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Electrophysiologic Characteristics at Initiation of Ventricular Tachycardia and Ventricular Fibrillation in a Canine Infarct Model

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Summary: Local ventricular activation time and the conduction time during sinus rhythm at the induction of ventricular tachycardia (VT) and ventricular fibrillation (VF) were investigated using a canine model of chronic myocardial infarction. Of 26 dogs studied, 15 had inducible VT, 10 had inducible VF, and 1 had no inducible arrhythmias. Bipolar local ventricular electrograms were recorded during sinus rhythm from 136 sites in 10 dogs with VT and 164 sites in 11 dogs with VF. Mean activation time in dogs with inducible VT was significantly longer than in dogs with inducible VF. Furthermore, simultaneous local ventricular electrograms were recorded during the induction of VT (74 episodes) or VF (38 episodes) from the infarct border zone at the endocardium (B-EN), the epicardium (B-EP), and normal sites (N-EN, N-EP). During VT induction, the activation time at N-EN and N-EP was significantly longer than during VF induction (N-EN: 94 ± 21, 70 ± 19 ms; N-EP: 83 ± 21, 64 ± 10 ms; $p < 0.05$). Conduction time was measured at the initiation of VT or VF induced by orthodromic or antidromic pacing. The conduction times of the last paced beat between N-EN and B-EP (35 ± 11, 62 ± 24 ms), N-EN and N-EP (35 ± 12, 14 ± 13 ms), B-EN and B-EP (16 ± 10, 38 ± 25 ms), and B-EP and N-EP (77 ± 27, 44 ± 12 ms) were significantly different in dogs with inducible VT ($p < 0.05$), but not in dogs with VF. Dispersion of effective refractory period was also observed in dogs with VT. Percent infarct in inducible VT was larger than in inducible VF (VT: 16 ± 5%; VF: 10 ± 2%; $p < 0.001$). These studies suggest that dogs with inducible VT have prolonged ventricular activation time and significantly different bidirectional conduction time during VT induction. This may serve as a substrate for reentry.

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Introduction

Ventricular tachycardia (VT) and ventricular fibrillation (VF) are inducible by programmed electrical stimulation in canine infarct models.[1–3] These models closely resemble induced ventricular arrhythmias in humans with coronary artery disease.[4–6] Inducible VT is considered a specific response in patients with documented sustained VT, but induced VF is considered a nonspecific event.[7, 8] Therefore, we have separately assessed the characteristic of substrates in VT and VF induced in the electrophysiologic laboratory.

The purpose of the study presented here was to determine whether there are electrophysiologic differences measurable in 3-dimensional electrocardiography between inducible VT and inducible VF in a canine heart model 2 weeks after anteroapical infarction. The duration of local ventricular activation and transmural conduction time during induction of ventricular arrhythmias was examined.

Materials and Methods

Surgical and Postoperative Treatment

Thirty-six mongrel dogs, weighing 14.5 to 27.3 kg (mean 19.1 ± 2.7 kg), were anesthetized with thiopental 20–25 mg/kg followed by a mixture of nitrous oxide and halothane, ventilated with an MA-1 respirator, and surgery was performed using sterile technique. The hearts were exposed through a left lateral thoracotomy in the fourth intercostal space and suspended in a pericardial cradle. The anterior wall and apex of the left ventricle were exposed and the left anterior descending coronary artery was ligated immediately distal to the first septal perforating branch. In addition, the most distal portion of all visible epicardial branches near the left ventricular apical area originating from the left circumflex and posterior descending coronary arteries were also ligated. The pericardium was sutured loosely, and the thoracotomy closed with a chest tube in place. The dogs were then extubated and received pentazocine lactate approximately every 8 h for the first 72 h to control postopera-

tive pain. The animals with infarct were placed in an intensive care setting and treated to prevent the development of early VF. Animals were given 2 mg/kg of lidocaine HCl intravenously during ligation. At the completion of the operation, the dogs were placed in a quiet recovery area and 250 mg IM of procainamide HCl was administered twice daily for 3 days.

