Reentrant Ventricular Arrhythmias in the Late Myocardial Infarction Period

6. Effect of the Autonomic System

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SUMMARY The effect of the autonomic system on conduction disorders in the infarction zone (IZ) and related reentrant ventricular arrhythmias (RVA) in the late myocardial infarction period in the dog was studied utilizing averaged recordings of the reentrant pathways from the epicardial surface of the IZ. Vagal (V) stimulation was found to have no significant direct electrophysiologic effect while sympathetic (S) stimulation resulted in a direct slight improvement of conduction in the IZ. However, because of the marked rate-dependency of conduction in the IZ, the effects of both V and S stimulation were modified through changes in the heart rate. The tachycardia produced by V stimulation resulted in improvement of conduction in the IZ and disappearance of RVA, while the tachycardia induced by S stimulation resulted in worsening of conduction in the IZ and the occurrence of RVA. Thus, in spite of its slight enhancing effect on conduction in the IZ, the propensity of S stimulation to induce RVA was primarily due to its tachycardic effect.

FOR MORE THAN A CENTURY, investigators have proposed that the autonomic system is involved in the genesis of cardiac arrhythmias.1 The role of the autonomic system in cardiac arrhythmias related to myocardial ischemia is of current interest because of its possible therapeutic implications. In recent years, there has been controversy over the beneficial effects of increased vagal tone on ventricular electrical stability during acute myocardial infarction,2-6 as well as over the possible deleterious effects of vagolytic drugs.6-7 Although it is widely accepted that sympathetic impulses can facilitate the development of serious ventricular arrhythmias in the setting of acute myocardial ischemia,8-10 and that sympathectomy and β-adrenergic blockade can have the opposite effect,11-18 the exact mechanisms by which these effects are mediated are not clear.

The role and nature of a direct electrophysiologic effect of sympathetic impulses on ischemic myocardium, as well as the contribution of indirect effects including heart rate acceleration and systemic pressor responses, have not been established. Studies of the effects of the autonomic system on the early phase of ventricular arrhythmias after acute myocardial infarction are usually complicated by the presence of a rapidly changing electrophysiologic situation and the limitations of available recording techniques19 so that exact changes in the electrophysiologic parameters are difficult to discern.

In the late myocardial infarction period (three to seven days postinfarction), the propensity for reentrant ventricular arrhythmias (RVA) is still main-

The results of this study were obtained from 12 adult mongrel dogs that were studied three to seven days following ligation of the left anterior descending artery just distal to the anterior septal branch. In all dogs a transmural infarction was evident on gross postmortem examination. Recordings were obtained from the epicardial surface of the IZ and adjacent normal zone (NZ) utilizing a composite electrode that records averaged signals of multiple close bipolar sites. In addition, one to three close bipolar recordings from the IZ and/or NZ were also obtained. Details of the surgical procedure and the recording techniques are described elsewhere.17 In addition to the electrograms, two standard electrocardiographic leads were recorded, leads II and aVr. All records were obtained on a multichannel oscilloscopic photographic recorder (Electronics for Medicine, DR-8) at paper speeds of 25-200 mm/sec. Electrocardiograms were recorded with the preamplifier set for frequencies of 0.1-200 cycles/sec and bipolar electrograms were recorded with filter frequencies of either 40-200 cycles/sec or 12-200 cycles/sec. The measurements' error was ± 3 msec at a paper speed of 200 mm/sec.

Dogs were anesthetized with intravenous sodium pentobarbital (30 mg/kg). A jugular vein was cannulated for the administration of fluids, and blood pressure in the femoral artery was monitored through a polyethylene catheter connected to a Statham transducer. The sinus node area was either crushed or excised to obtain a slower atrial or AV junctional rhythm. Atrial or His bundle pacing was used to control the ventricular rate. Atrial pacing was achieved
via two fine stainless steel wires (0.003 in diam) inserted through a 25-gauge hypodermic needle into the left atrial appendage. His bundle pacing was accomplished through the electrodes on the catheter recording the His bundle electrogram.22 Both regular pacing and premature stimulation were performed using a programmed digital stimulator that delivered rectangular impulses of 1.5 msec duration at approximately twice diastolic threshold. V stimulation was accomplished by delivery of 0.05 msec square wave pulses of 1–10 V intensity at a frequency of 20 Hz through two silver wires (0.012 in diam) inserted into the distal portion of the right or left vagosympathetic trunk.22 Stimulation was performed using an S-88 Grass stimulator and SIU 5 isolation unit. The voltage was gradually increased until idioventricular escape rhythm appeared, following progressive slowing of the atrial or AV junctional pacemaker. Ventricular rate was then controlled by His bundle pacing.

