\hline
\end{tabular}
\textsuperset{Values are mean\pmSD. RMP, resting membrane potential; APA, action potential amplitude; APD, action potential duration.}
\end{table}
1 day after ablation, a tissue block of equal size was excised from the nonaffected lateral wall to serve as a control. From each tissue block a 2–4-mm-thick endocardial layer was peeled off with the help of a scalpel. The preparation was pinned to the base of the tissue bath with the endocardial surface facing upward. The tissue was superfused at a flow rate of 30 ml/min with oxygenated Tyrode’s solution at 37°C. The preparations were allowed to recover for half an hour before starting endocardial extracellular mapping and intracellular recordings.

Mapping Procedures During Ventricular Tachycardia

Endocardial catheter mapping was performed with the same electrode catheter used for ablation. Under fluoroscopic guidance this catheter was moved to sites within 2 cm from the site of shock delivery and to some sites at a greater distance. Recordings were obtained from about 15 sites in each case. Surface electrocardiographic leads I, II, and III, a bipolar electrogram, and the two unipolar electrograms derived from this catheter were recorded simultaneously on paper using a multichannel Siemens ink jet recorder (Erlangen, Germany). Filters were set at 50–1,000 Hz for bipolar and 0.15–1,000 Hz for unipolar recordings. Local activation times were related to the onset of the QRS complex.

Endocardial mapping in the Langendorff-perfused hearts was performed with a hand-held stick multielectrode with 64 electrode terminals mounted at its tip. Terminals were arranged in an 8×8 matrix with interelectrode distances of 1 mm. The probe was introduced into the left ventricular cavity via the mitral ostium and positioned at various sites around the ablation lesion. Four hook electrodes were attached to the heart to obtain bipolar ventricular reference and atrial electrograms. The two ventricular electrodes were epicardially inserted into the left and right ventricular walls. Unipolar electrograms were derived from the stick electrode and a computer system, which has been described previously, was used for simultaneous recording from 64 sites at a filter setting of 1–1,000 Hz.

The same system was used for epicardial mapping of the in situ heart. Epicardial mapping was performed with a patch covered with 64 unipolar electrode terminals arranged in an 8×8 matrix with interelectrode distances of 6 mm. Surface electrocardiographic leads I, II, and III were used as reference signals.

Endocardial mapping in the tissue bath was performed with the hand-held stick multielectrode described above. The signals from three semiunipolar electrodes positioned in a triangle around the ablation lesion were used as reference signals. The reference electrodes consisted of two Ag wires (diameter 0.2 mm), one positioned on the endocardial surface and the other clipped at 3 mm above the tissue. To reduce electrode resistance, both tips were electrolytically covered with an AgCl layer.

Intracellular Recordings

Intracellular recordings were derived from the border zone around the ablation lesion, using conventional microelectrode techniques. Recording sites were 1–5 mm apart. Additional impalements were done at sites where activation mapping suggested the site of origin of the tachycardia. For control purposes, several impalements were performed at sites more than 1 cm from the border of the ablation lesion.
Histological Examination

Histological examination was performed in 11 dogs (seven of the 11 dogs studied at day 1 and the four dogs studied at day 7). The excised tissue blocks were fixed in formalin after marking the preparation (small needle insertions) at sites of particular interest based on the electrophysiological findings. Multiple slices were cut perpendicular to the endocardial surface through the ablation lesion and surrounding tissue. Slices were stained with hematoxylin and eosin.

Results

All ectopic ventricular complexes showed a superior axis in each dog. Although the mean polarity was similar in each individual dog, the initial part of the QRS complex could be different, as shown in Figure 1, suggesting multiple sites of origin. However, during sustained or longer episodes of non-sustained ventricular tachycardia all complexes were usually very similar.
Endocardial Mapping

Endocardial catheter mapping at sites within 2 cm from the site of shock delivery invariably revealed a site where local ectopic activation started before the onset of the QRS complex (Figure 2). In addition, unipolar recording at that site showed an initially negative deflection, suggesting activation spreading away from the recording site. Fractionated activity, indicating inhomogeneity, could be recorded at multiple sites around the site of shock delivery. This is clearly illustrated by the bipolar recording of Figure 2. At greater distances from the site of shock delivery, fractionation disappeared and activation did not precede the onset of the QRS complex.