Follow-Up Study

Sixteen to 24 (mean 18 ± 1) days later, the dogs were anesthetized with pentobarbital (30 mg/kg) followed by continuous infusion (80 mg/h) and ventilated mechanically through an endotracheal tube with 100% oxygen during the surgical procedure. The hearts were approached through the fifth intercostal space by a left lateral thoracotomy. Thirty-two (16 for stimulation and 16 for recording) teflon-coated 0.005" diameter stainless steel plunge electrodes with 0.5–1.0 mm barbs at the exposed ends were inserted parallel to the left anterior descending artery on the anterolateral left ventricle and anchored to the endocardial or subepicardial surface with 23 gauge needles. Of the 16 recording electrodes, 4 were placed 0.5 cm apart on the endocardial sites along the line of visually identified infarct border zone, 4 on the subepicardial sites along the border zone, 4 in the normal endocardial sites 1.5 cm from the border zone, and the remaining 4 electrodes on normal subepicardial sites. Another 16 pairs of teflon-coated 0.005" diameter stainless steel plunge electrodes for pacing were inserted and anchored at the closest sites possible to the recording electrodes (Fig. 1).

Electrocardiographic monitoring consisted of the following: standard lead II electrocardiograms (ECG), right ventricular endocardial electrograms for reference, border zone endocardial electrograms (A), border zone subepicardial electrograms (B), normal endocardial electrograms 1.5 cm from the border line (C), and normal subepicardial electrograms (D) in a longitudinal direction. All intracardiac electrograms were filtered at a bandpass of 30–500 Hz and recorded on photographic paper or an eight-channel FM recorder (SONY) for future playback and analysis. The systemic arterial pressure was monitored via a femoral arterial catheter.

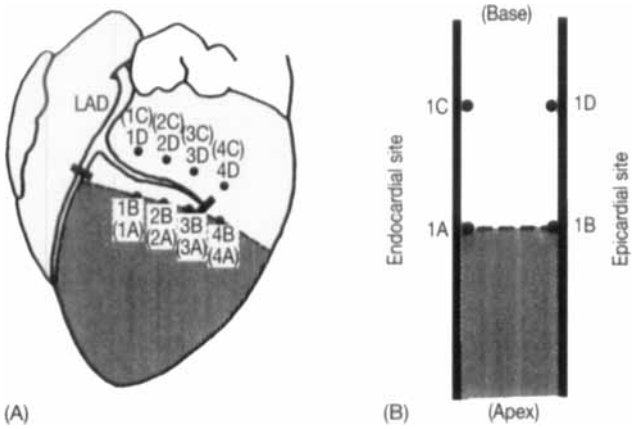

FIG. 1 Position of the plunge electrodes in superficial (A) and transmural (B) views. Closed circles indicate the position of two pairs of bipolar plunge electrodes (one for recording and one for pacing). Hatched area indicates an infarct area. Dotted lines indicate the border zone between infarct myocardium and normal myocardium. LAD = left anterior descending coronary artery.

Electrophysiologic Protocol

After recording ECGs during sinus rhythm in each longitudinal direction, programmed stimulations were performed at the previously described sites using a programmable stimulator (Medtronics Model 5325, Minneapolis, Minn.) at twice the diastolic threshold with a pulse width of 2.0 ms. Programmed single and double premature ventricular stimuli were delivered during ventricular pacing at drive cycle lengths of 250, 300, and 350 ms. Ventricular pacing of eight beats was followed by stimuli of increasing prematurity (S2) until ventricular refractoriness occurred. S2 was then placed 10 and 20 ms later than the ventricular refractory period, and an additional extrastimulus (S3) was added at a proper interval identified by 10 ms decrements until S3 failed to capture the ventricle. Programmed premature stimulation was done from all stimulation sites in all dogs (16 sites in each dog). Electrogram recordings were made in each longitudinal direction until induction of tachycardia and while it was sustained. Induced VF was terminated as soon as possible by electrical cardioversion and 100% oxygen was given to these dogs. Fifteen minutes were allowed to elapse between induction of VT or VF at the first stimulation and commencement of repeat stimulation. During the procedure, arterial oxygen saturation was adjusted to 95–97%.