Following study of the effect of V stimulation, the vagosympathetic nerves were then severed in the neck, and the right and left cardiac sympathetic nerves were dissected in the mediastinum. The stellate ganglia and the ansa subclavia were exposed beneath the parietal pleura between the first and second ribs posteriorly. The stimuli were delivered to the ventral limb of the intact ansa subclavia through bipolar silver wires. Bilateral stimulation of the sympathetic nerves was accomplished using the same technique as V stimulation. The voltage of stimulation was gradually increased until the control rate of the atrial or junctional pacemaker increased by at least 50%. A pressor response (20–30 mm Hg increase in mean arterial pressure) was also regularly observed. The ventricular rate was then manipulated by either atrial or His bundle pacing. Results were statistically analyzed using the paired t test.

Results

Effect of Vagal Stimulation

Previous studies have shown that conduction disorders in the IZ are consistently tachycardia-dependent with the conduction being markedly sensitive to changes in the cardiac cycle length.17–19 V stimulation was found to have no direct electrophysiologic effect on conduction disorders in the IZ or related RVA. This is illustrated in figures 1 and 2, which were obtained from the same experiment. Figure 1 shows control recordings. Traces from top to bottom represent standard lead II, an electrode catheter recording of the His bundle electrogram (Hbeg) and composite electrode recordings from the infarction zone electrogram (IZeg) and adjacent norm-

Figure 1. Control recordings from an experiment showing tachycardia-dependent conduction disorders in the infarction zone (IZ) and related reentrant ventricular arrhythmias. Panel A illustrates an escape atrial rhythm while panels B, C and D were obtained during atrial pacing. There is a 3:2 Wenckebach-like conduction pattern of the IZ potential (marked by arrows) in panel C associated with a manifest trigeminal rhythm. Pacing at a faster rate in panel D resulted in 2:1 block of the IZ potential and disappearance of manifest reentry. Abbreviations: Hbeg = His bundle electrogram; IZeg = infarction zone composite electrogram; NZeg = normal zone composite electrogram, H = His bundle potential; PI = paced impulse. Time lines are set at 1 sec intervals.
normal zone electrogram (NZeg). Panel A illustrates an escape atrial rhythm at a cycle length of 500 msec. The NZeg was a relatively sharp multiphasic deflection with a duration approximately equal to the QRS duration in the surface lead. On the other hand, the IZeg consisted of a multiphasic deflection, the latter part of which (marked by an arrow) was inscribed during the ST-T segment. As was explained elsewhere, this part reflected delayed activation in the IZ and is referred to in this study as the IZ potential. Exact repetition of the same configuration of the IZ potential in consecutive beats (marked by arrows in panel A) represents a 1:1 conduction pattern in the IZ.

Panels B, C and D were obtained during atrial pacing and illustrate the tachycardia-dependent conduction disorders in the IZ and related RVA. Panel B was recorded during atrial pacing at a cycle length of 395 msec. This cardiac cycle length was still associated with a 1:1 conduction pattern of the IZ potential. A critical narrow range of cardiac cycle lengths of 280–300 msec resulted in a Wenckebach-like conduction pattern of the IZ potential and the occurrence of reentrant beats with extrasystolic grouping (panel C). Analysis of the IZeg shows that the manifest trigeminal rhythm in panel C was related to a 3:2 Wenckebach-like conduction pattern of the IZ potential. The opening beat of the Wenckebach-like cycle was associated with a relatively more synchronized and sharp IZ potential. During the second beat of a 3:2 Wenckebach-like cycle, the IZ potential was replaced by a continuous series of low amplitude multiple asynchronous spikes that bridged the entire diastolic interval between the atrial and reentrant ventricular beats. A large amplitude relatively sharp component of the IZ potential was inscribed during the later part of the ST-T segment (marked by arrows). Panel D shows that further shortening of the cardiac cycle length to 270 msec resulted in a 2:1 conduction pattern of the IZ potential (marked by arrows) and the disappearance of manifest reentry.

