Sustained Ventricular Tachycardia in Recent Canine Myocardial Infarction

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SUMMARY To study recurrent ventricular tachycardia in the late phase of myocardial infarction (MI), transmural anteropapical infarcts were created by ligation of the left anterior descending (LAD) coronary artery in 25 dogs. Twenty dogs survived LAD ligation and underwent an open-chest electrophysiologic study an average of 20 days after MI. Programmed electrical stimulation was carried out using the extrastimulus technique and short bursts of rapid ventricular pacing via bipolar electrodes positioned at multiple left ventricular endocardial sites. Sixteen dogs had electrically induced ventricular tachycardia, and in 11, sustained ventricular tachycardia was reproducibly initiated and terminated by programmed ventricular stimulation. Short bursts of rapid left ventricular pacing from areas in perifarct zone was the most effective technique for initiating ventricular tachycardia. The electrophysiologic phenomena in this model of sustained ventricular tachycardia in 3-week-old MI included electrically induced changes in rate and morphology and biventricular capture without termination during tachycardia.

VENTRICULAR ARRHYTHMIAS during acute myocardial ischemia and within the first week of experimental canine myocardial infarction have been studied by Harris et al.,1 El-Sherif et al.2-6 and other investigators.7,8 However, the electrophysiologic mechanisms that underlie ventricular arrhythmias in later stages of myocardial infarction have not been as extensively investigated because of the lack of an experimental model of reproducible, sustained ventricular tachycardia. In this report we describe a model of nonsustained, electrically inducible, sustained ventricular tachycardia in dogs with 3-week-old anteropapical myocardial infarctions. The stability, reproducibility and relatively high yield of inducible sustained ventricular tachycardia make this model promising for further investigation of the electrophysiologic properties of sustained ventricular tachycardia during the late phase of myocardial infarction.

Methods

Experimental Myocardial Infarction

Twenty-five mongrel dogs that weighed 15-25 kg were anesthetized with i.v. pentobarbital (15 mg/kg) and i.v. methohexital (5 mg/kg). After induction of anesthesia, each dog was mechanically ventilated with 100% oxygen. Surgery was performed using a sterile technique. Intravenous boluses of methohexital, 25 mg, were given approximately every 10 minutes during the course of surgery. The heart was approached via a left lateral thoracotomy and the anterior wall and apex of the left ventricle were exposed. The left anterior descending coronary artery was ligated immediately distal to the first diagonal branch. All visible epicardial branches in the left ventricular apical area originating from the left circumflex or posterior descending coronary arteries were also ligated. The pericardium was closed loosely. Ventricular ectopy that developed immediately after left anterior descending coronary artery ligation was treated with i.v. lidocaine (2 mg/kg). The thoracotomy was closed with a chest tube in place and each dog was extubated. Each dog received periodic injections of meperidine (1.5 mg/kg) for analgesia during the recovery period.

Electrophysiologic Study

Electrophysiologic studies were performed an average of 20 ± 4 days after the creation of experimental myocardial infarction. The postmyocardial infarction dogs were anesthetized with pentobarbital (30 mg/kg) and ventilated mechanically through an endotracheal tube with 100% oxygen. The heart was approached via a median sternotomy. Atrial pacing wires were sutured onto the appendage of the right atrium. The infarcted area of anteropapical left ventricle was identified visually as a discolored and akinetic region of scar tissue. Teflon-coated stainless-steel bipolar plunge wires were used to record electrical activity and to stimulate the endocardium of multiple sites.

One pair of bipolar plunge wires was positioned in the free wall of the right ventricle and another in the right ventricular outflow tract. Two pairs of bipolar plunge wires were positioned in each of the following normally contracting left ventricular areas: posterobasal region, inferior wall and lateral wall. Three or four pairs of bipolar plunge wires were positioned 2 cm apart centrally in the visually identified akinetic anteropapical region. At postmortem examination, this region uniformly showed transmural infarction at its center. These wires are subsequently referred to as electrodes within the infarct zone. Six to eight pairs of...
plunge wires were positioned circumferentially around the infarct approximately 2 cm apart.

Postmortem findings in these areas revealed mixed histologic findings with areas of various degrees of subendocardial infarction as well as normal myocardium. The wires in these areas are subsequently referred to as electrodes in the border zone of the infarct or perinfarction zone. In addition, a pair of bipolar plunge wires was used to record a His bundle electrogram in eight dogs. A reliable His bundle recording was obtained during ventricular tachycardia in four. The plunge wires were connected to a switch box, which was connected to a programmable electronic stimulator (W.P. Instruments, Inc.) as well as to a multichannel recorder and oscilloscope (Electronics for Medicine VR-12). A surface ECG, the systemic arterial pressure and the central venous pressure were recorded simultaneously with local electrograms from multiple ventricular sites described above. Electrophysiologic data were stored for later retrieval and analysis on FM magnetic tape (A.R. Vetter, Inc.).