Measurement

In sinus rhythm, the total duration of ventricular activation time was measured in ms from the onset of the local ventricular electrogram to the end of the local ventricular electrogram for all 16 sites (4 endocardial sites on the infarct border, 4 subepicardial sites on the infarct border, 4 endocardial sites on normal tissue, and 4 subepicardial sites on normal tissue). If VT or VF was induced, local ventricular activation time (VAT), conduction time (CT), and stimulus latency response to the drive train of eight paced beats (S1) and after the first and second extrastimuli (S2, S3) were measured in all recording sites at the initiation of ventricular tachyarrhythmias (Fig. 2). The duration of VAT was measured after S1, S2, and S3 from the onset to the end of the local electrogram at the induction of arrhythmias. The duration of the conduction time was measured between the onset of electrogram at the stimulated site and the onset of local electrical activity at the induction of arrhythmias. When the ventricular tachyarrhythmias were induced from two sites in the same dog, we analyzed the conduction time between one pacing site to the other recording site, that is, bidirectional conduction time among two points. We defined that "orthodromic" indicated the direction from cardiac base to apex or the direction from endocardium to epicardium and "antidromic" indicated the direction from apex to cardiac base or the direction from epicardium to endocardium. Stimulus latency was measured as the time from the stimulus artifact to the onset of electrical activity

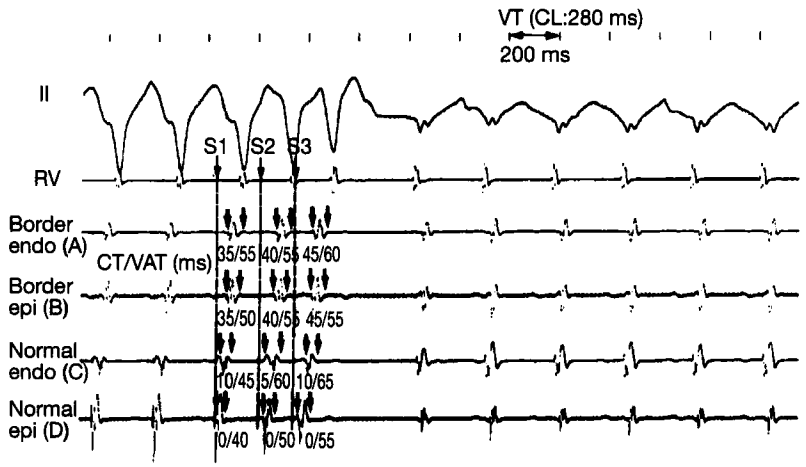

FIG. 2 Initiation of sustained ventricular tachycardia (VT) with a cycle length (CL) of 210 ms by two premature ventricular extrastimuli (S1-S1: 250 ms, S1-S2: 190 ms, S2-S3: 170 ms) (dotted lines) introduced at the border epicardium site B. Lead II is shown, together with bipolar recording from the right ventricle (RV), endocardial recording from the border zone (A) and normal myocardium (C), and subepicardial recording from the border zone (B) and normal myocardium (D). Arrows represent an onset or an end of local electrograms. The conduction time (CT), between the stimulation and the onset of local electrogram and the local ventricular activation time (VAT) between the onset of local electrogram and the end of local electrogram were measured in ms and are represented below each electrogram.

at the stimulation site at the induction of arrhythmias. All data were measured at the initiation of VT or VF only.

Postmortem Evaluation of the Heart

The dogs were euthanized with KCl intravenously and the hearts rapidly removed. The left ventricle was sectioned transversely at 0.5 cm intervals from apex to base and incubated at 37°C for 15 min in a buffered tri-phenyltetrazolium chloride (TTC) solution. This oxidation-reduction indicator produces a bright red coloration of tissue with normal dehydrogenase activity. Each section was weighed and drawn on a clear plastic sheet with indications for infarcted and noninfarcted areas. These areas were measured by planimetry, and the area of necrosis expressed by percentage of total left ventricular myocardium.