Figure 2 represents a continuous tracing and illustrates the effect of V stimulation. During V

**FIGURE 2.** Control recordings from an experiment showing tachycardia-dependent conduction disorders in the infarction zone (IZ) and related reentrant ventricular arrhythmias. Panel A shows simultaneous His bundle and atrial pacing during vagal stimulation. Pacing was abruptly terminated in panel B, revealing atrial standstill and an underlying idioventricular rhythm. Vagal stimulation had no effect on the critical cardiac cycle length associated with Wenckebach-like conduction pattern of the IZ potential and related reentrant rhythm (compare panel A with control recording in fig. 1, panel C). Abbreviations: LAeg = left atrial electrogram; IZ = infarction zone composite electrogram; NZeg = normal zone composite electrogram; PI = paced impulse.
stimulation the ventricular rate was controlled by His bundle pacing. Panel A shows simultaneous His bundle and atrial pacing as revealed in the left atrial electrogram (LAEg). His bundle pacing at a critical cardiac cycle length of 300 msec resulted in a 3:2 Wenckebach-like conduction pattern of the IZ potential, and both manifest and concealed trigeminal rhythm. This was exactly similar to control recordings during atrial pacing at the same cycle length (fig. 1, panel C). Abrupt termination of His bundle pacing while maintaining V stimulation revealed atrial standstill and an underlying idioventricular rhythm (fig. 2, panel B). The slow idioventricular rhythm was associated with a 1:1 conduction of a sharp and only slightly delayed IZ potential (marked by arrows), as well as termination of the reentrant ventricular rhythm seen at relatively fast heart rates.

The effect of V stimulation on conduction of the IZ potential in 10 experiments is summarized in table 1 and diagrammed in figure 3. V stimulation had no significant effect on both the cardiac cycle length at which Wenckebach-like conduction of the IZ potential occurred (P > 0.2) and the range of cardiac cycle lengths associated with the Wenckebach-like conduction pattern (P > 0.1). This reveals that V stimulation had no direct electrophysiologic effect on conduction disorders in the IZ and related RVA. However, the bradycardia associated with V stimulation consistently resulted in improvement of conduction in the IZ with concomitant disappearance of RVA.

**Effect of Sympathetic Stimulation**

Figure 4 illustrates the effect of S stimulation in the same experiment shown in figures 1 and 2. Panel A shows the control atrial rhythm at a cycle length of 445 msec. S stimulation resulted in gradual acceleration of the rate of an AV junctional pacemaker (panels B, C and D). Shortening of the cardiac cycle length was associated with fractionation and delay of the IZ potential. Panel C shows that a cardiac cycle length of 300 msec was associated with a regular 1:1 conduction of a fractionated and delayed IZ potential. This conduction pattern, however, was not associated with manifest reentrant rhythms. By contrast, a similar cardiac cycle length during control recordings in figure 1, panel C was associated with a Wenckebach-like conduction pattern of the IZ potential and a manifest reentrant rhythm. Panel D shows that slight further shortening of the cardiac cycle length to 285 msec did

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**Table 1. Effect of Vagal and Sympathetic Stimulation on Wenckebach-like Conduction of the Infarction Zone Potential**

<table>
<thead>
<tr>
<th>Experiment no.</th>
<th>Control</th>
<th>Vagal stimulation</th>
<th>Sympathetic stimulation</th>
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<tr>
<td></td>
<td>Onset of WC (CCL in msec)</td>
<td>Range of WC (msec)</td>
<td>Onset of WC (CCL in msec)</td>
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<tr>
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<td>275</td>
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Mean ± sd: 326.5 ± 26 (26 ± 3.9) for WC, 327.5 ± 66 (27.5 ± 4.3) for V stimulation, 310 ± 63.8 (24.5 ± 4.4) for sympathetic stimulation. 