The effective refractory periods of the right ventricular endocardium and of multiple endocardial sites from the normal and the infarcted areas of the left ventricle were determined by the introduction of progressively premature ventricular stimuli after every six ventricular paced beats at a fixed cycle length of 500 msec (ventricular extrastimulus technique). The stimulus strength was maintained at twice diastolic threshold.

Ventricular pacing followed by double premature stimuli as well as short (three to five beats) bursts of rapid ventricular pacing (cycle length 250 msec to 150 msec) from multiple ventricular sites were used to initiate ventricular tachycardia. The following protocol for programmed ventricular stimulation was used in each dog. Programmed double premature ventricular stimuli were applied during fixed rate ventricular pacing at cycle lengths of 500 msec and 400 msec. This technique was applied first using the bipolar plunge wires in the right ventricular free wall, subsequently using each of the bipolar plunge wires in the normal left ventricular myocardium, and finally using each of the plunge wires within and at the periphery of the infarct zone. This was followed by application of brief bursts of rapid ventricular pacing via each pair of bipolar endocardial wires in the same order described above. Thus, in each dog, programmed ventricular stimulation was performed from every bipolar electrode in the order described above, except the His bundle recording electrode.

Premature ventricular stimulation and rapid ventricular pacing were used to terminate the induced ventricular tachycardias. When ventricular fibrillation occurred, it was terminated by direct current countershock (15-20 J) applied to the epicardium using a Hewlett-Packard 7802B defibrillator.

Seven dogs received i.v. verapamil in an average dose of 3.8 ± 1.9 mg (range 1.5-10 mg) after completion of the initial electrophysiologic study. Fifteen minutes after verapamil, programmed ventricular stimulation was repeated according to the protocol described above.

Arterial blood gases and the serum sodium and potassium concentrations were monitored frequently and were maintained within the physiologic range throughout the experiments.

Pathology

After completion of the electrophysiologic study, the dogs were sacrificed and the hearts were preserved in 10% formalin. Gross and histopathologic studies were performed. The percent of infarcted myocardium was estimated from serial coronal sections of the left ventricle. Each slice was photographed and the size of the infarcted tissue was estimated by the planimetry method. Histologic samples, prepared with hematoxylin-eosin stain, confirmed the presence of infarcted and normal tissue in each coronal section.

Results

Twenty of the 25 dogs survived left anterior descending coronary artery ligation without early or late postoperative complications and underwent an open-chest electrophysiologic study an average of 20 ± 4 days after myocardial infarction (range 5–28 days, table 1). All dogs were hemodynamically stable at the time of the electrophysiologic study. The mean arterial pressure at the initiation of electrophysiologic studies was 94 ± 9 mm Hg, and the mean central venous pressure was 5 ± 2 mm Hg. One dog had stable accelerated idioventricular rhythm at 95 beats/min. All other dogs were in sinus rhythm (mean heart rate of 105 ± 10 beats/min).

Electrophysiologic Data

Ventricular Tachycardia

At least one episode of sustained ventricular tachycardia was initiated by programmed electrical stimulation in 16 of the 20 dogs that underwent electrophysiologic study. Eleven dogs (55%) had reproducible, sustained ventricular tachycardia that could be initiated and terminated repeatedly by the same modes of programmed ventricular stimulation. Table 1 is a list of the modes of initiation and termination and rates of the ventricular tachycardias, as well as the age of the myocardial infarction and the infarct size in these animals. An example of reproducible sustained ventricular tachycardia, initiated and terminated by programmed stimulation, is shown in figure 1. None of the 20 dogs manifested spontaneous ventricular tachycardia at the times of electrophysiologic study.

Initiation and Termination of Ventricular Tachycardia. Brief (three to five beats) bursts of rapid ventricular pacing from the right ventricle and from multiple areas in the left ventricle at cycle lengths of 250–150 msec were the most reliable method of initiating ventricular tachycardia (figs. 1 and 2). Left ventricular stimulation using this technique resulted in initiation of sustained ventricular tachycardia in 16 dogs and
### Table 1. Characteristics of Electrically Induced Ventricular Tachycardia

<table>
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<tr>
<th>Dog</th>
<th>VT rate (beats/min)</th>
<th>Reproducible VT</th>
<th>VT Initiation</th>
<th>VT Termination</th>
<th>Distinct ECG morphology</th>
<th>Days post-MI</th>
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*In these two dogs, ventricular fibrillation frequently resulted by BRVP during VT.