The endocardial ectopic activation patterns recorded from hearts connected to the Langendorff perfusion system showed a circumscribed area from which activation spread centrifugally, as illustrated in the upper panel of Figure 3. In one heart an activation pattern of Purkinje activity could be constructed as well. Signals recorded at the earliest activated endocardial site were always initially negative (tracings a, b, and c in Figure 3), which suggests recording at a site where activation arose. However, if Purkinje potentials were recorded at or near the earliest activated site, they preceded muscle activation (arrows in Figure 3). The earliest sites were always located in the border zone between healthy tissue and the ablation lesion (sites a–c in the upper panel of Figure 3).

Epicardial Mapping

Epicardial maps of ectopic activation showed a circumscribed area of epicardial breakthrough from which activation spread centrifugally, as shown in the upper panel of Figure 4. Ectopic complexes with different morphologies had different sites of breakthrough. In all dogs in which epicardial mapping was performed, earliest activation of the ventricular tachycardias occurred after the onset of the QRS complex (tracings of Figure 4). In addition, electrograms derived from the site of epicardial breakthrough always revealed an initially positive deflection, indicating that the depolarization front passed the electrode (electrogram marked with asterisk in Figure 4). These observations indicate that the origin of ventricular tachycardia is not located epicardially.
In Vitro Measurements

In all dogs studied 1 day after shock delivery, endocardial tissue blocks that enclosed the clearly visible ablation lesion (diameter 1–2 cm, Figure 5) showed spontaneous episodes of tachycardia after immersing the preparation in the tissue bath. Nine sustained episodes with a mean cycle length of 440 (range 200–760) msec were recorded. Control tissue blocks consisting of normal endocardium and subendocardium from the lateral ventricular wall or the epicardium, subepicardium, and intramural tissue from below the site of shock delivery (including deeper parts of the ablation lesion) were either electrophysiologically silent or showed a very low rate of spontaneous activation (Figure 6). Similar observations were obtained on day 7 while studying the endocardial and subendocardial tissue that included the ablation lesion.

Extracellular Recordings in Tissue Bath

Endocardial activation mapping during ventricular tachycardia was performed in all preparations studied on day 1. Morphologically different tachycardias originated from different sites within 1 cm from the border of the ablation lesion.

In most recordings, deflections of the specialized conduction system were found. Purkinje potentials preceded muscle deflections at the same site in all cases. Extensive mapping, with construction of both muscle and Purkinje activation maps, was possible in three cases with sustained tachycardia that remained stable with respect to morphology and cycle length. An example of both maps is shown in Figure 7. These maps were constructed from 512 signals recorded with the multielectrode positioned at eight adjacent areas. Sites of earliest activation show early Purkinje potentials and initially negative muscle deflections, suggesting the potential involvement of the Purkinje system in the mechanism of the tachycardia and the subendocardial origin of the arrhythmia.

Intracellular Recordings 1 Day After Shock Delivery

Microelectrode recordings were made at distances of 1–5 mm along the entire endocardial border of the ablation lesion. If possible, recordings were made from both a superficial and an adjacent deeper cell layer. Paired impalements were obtained from 75 positions. In all cases the superficial cells showed abnormal electrophysiological characteristics, which are summarized in Table 1. In contrast, deeper cells had normal or close-to-normal features. Figure 8 shows an example of action potentials obtained from the same site after consecutive impalements and withdrawals of the microelectrode. The superficial cells are clearly very abnormal, whereas the deeper cells have a normal appearance.

In some cases these abnormal subendocardial superficial cells marking the border zone were detected up to 1 cm from the macroscopically visible border of the central ablation lesion. However, at other sites in the same preparation the superficial layer was normal 2 mm from the border. This means that extension of the border zone may vary considerably at different sites. At several times during each experiment impalements were made at sites more than 1 cm from the border of the lesion. Intracellular recordings at these reference sites invariably showed close-to-normal characteristics for both the superficial and deeper layers.