Abbreviations: WC = Wenckebach-like conduction of the infarction zone potential; CCL = cardiac cycle length; NS = not significant; S = significant.

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**Figure 3. Diagrammatic illustration of the effect of vagal stimulation on conduction of the infarction zone (IZ) potential in 10 experiments.** Vagal stimulation had no significant effect on the critical range of cardiac cycle lengths (CL) associated with Wenckebach-like conduction pattern of the IZ potential.
result in consecutive 4:3 and 6:5 Wenckebach-like conduction cycles of the IZ potential and a manifest reentrant rhythm.

The effect of S stimulation on conduction of the IZ potential in 10 different experiments is summarized in table 1 and diagrammed in figure 5. S stimulation resulted in a slight shift of the cardiac cycle length at which Wenckebach-like conduction of the IZ potential occurred to relatively short cycle lengths. The difference was statistically significant (P < 0.001). There was, however, no significant change in the width of the zone associated with the Wenckebach-like conduction pattern (P > 0.2). This reflects a slight but consistent improvement of conduction in the IZ following S stimulation. The slight improvement of impulse conduction in the reentrant pathway could result in the disappearance of manifest reentrant rhythms when the cardiac cycle length was kept constant (compare control recording in fig. 1, panel C and the recording in fig. 4, panel C). However, worsening of conduction in the IZ secondary to the significant tachycardia associated with S stimulation, could consistently offset the slight direct improvement of conduction. Thus, the overall effect of S stimulation was a tachycardia-related worsening of conduction in the reentrant pathway with possible induction of manifest reentrant rhythms.

In two experiments, the range of cardiac cycle lengths associated with Wenckebach-like conduction of the IZ potential could not be precisely delineated. Spontaneous reentrant beats were observed during control junctional escape rhythm. Bradycardia induced by V stimulation consistently resulted in improvement of conduction of the IZ potential and the concomitant disappearance of reentrant beats. On the other hand, tachycardia induced by S stimulation resulted in an increased frequency of reentrant beats.

**FIGURE 4.** Recordings obtained from an experiment showing tachycardia-dependent conduction disorders in the infarction zone (IZ) and related reentrant ventricular arrhythmias. Panel A shows an escape atrial rhythm while panels B, C and D were obtained during sympathetic stimulation. Note gradual increase in the rate of an AV junctional pacemaker resulting in tachycardia-dependent worsening of conduction of the IZ potential. The fast junctional rhythm in panel D resulted in Wenckebach-like conduction pattern of the IZ potential and manifest reentrant beats. Abbreviations: Hbeg = His bundle electrogram; IZeg = infarction zone composite electrogram; NZeg = normal zone composite electrogram.

**FIGURE 5.** Diagrammatic illustration of the effect of sympathetic stimulation on conduction of the infarction zone (IZ) potential in 10 experiments. Sympathetic stimulation resulted in a slight shift of the critical range of cardiac cycle lengths (CL) associated with the Wenckebach-like conduction pattern of the IZ potential to relatively short cycle lengths. This reflects a slight improvement of conduction in the IZ.
Discussion

The Autonomic System and Ventricular Arrhythmias

As early as 1859, Einbrodt demonstrated that V stimulation can protect against electrically induced ventricular fibrillation. Later, both experimental and clinical observations showed that V stimulation, as well as vagomimetic drugs, are effective in prevention or suppression of ventricular arrhythmias, while vagotomy and vagolytic drugs may have the opposite effect. A few reports, however, contended that under certain circumstances, V stimulation may also provoke ventricular arrhythmias. Interest in the effect of V stimulation on ventricular arrhythmias was rekindled when Kent et al. demonstrated that V stimulation increased the ventricular fibrillation threshold in acute myocardial ischemia, and the increased vagal tone decreased the incidence of ventricular fibrillation that occurs spontaneously during experimental acute coronary occlusion. These vagal effects were thought to be independent of the bradycardia produced by V stimulation. Some investigators, however, questioned the presence of a direct vagal effect on ventricular vulnerability and suggested that vagal enhancement of ventricular electrical stability is indirect and is achieved by counteracting the effects of heightened sympathetic tone. Other investigators utilizing a similar experimental design to that of Kent et al. failed to illustrate a salutary effect of V stimulation on the ventricular fibrillation threshold in ischemic ventricles. Furthermore, cholinergic innervations of the ventricular conduction system were presumed to mediate the vagal effect. It is well-established, however, that the early phase of ventricular arrhythmias that follows acute myocardial infarction, and can culminate in ventricular fibrillation, is almost exclusively related to electrophysiologic derangement in ischemic myocardium with little if any participation from the specialized conduction system.