Definitions

Sustained ventricular tachycardia: VT that lasted for more than 30 s or required termination by pacing or cardioversion; nonsustained ventricular tachycardia: VT that lasted for three or more complexes and terminated spontaneously within 30 s. Ventricular fibrillation: disorganized rhythm with irregularly timed endocardial electrical activity, no clearly defined QRS complexes on the ECG, or QRS complexes of continuously varying configurations.

Data Analysis

For continuous variables, paired and unpaired *t*-tests were used. Values are mean ± standard deviation. Differences were considered significant at $p < 0.05$.

Results

Twenty-six dogs survived at least 2 weeks after the initial thoracotomy. Four dogs died within 24 h of the first operation and another 6 dogs died 4 to 14 days following surgery.

Induction of Ventricular Arrhythmias

Of the 26 surviving dogs, 15 (58%) had inducible VT [11 had sustained monomorphic VT (42%) and 4 had nonsustained VT (15%)]. VF was inducible in 10 dogs (38%). Only one dog had neither inducible VT nor VF. The mean cycle length of the induced sustained monomorphic VT was 226 ± 34 ms (range 170–280 ms). VF was induced at 38 of 136 pacing sites in 10 dogs (28%). Sustained monomorphic VT was induced at 74 of 164 pacing sites in 11 dogs (45%) (VT vs. VF; $p = 0.0022$). Sustained monomorphic VT was reproducibly induced in 8 of 11 dogs. The remaining three dogs had only one induction of VT. All induced sustained VTs were well tolerated hemodynamically. The incidence of tachycardia induction at each pacing site was (a) the border zone between the infarct and the normal area endocardium: VT 23/42 (55%), VF 17/37 (46%); (b) border epicardium: VT 19/40 (48%), VF 6/27 (22%); (c) the normal endocardium VT 17/41 (41%), VF 11/36 (31%); (d) the normal epicardium VT 15/41 (37%), VF 4/36 (11%). None of these differences were significant.

Sinus Mapping

Local electrograms during sinus rhythm were recorded from 300 myocardial sites in 11 dogs with inducible VT, 10 dogs with inducible VF, 4 dogs with nonsustained VT, and 1 noninducible dog. Although the maximum VAT showed no significant difference among tachyarrhythmias in each site, mean

TABLE I Duration of ventricular activation time during sinus rhythm

	Inducible sustained VT	Inducible VF	Nonsustained VT	Non-inducible
N	11	10	4	1
Max. VAT (ms)	96 ± 22	94 ± 29	85 ± 15	65
Mean VAT (ms)	70 ± 5	62 ± 4 [a]	62 ± 3 [c]	52
Endo. VAT (ms)	72 ± 5	64 ± 5 [a]		
Epi. VAT	67 ± 9	59 ± 7 [b]		

[a] $p < 0.005$ vs. sustained VT; [b] $p < 0.05$ vs. sustained VT.
Abbreviations: VT = ventricular tachycardia, VF = ventricular fibrillation, Max. VAT = maximum local ventricular activation time; Endo. = endocardium, Epi. = epicardium.

VAT in dogs with inducible VT was significantly longer than in those with inducible VF or nonsustained VT (Table I).

Extrastimuli and Refractory Periods

The mean S2-S3 coupling intervals at which VT and VF could be induced were 153 ± 26 ms and 137 ± 18 ms, respectively ($p < 0.001$). Three dogs with inducible VT required only one extrastimulus for VT induction. The mean effective refractory periods (ERP, basic drive cycle length = 300 ms) from all pacing sites (VT: 132 sites, VF: 72 sites) were 162 ± 21 ms in VT dogs and 173 ± 16 ms in VF dogs ($p < 0.001$). Using the four dogs with nonsustained VT and a noninducible dog as controls, ERP in these dogs (66 sites) was 155 ± 18, significantly shorter than that of VT and VF dogs (vs. VT: $p = 0.018$, vs. VF: $p < 0.001$). Furthermore, ERP from sites of inducible VT or VF was 164 ± 16 ms in VT and 171 ± 14 ms in VF dogs ($p = 0.024$) (Fig. 3). Differences between maximum ERP and minimum ERP were 38 ± 12 ms in VT dogs and 27 ± 7 ms in VF dogs, respectively ($p < 0.05$) (Fig. 4).