**Abbreviations:** VT = ventricular tachycardia; MI = myocardial infarction; LV = left ventricular; BRVP = bursts of rapid ventricular pacing; CTPB = critically timed premature beats; Sp = spontaneously.

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**Figure 1.** In this example, ventricular tachycardia (VT) (cycle length 300 msec) is initiated by a short burst of rapid ventricular pacing (RVP) at a cycle length of 220 msec applied during normal sinus rhythm (NSR). VT is terminated by another short burst of RVP. S refers to the stimulus artifact. Surface ECG leads I and II, a right atrial electrogram, and local electrograms from left ventricular endocardial sites are displayed. The bottom tracing is recorded from the periphery of the infarct zone.
reproducible initiation of sustained ventricular tachycardia in 11 dogs (table 1).

The exact site of stimulation using bursts of ventricular pacing was unimportant in five dogs with reproducible ventricular tachycardia. In these dogs brief bursts of rapid pacing from the endocardium of the right ventricular free wall, normal (noninfarcted) left ventricle, and the infarct border zone (see Methods) all resulted in initiation of the same ventricular tachycardia (figs. 2A and 2B). In the other six with reproducible ventricular tachycardia, however, the successful initiation of ventricular tachycardia was critically dependent upon the site of stimulation. In these dogs, bursts of rapid ventricular pacing from the right ventricle and from the normally contracting segments of the left ventricle did not initiate ventricular tachycardia. Rapid pacing from specific areas in the periphery of the infarct zone was required to initiate ventricular tachycardia in these dogs (fig. 3). Once these “trigger zones” were identified, ventricular tachycardia could be initiated reproducibly by rapid pacing from these sites. Rapid pacing via electrodes in the center of the infarct zone (see Methods) was tried in all 20 dogs that underwent electrophysiologic study. This method initiated ventricular tachycardia in only two dogs. Failure to attain 1:1 capture during rapid pacing at short cycle lengths in the center of the infarct zone was responsible for the inability to initiate ven-
tricular tachycardia from this site. Thus, the frequency of induced ventricular tachycardia was highest when rapid pacing was applied from the infarct border zone areas.

In contrast to brief bursts of rapid ventricular pacing, the introduction of critically timed double left ventricular premature depolarizations during fixed-rate ventricular pacing from normal and infarcted left ventricular myocardium initiated sustained ventricular tachycardia in only four of 11 dogs. Critically timed double right ventricular premature depolarizations during fixed-rate right ventricular pacing were also performed in these 11 dogs. This technique successfully initiated sustained ventricular tachycardia in only two dogs.

Electrically induced sustained ventricular tachycardia was an organized, stable rhythm that showed no tendency to degenerate spontaneously into ventricular fibrillation. When hemodynamically well-tolerated (nine dogs), induced ventricular tachycardia was allowed to continue for 10–12 minutes. No episodes of spontaneous progression from ventricular tachycardia to ventricular fibrillation were observed.

Termination of induced sustained ventricular tachycardia was most reliably achieved with brief bursts of rapid left ventricular pacing at cycle lengths shorter than those of the tachycardia (fig. 1). This technique resulted in termination of ventricular tachycardia and reversion to normal sinus rhythm in 12 of 16 dogs with ventricular tachycardia. In two dogs with ventricular tachycardia rates of 353 beats/min and 343 beats/min, brief bursts of left ventricular pacing applied during ventricular tachycardia resulted in ventricular fibrillation, which was terminated by DC countershock. Ventricular tachycardia terminated spontaneously in two dogs that did not have reproducible ventricular tachycardia (table 1). Rapid pacing from areas in the infarct border zone with short effective refractory periods terminated ventricular tachycardia with the greatest degree of reliability in 12 dogs with sustained ventricular tachycardia. In contrast, we could terminate ventricular tachycardia by rapid pacing from the center of the infarct zone in only two dogs. Ventricular tachycardia was terminated by rapid pacing from the normal left ventricular areas in eight dogs and from the right ventricle in five.