In spite of the controversy of the role of V stimulation on ventricular arrhythmias, sympathetic impulses have been firmly implicated in the genesis of ventricular arrhythmias. As early as 1912, Levy first demonstrated that ventricular arrhythmias can be evoked by stimulating certain areas in the hypothalamus. Recent studies by Verrier et al. have shown that posterior hypothalamic stimulation can reduce the ventricular fibrillation threshold secondary to the direct action of sympathetic nerves upon the myocardium. A similar mechanism is thought to explain the effect of psychologic stress in lowering the threshold for repetitive extrasystoles. In the setting of acute myocardial ischemia, S stimulation was shown to facilitate the development of ventricular arrhythmias, whereas sympathectomy or β-adrenergic blockade decreased the incidence of ventricular arrhythmias.

Recently, it became evident that stimulation or ablation of either the right or left stellate innervation to the ventricles can have different effects on the ECG, ventricular refractory periods, ventricular fibrillation threshold, and incidence of ventricular arrhythmias associated with coronary occlusion. In the present study, bilateral stimulation of the sympathetic nerves was designed to obviate the potential difference in the response to unilateral S stimulation as well as to resemble more closely spontaneous episodes of increased cardiac sympathetic neural discharge. Furthermore, previous studies suggest that although the anterior ventricular myocardium is innervated predominantly by fibers arising in the right stellate ganglion, while the posterior ventricular surface is innervated principally by fibers arising in the left stellate ganglion, considerable overlap between right and left sympathetic innervations does exist. In the present study, since the effect of S stimulation on conduction disorders in the IZ produced by ligation of the anterior descending coronary artery was analyzed, bilateral stimulation of sympathetic nerves was necessary to ensure adequate distribution of sympathetic neural discharge in the IZ and adjacent myocardium. A similar approach was recently utilized by Millar et al. in studying the effect of S stimulation on conduction delays induced by acute ligation of the anterior descending coronary artery.

The underlying mechanisms for the arrhythmogenic action of S stimulation are not fully known. Apart from the possible effect of S stimulation in enhancing automaticity of Purkinje fibers, its mechanism of action on the more serious RVA in myocardial ischemia is controversial. A direct electrophysiologic action of adrenergic neurohumoral agents at localized myocardial sites resulting in dispersion of repolarization has been suggested. The disparity of refractoriness was thought to lead to ventricular arrhythmias by a mechanism of focal re-excitation. However, S stimulation results in concomitant heart rate acceleration and systemic pressor responses which can alter cardiac vulnerability. Because of the exquisite rate-dependency of conduction disorders in acutely ischemic myocardium, the tachycardia induced by S stimulation can facilitate reentrant arrhythmias. On the other hand, during acute ischemia both the tachycardia and systemic pressor responses, by virtue of their hemodynamic and metabolic effects, can increase the degree of ischemic injury of the myocardium which would also increase the likelihood of reentrant arrhythmias. Some studies, however, have suggested that the arrhythmogenic effect of S stimulation was independent of accompanying changes in the heart rate and blood pressure. These studies seem to emphasize the sole factor of increased dispersion of refractoriness of the myocardium in the induction of RVA and ventricular fibrillation.