Stimulus Latency at the Pacing Site

The duration of stimulus latency during initiation of VT and VF at each pacing site was examined. There was no significant difference in the latency after stimulus S1 and S2 between VT and VF. However, the duration of stimulus S3 latency at the border endocardium and the normal epicardium in dogs with VF was significantly longer than in dogs with VT. (Fig. 5).

Local Activation Time

The local activation time at the border endocardium, the border epicardium, and the normal endocardium response to S1 and S2, which induced ventricular tachyarrhythmias, was not significantly different between dogs with inducible VT versus VF. The local activation time response to S1 and S2 at the normal epicardium at which VT was inducible was higher than that at which VF was induced (S1: VT 65 ± 20 ms, VF 59 ± 11 ms, $p = 0.031$; S2: VT 69 ± 20 ms, VF 64 ± 12 ms, $p = 0.063$). The local activation time at the border endocardium and the border epicardium response to S3, which induced VT and VF, was not significantly different. However, the local activation time at the normal endocardium and the normal epicardium response to S3 which induced VT was significantly higher than in VF (normal endocardium: VT 94 ± 21 ms, VF 83 ± 21 ms, $p = 0.028$; normal epicardium: VT 70 ± 19 ms, VF 64 ± 10 ms, $p = 0.038$) (Fig. 6).

Ventricular Conduction Time

The duration of conduction time at two points close on the myocardium in response to S1 and S2 at the induction of arrhythmia was not significantly different between VT and VF. The conduction time from the border endocardium to the normal epicardium in response to S1 was 21 ± 8 ms in VT and 16 ± 5 ms in VF ($p = 0.0348$). The duration of conduction time at which VT was inducible in response to S3 was significantly shorter than in VF (VT vs. VF; border endocardium to normal endocardium 14 ± 15 ms vs. 26 ± 17 ms, $p = 0.0484$; border epicardium to normal endocardium 62 ± 24 ms vs. 28 ± 16 ms, $p = 0.0478$; border endocardium to border epicardium 16 ± 10 ms vs. 31 ± 21 ms, $p = 0.00685$; border epicardium to normal epicardium 77 ± 27 ms vs. 33 ± 17 ms, $p = 0.0262$).

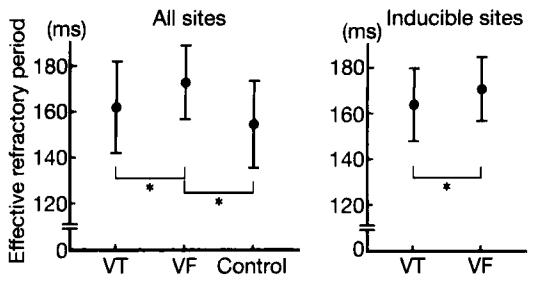

FIG. 3 Comparison of effective refractory periods between hearts inducible into VT versus VF. ERP in VF was significantly longer than in VT. * $p < 0.05$.

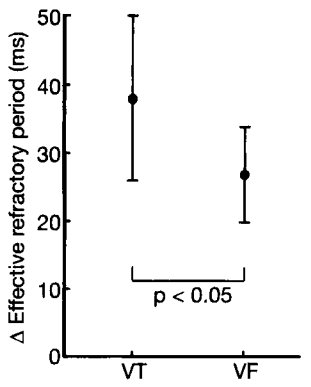

FIG. 4 Comparison of dispersion of effective refractory periods between hearts inducible into VT versus VF. Dispersion of ERP in VT was significantly larger than in VF.