Multiple impalements were performed at the site of earliest endocardial activation as determined by extracellular mapping during tachycardia. However, at the time of impalement a manifest tachycardia was frequently absent. In three cases a local tachycardia, confined to a few superficial cells tachycardia was frequently absent. In three cases a local tachycardia, confined to a few superficial cells tachycardia was frequently absent. In three cases a local tachycardia, confined to a few superficial cells tachycardia was frequently absent. In three cases a local tachycardia, confined to a few superficial cells tachycardia was frequently absent. In three cases a local tachycardia, confined to a few superficial cells tachycardia was frequently absent. In three cases a local tachycardia, confined to a few superficial cells tachycardia was frequently absent. In three cases a local tachycardia, confined to a few superficial cells tachycardia was frequently absent. In three cases a local tachycardia, confined to a few superficial cells.
Intracellular Recordings 7 Days After Shock Delivery

Impalement around the central ablation lesion was attempted in the same way as described for the day 1 experiments. In some areas of the border zone impalement failed frequently because of microelectrode fracture. In other areas, even areas very close to the border of the lesion, impalement was possible and normal resting membrane potentials and action potential amplitudes were obtained in the subendocardial superficial layer (97 impalements, Table 1). Within 1 mm from the border of the central lesion, however, action potential duration could be remarkably short due to shortening of phase 2.

Histology

At 1 day and at 7 days after the shock the site of shock delivery was always clearly visible as a well-demarcated but irregular pale area with an endocardial diameter of 1–2 cm (Figure 5). This area was often hemorrhagic and showed fibrin precipitation at its endocardial surface. The depth of the pale tissue reached about 1 cm, or more than half of the wall thickness. We refer to this area as the central ablation lesion. Histologically it consisted of necrotic muscle cells and hemorrhage at day 1, whereas at day 7 necrotic areas were embedded in relatively large amounts of granulation tissue.

The central ablation lesion was macroscopically surrounded by tissue that was not clearly different from tissue farther from the central lesion. However, at sites with electrophysiologically abnormal cellular characteristics at day 1, a histologically very thin (some cell layers thick) layer of abnormal tissue was found immediately below the endocardium. This superficial layer could be found up to 1 cm from the border of the central ablation lesion and consisted of necrotic and edematous cells, interstitial edema, hemorrhage, and infiltration. Myocardial cells below this thin layer were normal or close to normal (Figure 11).

At 7 days after the shock the area around the central ablation lesion showed either a normal subendocardium or a markedly fibrotic subendocardial superficial layer that could extend up to 1 cm from the border of the central lesion. Absence of this layer was a prerequisite for previous successful impalement.
Discussion

Experimental DC shock catheter ablation of the ventricular myocardium in dogs is usually complicated by ventricular tachyarrhythmias.\(^7\)\(^-\)\(^11\) Subsequent sudden death has been reported by several authors.\(^7\)\(^,\)\(^8\)

In a previous article we described the results of continuous postablation Holter monitoring up to 1 week after the shock.\(^11\) The time course of arrhythmogenicity could be divided into four phases. During the immediate postshock phase ventricular flutter occurred in some cases. This was followed by a "silent gap" without any tachyarrhythmia. The third phase started within 10 minutes after the shock and lasted about 3 or 4 days. This phase was characterized by a high incidence of episodes of sustained and nonsustained monomorphic ventricular tachycardia. Arrhythmogenicity markedly declined at the end of this phase. During the fourth and presumably chronic phase, tachycardia occurred very rarely or was completely absent.

In the present study, the site of origin and the mechanism of arrhythmias during the phase of monomorphic ventricular tachycardia were studied and related to the histological substrate. Findings were compared with those obtained during the fourth phase when tachycardia was absent.

Levine et al\(^12\) extensively studied the cellular electrophysiological changes induced by DC shock ablation in an in vitro model. Unfortunately, no tachycardia occurred in their epicardial preparations and the authors could only suggest potential mechanisms. This problem was avoided by our in vivo endocardial ablation. In addition, our method was very similar to clinical DC shock catheter ablation. We only used 30 J of delivered energy to obtain a relatively small ablation lesion (diameter maximally 2 cm). In our previous study\(^11\) the incidence and duration of episodes of ventricular tachycardia were not different when 30 or 250 J were delivered.