Effect of the Autonomic System in Subacute Ischemic Reentrant Ventricular Arrhythmias

This study has demonstrated that vagal impulses have no significant direct electrophysiologic effect on conduction disorders in ischemic myocardium or related RVA in the late myocardial infarction period.
Stimulation of either vago-sympathetic trunks, especially with low voltage stimuli, was reported to result in stimulation of sympathetic fibers to the heart.\textsuperscript{44-46} This was attributed to either direct stimulation of sympathetic fibers in the vago-sympathetic trunk or to the acetylcholine produced during V stimulation causing the release of norepinephrine.\textsuperscript{48} At least in theory, the lack of vagal effect in our study could be due to a simultaneous excitation of sympathetic fibers. However, this seems unlikely in view of the consistent sinus bradycardia and/or AV block with the escape of slow idioventricular rhythm produced by stimulation of the vago-sympathetic trunks. On the other hand, S stimulation resulted in a direct slight improvement of conduction in the IZ. Because of the marked rate-dependency of conduction in the IZ, both V and S stimulation acted indirectly through modulation of the heart rate. In particular, the propensity of S stimulation to induce reentrant rhythms was primarily due to its tachycardic effect, and in spite of the fact that sympathetic impulses have a direct slight enhancing effect on conduction in the IZ. Recent studies utilizing the same experimental model tend to substantiate these observations.\textsuperscript{47, 48} The \(\beta\)-adrenergic blocking drug, practolol, was found to selectively prolong refractoriness and depress conduction of potentially reentrant pathways in the IZ.\textsuperscript{47} Since practolol did not have direct membrane effects on ischemic myocardial cells,\textsuperscript{49} the effect of the drug in vivo was related to the abolition of the permissive effect of intrinsic catecholamine on conduction in the IZ. Our observations suggest that tonic adrenergic impulses have an enhancing effect on conduction in ischemic myocardium, and that S stimulation could result in slight further improvement of conduction if the heart rate is kept constant. A recent study by Millar et al.\textsuperscript{50} found that S stimulation could improve ventricular conduction delays induced by acute ischemia. This observation, which is similar to our findings in the subacute ischemic stage, lends credence to the concept that the arrhythmogenic effect of S stimulation in acute ischemia may be mediated through its hemodynamic and metabolic effects in spite of a possible direct effect on the improvement of the electrophysiological properties of ischemic myocardium.

In recent years it has become obvious that arrhythmias induced by a critically timed premature stimulus or an increase in the cardiac rate to a critical level, as is the case in the present canine model, can be attributed to either reentry or triggered automaticity.\textsuperscript{49} In the present model, however, ventricular arrhythmias were regularly associated with characteristic patterns of conduction delay in ischemic myocardium and, in particular, with the demonstration of continuous electrical activity that bridged the diastolic interval between the initiating and ectopic beats as well as between consecutive ectopic beats.\textsuperscript{17-19} This seems to be most consistent with a reentrant mechanism. Epicardial mapping of reentrant pathways in the present canine model has been recently accomplished using multiple close bipolar recordings and a multiplexed data recording system.\textsuperscript{50} Although the reentrant origin of ventricular arrhythmias occurring in the present canine model appears to be well-established, the ionic mechanisms underlying conduction delay and block in ischemic myocardial cells needs to be clarified. Our preliminary in vitro observations on depressed ischemic myocardial cells utilizing specific blockers of the Na and Ca currents (tetrodotoxin and verapamil, respectively) suggest that ischemia primarily results in depression of the fast Na channel.\textsuperscript{46, 51, 52} Observations on the different ionic conductance changes produced by ischemia are relevant to the possible mechanisms by which S stimulation may improve conduction in ischemic myocardium. Catecholamine was thought to have no effect on both the rate of rise and the amplitude of the resting potential in normal cardiac muscle cells.\textsuperscript{53} However, it has been known for many years that catecholamine may have a restorative action on depressed membrane potentials.\textsuperscript{54} Early in vivo studies by Wallace and Sarnoff\textsuperscript{55} tend to support these observations. Recent studies by Gelband and associates\textsuperscript{56} have clearly demonstrated that catecholamine may hyperpolarize depressed atrial fibers and thereby improve action potential characteristics and conduction. This action of catecholamine can have an antiarrhythmic effect resulting in abolition of spontaneous ectopic activity. The possibility that catecholamine released by S stimulation in the present study had a restorative action on depressed ischemic myocardial cells forming part of the reentrant pathway seems consistent with these observations. One possible ionic mechanism by which catecholamine may hyperpolarize depressed cardiac cells is stimulation of the myocardial sodium-potassium pump mechanism.\textsuperscript{57}

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