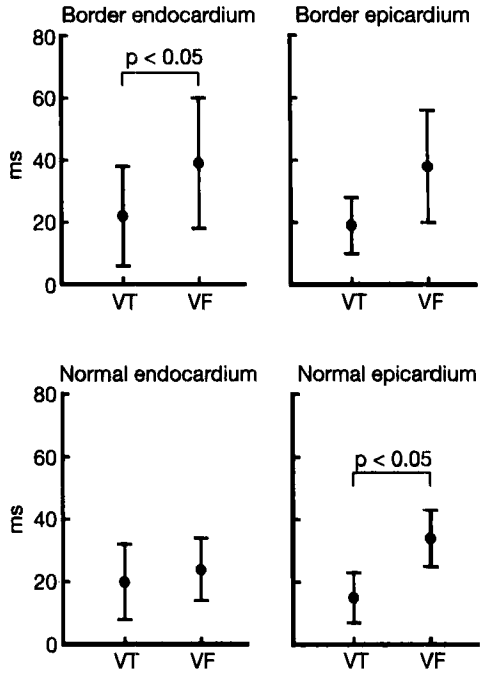

FIG. 5 Stimulus latency at pacing sites. The duration of stimulus latency at the border endocardium and normal epicardium in dogs with VF was significantly longer than in VT.

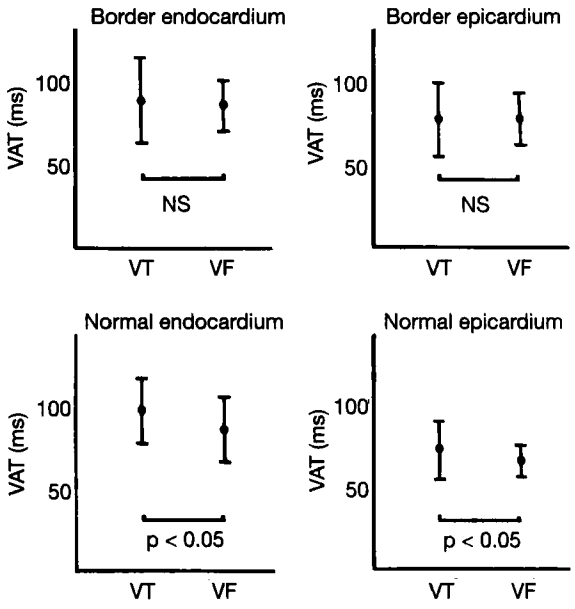

FIG. 6 Local ventricular activation time for the last extrastimulus at induction of VT or VF. The local activation time response at the normal endocardium and normal epicardium to the last extrastimulus at which VT was induced was significantly higher than in VF. VAT = local ventricular activation time.

Bidirectional Ventricular Conduction Time

In dogs with VT, the duration of bidirectional conduction time in response to S1 between the border endocardium and the normal epicardium was significantly different (orthodromic: 21 ± 8 ms, antidromic: 30 ± 9 ms; $p = 0.0348$). The duration of bidirectional conduction time in response to S2 between the normal endocardium and the border epicardium (orthodromic: 26 ± 15 ms, antidromic: 40 ± 15 ms; $p = 0.0318$) and between the border endocardium and the normal epicardium (orthodromic: 25 ± 11 ms, antidromic 38 ± 11 ms; $p = 0.0046$) were also significantly different in dogs with VT. Furthermore, the duration of bidirectional conduction time response to S3 between the normal endocardium and border epicardium (orthodromic: 35 ± 11 ms, antidromic: 62 ± 24 ms; $p = 0.00415$), the normal endocardium and the normal epicardium (orthodromic: 35 ± 12 ms, antidromic: 14 ± 13 ms; $p = 0.0054$), the border endocardium and the border epicardium (orthodromic: 16 ± 10 ms, antidromic: 38 ± 25 ms; $p = 0.0138$), and the border epicardium and the normal epicardium (orthodromic: 77 ± 27 ms, antidromic: 44 ± 12 ms; $p = 0.025$) were significantly different in VT dogs, while no significant difference existed in VF dogs (Figs. 7, 8).

Infarct Size of the Heart

Postmortem gross examination of serial coronary sections of the left ventricle in all 11 dogs with inducible VT showed transmural anteroapical infarction. However, transmural infarction was observed in only six of nine dogs with inducible VF and one of five dogs with NSVT or noninducible ventricular arrhythmias. The percent mean infarct size in dogs with inducible VT was significantly larger than in VF or in noninducible dogs (VT: 16 ± 5%; VF: 10 ± 2%; noninducible: 6 ± 3%, VT vs. VF: $p < 0.001$, VF vs. noninducible: $p < 0.05$).