Electrophysiologic Features of Ventricular Tachycardia. The ventricular tachycardia cycle lengths are shown in table 1. A His bundle electrogram was recorded reliably during ventricular tachycardia in four dogs. The ventricular electrogram during the tachycardia was never regularly preceded by a His bundle electrogram. Electrogram recordings from one or more endocardial sites within the periphery of the infarct zone during rapid pacing that initiated ventricular tachycardia frequently displayed late, fractionated potentials (figs. 3B and 4), suggesting that initiation of ventricular tachycardia was preceded by conduction delay and block in these areas. During sinus rhythm, recordings from bipolar plunge wires positioned at multiple endocardial sites in the normal left ventricle and the infarct zone demonstrated highly synchronous activation (fig. 4), whereas during ventricular tachycardia, there was asynchronous activation with temporal dispersion of up to 80 msec in ventricular activation recorded from the left ventricular endocardial electrodes (fig. 4). Local endocardial electrograms from at least one site at the periphery of the myocardial infarct zone preceded local electrograms recorded from the center of the infarct zone and from the normal left ventricular myocardium in nine dogs (figs. 1 and 4).

Ventricular capture by single or multiple induced ventricular premature depolarizations introduced during ventricular tachycardia without termination of the tachycardia was observed in seven dogs. Rapid ventricular pacing-induced alterations in the characteristics of the ventricular tachycardia appeared in four dogs. These pacing-induced alterations in the tachycardia included changes in rate and/or QRS complex morphology and ventricular activation sequence as recorded from multiple endocardial electrodes. Two morphologically distinct forms of inducible sustained ventricular tachycardia in the same dog were observed in five dogs (table 1).

**Effective Refractory Periods**

Determination of the effective refractory period of the ventricle from multiple endocardial sites within and around the infarct zone revealed wide heterogeneity in the values observed in each dog. The refractory periods within and at the borders of the infarct zone differed by a range of 30–70 msec in individual dogs (mean range 49 ± 18 msec). In contrast, effective refractory period determinations from multiple endocardial sites in the normal left ventricular myocardium revealed minimal heterogeneity (mean range 7 ± 4 msec). Within infarct zones there were individual regions with effective refractory periods both shorter and longer than those measured in normal left ventricular myocardium in the same hearts (table 2). This was a consistent finding in all 20 dogs.

<table>
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<th>Table 2. Effective Refractory Periods</th>
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<tr>
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Abbreviation: ΔERP = difference between the longest and shortest effective refractory period.
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FIGURE 3. (A) Initiation of sustained ventricular tachycardia (VT) with a cycle length of 290 msec by a burst of rapid ventricular pacing (RVP) at a cycle length of 200 msec from the periphery of the left ventricular infarct zone. ECG leads I and II, a right atrial electrogram, and local electrograms from left ventricular endocardial sites are shown. S refers to the stimulus artifact. (B) The initiation of VT by a burst of RVP at a cycle length of 200 msec from the periphery of the left ventricular infarct zone in another animal. RVP from the periphery of the infarct zone was the only reliable method of initiating VT in these dogs.

FIGURE 4. Endocardial activation sequence during normal sinus rhythm (NSR) and sustained ventricular tachycardia (VT). During NSR, bipolar recordings from multiple left ventricular endocardial sites demonstrate synchronous activation of all sites. The second local electrogram of the infarct zone records local activity from the periphery of the infarct. During VT, local electrograms recorded from this site precede those from other sites as well as the onset of surface ECG by variable intervals. ECG lead I and a right atrial electrogram are also shown. VT is initiated by a burst of rapid ventricular pacing (RVP).
Verapamil

Verapamil slowed the sinus rate from a mean of 109 ± 10 beats/min to a mean of 72 ± 13 beats/min and produced various degrees of atrioventricular block in the seven animals to whom it was administered. Fifteen to 30 minutes after verapamil administration, sustained ventricular tachycardia could be initiated by programmed cardiac stimulation in all five dogs that had sustained ventricular tachycardia before verapamil. The rate and QRS complex morphology of induced ventricular tachycardia was not altered by verapamil.

Pathology

Examination of serial coronal sections of the left ventricle in 17 of the 20 dogs studied revealed transmural anteropapical infarcts in all 17 dogs. In no dog was the infarct limited to the subendocardial region, although in two a narrow strip of subepicardial muscle was spared. The largest infarct occupied 25% of the total left ventricular mass and the smallest one involved 5% of the left ventricle (mean 14 ± 4%). The middle and upper portions of the interventricular septum as well as the posterior wall of the left ventricle were free of infarction in all the hearts examined. The size of the myocardial infarcts in these 17 dogs is shown in table 1. It can be seen that there was no correlation between infarct size and the rate, morphology, or mode of initiation and termination of ventricular tachycardia. The mean infarct size in dogs with electrically induced sustained reproducible ventricular tachycardia was 16 ± 5% and in dogs with no inducible tachycardia, 12 ± 5% (NS).