Episodes of spontaneous ventricular tachycardia remained during epicardial mapping of in situ hearts, in Langendorff-perfused hearts, and in the tissue bath. The tachycardia was unequivocally related to the ablation lesion because only the parts of the heart in the tissue bath enclosing this lesion (or the endocardial portion of the lesion) showed the arrhythmia, whereas other parts were either electrophysiologically silent or showed a low rate of electrical activity.

The site of origin was not subepicardially located because local activation never started before the onset of the QRS complex during epicardial mapping and because the initial deflection of the unipolar signal at the earliest epicardial site was always positive. In addition, parts of the ventricular wall enclosing the epicardium and subepicardial portions of the central ablation lesion did not show tachycardia in the tissue bath.

Endocardial mapping revealed that the earliest activation during tachycardia always preceded the onset of the QRS complex and was always derived from the border zone around the macroscopically visible central ablation lesion. The initially negative deflections of the unipolar signals derived from the earliest site of muscle activation support the subendocardial location of the site of origin. Recording of Purkinje potentials before the earliest muscle activation suggests that the tachycardia originates from subendocardial Purkinje fibers in the border zone.

![Figure 7. Endocardial extracellular mapping with multielectrode during sustained monomorphic ventricular tachycardia in tissue bath. Muscle and Purkinje activation maps shown in upper panel are constructed from 512 local activation times obtained with multielectrode at eight adjacent areas enclosing ablation lesion and its close surroundings. Local activation times are measured with respect to time 50 msec before intrinsic deflection of one reference signal. Isochrones are drawn at 5-msec intervals. Earliest muscle activation occurs within 1 cm from border of ablation lesion. However, comparison of muscle and Purkinje activation maps shows that Purkinje activity precedes muscle activation at all sites. Lower panel shows reference electrogram and four unipolar electrograms derived from sites a, b, c, and d indicated in upper panel. Muscle deflections show initial negativity but are preceded by Purkinje potentials (arrows).](http://circ.ahajournals.org/doi/10.1161/01.CIR.84.1.274)
Both cellular electrophysiological and histological examination 1 day after shock delivery showed that this normal-looking border zone consisted of a very thin subendocardial layer of abnormal cells. A 30-J shock, as used in our study, created a globular central ablation lesion with a radius and a depth of about 1 cm, which was surrounded by a disk-shaped subendocardial border zone with a depth of much less than 1 mm and a radial extension of from several millimeters to 1 cm. Endocardial mapping localized the site of origin of the tachycardia to within the area where the abnormal superficial subendocardial layer was found. At 1 week after the shock, when tachycardia was absent, the necrotic and edematous cells of this subendocardial layer were replaced by fibrosis. Thus, the mapping data combined with the cellular electrophysiological and histological findings strongly suggest that the site of origin of postablation ventricular tachycardia is located within the superficial subendocardial border zone. This is in accordance with the potential arrhythmogenic role of Purkinje cells since these cells are very abundant in the subendocardial layer.

Abnormal cellular electrophysiological characteristics have been recorded during the first day after the shock and were exclusively confined to the superficial subendocardial border zone. In accordance with the observations of Levine et al., we found a reduced resting membrane potential, a reduced action potential amplitude, and a reduced dV/dt of phase 0. At variance with the results of these authors, however, small areas were found with enhanced phase 4 depolarization, and this phenomenon was associated with the recording of a local tachycardia in three cases. The occurrence of the tachycardia was causatively related to enhanced phase 4 depolarization, and the arrhythmia originated from the impaled and possibly other surrounding cells because of 1) the recording of a smooth transition between phase 4 and phase 0 and 2) the association between cycle length changes and the steepness of phase 4 depolarization. Abnormal automaticity was considered to be the mechanism of the tachycardia since phase 4 depolarization started from a reduced resting membrane potential. The cycle length of the tachycardia varied considerably and showed gradual as well as sudden lengthening and shortening in the presence of con-
FIGURE 9. Recording of regular local ventricular tachycardia. Shown are extracellular bipolar electrogram derived from reference electrode 1 cm from border of central ablation lesion and simultaneous tracing of intracellular recording derived from cell in superficial subendocardial border zone within 2 mm from border of central ablation lesion. Cell shows depressed resting membrane potential (−62 mV) and phase 4 depolarization giving rise to regular tachycardia at cycle length of 400 msec. Action potential amplitude is about 56 mV. Smooth transition between phase 4 and upstroke of action potential suggests that cell is functioning as a pacemaker. Similar rapid spontaneous activity could be recorded in some other cells in an area of less than 1 mm². In contrast, extracellular reference electrogram shows very slow spontaneous activity, which, in this case, is dissociated completely from tachycardia. Similar slow dissociated activity was recorded at other remote sites. These findings demonstrate that small area with local tachycardia is surrounded by zone of exit as well as entrance block.