Discussion

The present study suggests differences in electrophysiologic characteristics between the induction of VT and VF in an experimental dog model of myocardial infarction. We have shown that these tachyarrhythmias differ with respect to the duration of activation time, the conduction time, and the transmural bidirectional conduction time between two points during the initiation of arrhythmia.

Induction of Arrhythmias

Perhaps the most important finding obtained from this programmed stimulation method is that it can be performed from various sites, nearly equaling the number of recording electrodes. Previous studies[9–12] have reported that both sustained or nonsustained VT and VF were induced in 38 to 60% (49 ± 7%) and 18 to 50% (34 ± 11%) of dogs with MI, respectively, using two or three extrastimuli and occasionally using burst pacing or high current during the programmed stimulation. These data are consistent with our study (VT: 58%, VF: 38%). However, we used only two extrastimuli for induction and did not

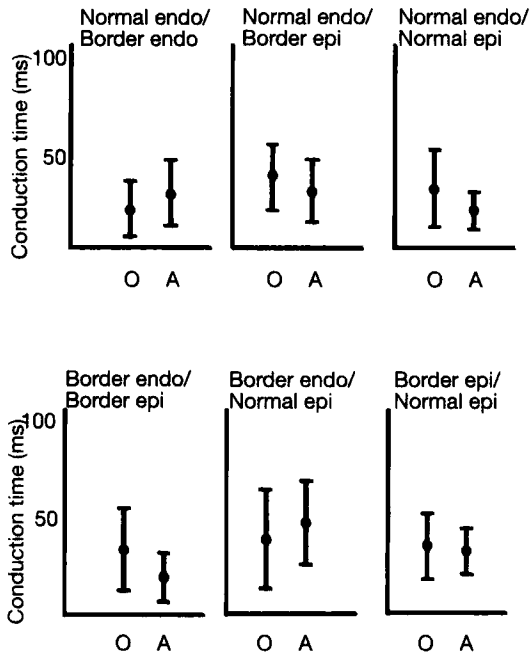

FIG. 7 Conduction time response to the last extrastimulus at induction of VF. The duration of conduction time in response to the last extrastimulus between the orthodromic direction and the antidromic direction was not significantly different at any site in dogs with VF. O = orthodromic, A = antidromic.

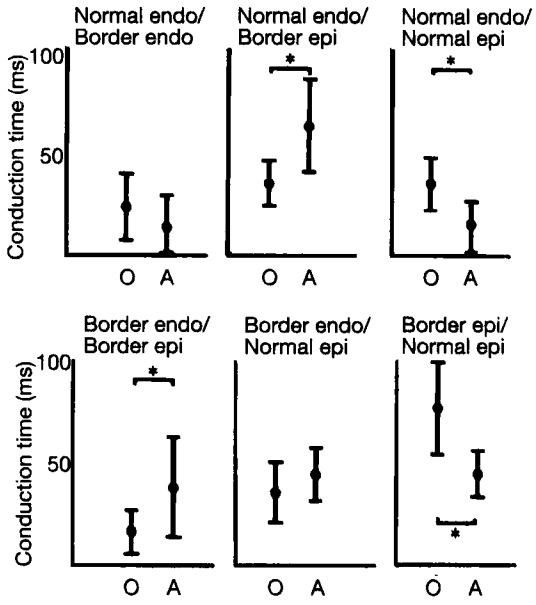

FIG. 8 Conduction time response to the last extrastimulus at induction of VT. The duration of bidirectional conduction time in response to the last extrastimulus between the normal endocardium and the border epicardium, the normal endocardium and the normal epicardium, the border endocardium and border epicardium, and the border epicardium and the normal epicardium was significantly different. O = orthodronic, A = antidromic. * $p < 0.05$.