Discussion

This study demonstrates that nonsuperventricular, electrically induced sustained ventricular tachycardia can be initiated and terminated in a majority of dogs studied by programmed cardiac stimulation during the late (3-week) phase of experimental canine myocardial infarction. Furthermore, in this model, in approximately half of the dogs, electrically induced sustained ventricular tachycardia was reproducible. The anatomic substrate of this model was an anteropapical transmural infarct of uniform location in the left ventricle, which was consistently created by ligating the left anterior descending coronary artery and all the visible collaterals. The most successful method of initiating sustained ventricular tachycardia was the application of brief bursts of rapid left ventricular pacing from endocardial sites in the periphery of the infarction zone. The induced ventricular tachycardias were organized, stable rhythms that persisted for many minutes and demonstrated many electrophysiologic phenomena encountered in electrically induced human ventricular tachycardia seen in the setting of chronic ischemic heart disease. 9, 10

Electrically induced ventricular tachycardia in a model of recent myocardial infarction has been previously studied by El-Sherif et al. 4, 5 There exist several important differences between this latter model and the one described in our study. In our model, the experimental myocardial infarction was created by ligating as many visible collaterals as possible in addition to the left anterior descending coronary artery. This resulted in large and easily identifiable transmural infarctions, instead of the creation of patchy areas of subendocardial infarction that can result from the proximal ligation of a single coronary artery in the dog. El-Sherif et al. investigated the electrophysiology of ventricular arrhythmias in 3-week-old canine myocardial infarction. We studied the dogs approximately 3 weeks after experimental myocardial infarction. Healing is nearly complete at 3 weeks, 11 so the electrophysiologic properties of the myocardium in our model are more likely to resemble those that prevail during the chronic state. This also corresponds to the time when most patients are discharged from the hospital after myocardial infarction. A subgroup of patients may be at risk for developing life-threatening ventricular arrhythmias at this time, as suggested by recent data from the Framingham Study in which a high incidence of unexpected sudden death was observed during the first month after hospital discharge. 12 However, clinical data concerning electrically induced ventricular arrhythmias in patients during the late phase of acute myocardial infarction are scarce, and the incidence of inducible ventricular tachycardia during this phase is not known.

Another feature of our model that is different from that of El-Sherif et al. is our use of multiple bipolar endocardial electrodes for recording and pacing. A composite epicardial electrode was used for recording and identifying areas of continuous electrical activity in the previous model. 4, 5 A recent study, however, suggested that in 1-2-day-old experimental myocardial infarction, the earliest point of activity during ventricular tachycardia was located in the endocardium and that epicardial breakthrough points could be centimeters away from this area. 6 Thus, to better locate the origin of the tachycardia, we used a grid of bipolar endocardial wires. Even in areas with the greatest degree of conduction delay and fractionation of local electrograms, continuous electrical activity was never recorded from the individual bipolar endocardial electrograms during the sustained, organized form of ventricular tachycardia observed in this model. Despite the limited number of recording electrodes, analysis of the endocardial activation sequence from multiple plunge wire electrodes in nine dogs revealed early electrical activity (preceding onset of the surface ECG) emanating from the periphery of the infarct zone during ventricular tachycardia in all nine dogs.

It is important to pace from multiple sites, especially the perinfarction zone of the left ventricle, in initiating sustained ventricular tachycardia. Stimulation of the right ventricle alone initiated sustained ventricular tachycardia in only five of 16 animals. Pacing from normal areas of the left ventricle was also insufficient to cause bursts of rapid ventricular pacing from the perinfarction zone for the
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The mechanism of sustained ventricular tachycardia is indeed reentrant, available evidence suggests that the anatomic pathway involved is relatively small. In four dogs in which a recording from the bundle of His was obtained, dissociation between the His bundle and local ventricular electrograms was present during ventricular tachycardia. Furthermore, initiation of ventricular tachycardia was dependent on retrograde conduction delay or reentry within the His-Purkinje system. These observations suggest that the proximal His-Purkinje system is not a requisite component of the tachycardia pathway. The ability to capture both ventricles with single and multiple premature ventricular depolarizations as well as with bursts of ventricular pacing without interrupting the ventricular tachycardia also suggests the presence of a relatively small pathway. It is unlikely that an anatomically large pathway in the ventricles would escape penetration by one or more induced depolarizations that resulted in biventricular capture. However, direct localization of pathways will require extensive mapping techniques.

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References

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