FIGURE 10. Recording of irregular local ventricular tachycardia in same preparation as in Figure 9. There was no spontaneous activity in extracellular reference electrogram. Intracellular activity was recorded from cell in same small area from which signal of Figure 9 was derived. Recording of phase 4 depolarization and local irregular ventricular tachycardia. Changes in cycle length were associated with local modulation of phase 4 depolarization.
tinuous entrance block to the small area of the local tachycardia, or even during the periodic absence of activation from other parts of the preparation. These observations are in accordance with an automatic mechanism.

The localization of the local tachycardia demonstrates the ultimate proof that the superficial subendocardial border zone is the histological substrate of monomorphic ventricular tachycardia during the third postablation phase. Absence of enhanced phase 4 depolarization as well as episodes of tachycardia in the study reported by Levine et al. can be explained by their use of epicardial preparations.

One may criticize our statement that all postablation episodes of monomorphic ventricular tachycardia originate in the subendocardial border zone and are based on abnormal automaticity since the actual site of origin and the mechanism of a local tachycardia could be elucidated at the cellular level in only three cases. However, it is very difficult to detect a small site (less than 1 mm²) with transient pacemaker activity using the conventional microelectrode technique. Although detection was guided by previous extracellular mapping of tachycardia episodes, subsequent episodes could originate from different sites all around the central ablation lesion. Thus, a localized site of origin may not show pacemaker activity at the time of impalement. In addition, a very small number of cells with this activity may easily be missed. In the three cases in which detection was possible, the site with a local tachycardia was surrounded by a zone of exit as well as entrance block. Since the previous manifest tachycardia (global activation of the whole preparation) originated from the area where afterward a local tachycardia was detected and since the local tachycardia showed a behavior very similar (length and variation of cycle length) to that of the manifest tachycardia, we believe that the two tachycardias are actually identical. Thus, manifestation of a tachycardia in the whole preparation depends on both the presence of enhanced phase 4 depolarization and the absence of exit block at the site of tachycardia origin. Both phenomena may occur transiently at multiple sites in the subendocardial border zone. In this way subsequent episodes may originate from different foci.

All postablation episodes of monomorphic ventricular tachycardia have very similar electrocardiographic characteristics, which have been described extensively in a previous article. All characteristics were in accordance with abnormal automaticity as the mechanism. In addition, tachycardia was not inducible with programmed electrical stimulation. Although we cannot exclude other potential mechanisms with certainty, supporting evidence is lacking. Thus, it seems very likely that abnormal automaticity is the most important, if not the only, mechanism of postablation monomorphic ventricular tachycardia. Since very abnormal action potentials were derived from cells with abnormal automaticity, we have no definite proof that these cells belong to the Purkinje system although their subendocardial location and the results of endocardial mapping strongly support that hypothesis.

Further research may be directed toward the use of our model for the study of abnormal automaticity and the influence of pharmacological interventions.

In conclusion, monomorphic ventricular tachycardia after DC shock ablation originates from cells with abnormal automaticity in the superficial subendocardial border zone around the central ablation lesion. Within 1 week edematous and necrotic cells in this border zone are replaced by a fibrotic layer.
and this transition is associated with the disappearance of tachycardia.

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References


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