use burst pacing or high current stimulation. Furthermore, the rate of induced sustained VT in this study was lower than previously reported,[13] and induced VT was well tolerated hemodynamically. This may be due to different models of producing MI or the stimulation protocol. In the study reported here, a homogeneous infarct model with left ventricular aneurysmal dilation was created by ligating the left anterior descending coronary artery as well as all epicardial branches serving the left ventricular apex as previously reported by Garan *et al.*[10] In their model, virtually all induced VT originated from endocardial areas which is similar to the postmyocardial infarction VT seen in patients.[14] Michelson *et al.*[9] emphasized the importance of the stimulation site for induction of VT. However, in our study, there was no significant difference in VT induction within limited sites of stimulation including endocardial and subepicardial sites of the border zone or within normal tissue. This result is consistent with the recent clinical paper of Dailey *et al.*[15] who reported that the results of programmed stimulation from epicardial sites were concordant with those from the endocardium. In our study, VF was difficult to induce with stimulations from subepicardial sites.

Infarct Size and Induced Arrhythmias

It has been noted that the occurrence of VT is associated with large infarct size in dogs[3, 16] and in the clinical setting.[4] Our data are compatible with those studies. Large infarct size may be a contributing factor to the development of a reentrant circuit.

Local Activation Time

Several studies in patients with previous myocardial infarction[4–6, 17, 18] or the chronic canine infarct model[1–3] have suggested that hearts with inducible VT have longer local electrogram durations in sinus rhythm than those with inducible VF. We found that, although there was no difference in the maximum VAT during sinus rhythm between dogs with VT and VF, the mean VAT of dogs with VT was significantly longer than of those with VF. A significant difference in VAT either at the subendocardium or the subepicardium between VT and VF was also noted, but this difference was more prominent at the subendocardium. This varies from the results of Denniss *et al.*[3] who noted that there were differences in local activation time between VT and VF in the epicardium but not the endocardium. This may be due to differences in the infarct dog model used. We also found that the VAT after drive train pacing or the second extrastimulus in dogs with VT were longer than in those with VF. We also found that the differences in induction of VT and VF were related to critical differences of the coupling interval of the last extrastimulus and the refractory periods. This is consistent with the results of previous studies.[3, 19] The present study also demonstrates that not only prolongation of ERP but also disproportion of ERPs were an important factor in VT induction. Furthermore, we demonstrate that the stimulus latency after S2 at the stimulation site in dogs with VF was longer than in VT, as previously reported.[20] Our results were contrary to Denniss *et al.*[3] who stated that there were no significant dif-

ferences in the local activation time during S1, S2, or S3 between inducible VT and VF. Although we measured conduction time after S1, S2, or S3 from the onset of the local electrogram, Denniss *et al.* measured it from the stimulus artifact to the end of the electrogram.[3] Therefore, activation time and the stimulus latency may cancel each other. We did not compare the activation time with a control group given the small sample size. Also, we used data obtained only during the initiation of VT or VF as VAT after the programmed stimulation, and therefore had no available data for a control group. However, this present study strongly suggests that, when compared with VF, the induction of VT is intimately associated with local conduction delay in sinus rhythm and marked conduction delay present after ventricular extrastimuli.

At the induction of tachyarrhythmia, we compared the conduction time between the onset of electrical activity at the stimulation site and the onset of other local electrograms with the conduction time of arrhythmias induced from the opposite direction. The present study shows that the conduction time from the orthodromic direction was different from that in the antidromic direction in dogs with VT. This finding may indicate the presence of heterogeneity in conduction or the unidirectional conduction block substrate of reentry.

Limitations of the Study

We analyzed data from 12 to 16 sites of the subendocardium or the subepicardium in each dog during induction of VT or VF. The distance between the bipolar plunge electrodes was approximately 15 mm and we were unable to record from the intramyocardial electrical activity. Since the myocardial mass in the zone of reentry may be smaller, precise location of the reentrant circuit from our data is limited. Further investigation involving a greater number of electrodes with recording from the intramyocardium may be useful in further determining the mechanisms of arrhythmia induction.

Conclusion

We have demonstrated differences in electrophysiologic characteristics during induction of VT and VF in a canine infarct model. Longer local activation time and heterogeneous local conduction were present at the induction of VT and may be associated with a substrate for reentry.

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