Response of Resistant Ventricular Tachycardia to Bretylium

Relation to Site of Ectopic Focus and Location of Myocardial Disease

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SUMMARY

Ventricular tachycardias were determined to be of either right- or left-sided origin in 25 patients whose arrhythmias were life-threatening and resistant to lidocaine and other antiarrhythmic drugs. All five patients with right ventricular tachycardia responded well to bretylium and survived. Eleven of 20 patients with left ventricular tachycardia did not do well. Four did not respond to bretylium but survived; five had no response and died, and two responded but died when hypotension prevented continued treatment. Eight of these 11 had acute anterior myocardial infarction or ischemia. Of nine patients with left ventricular tachycardia who responded well to bretylium and survived, only two had anterior infarction, and none had anterior ischemia. Because bretylium was efficacious in all patients with right ventricular tachycardia or inferior myocardial infarction in this study, it seems warranted to investigate further the relationship between drug responsiveness and the site of ectopic impulse formation and the location of myocardial disease.

Additional Indexing Words:
Arrhythmias
Inferior myocardial infarction
Anterior myocardial infarction

Bretylium tosylate (bretylium), an investigational adenrenergic blocking drug, gained medical attention as an antihypertensive agent. Its unpredictable gastrointestinal absorption and the rapid development of tolerance, however, made it unsatisfactory for long-term antihypertensive therapy. Leveque first demonstrated the antiarrhythmic properties of bretylium, and subsequent studies by Bacaner showed this drug to be effective in preventing and correcting ventricular arrhythmias in dogs. Further investigation demonstrated the efficacy of bretylium in the management of ventricular premature beats and ventricular tachyarrhythmias in man, including those following acute myocardial infarction and prosthetic valve insertion.

We report here our experience with the use of bretylium in the treatment of ventricular tachycardia in 25 patients in whom this arrhythmia was resistant to lidocaine and other antiarrhythmic drugs. Furthermore, we report observations that suggest a correlation between the efficacy of bretylium, on the one hand, and the site of the ventricular ectopic focus and the location of myocardial disease, on the other.

Material and Methods

Twenty-five patients, 18 men and seven women, with ventricular tachycardia were treated with bretylium (tables 1, 2, and 3). The patients' ages ranged between 30 and 78 years with a mean of 56.8 years. Twenty patients had ischemic heart disease. Of these, 12 developed electrocardiographic changes of a recent myocardial infarction; two had recent ischemia added to old myocardial infarctions; two had only recent ischemia; and four had only old myocardial infarctions. As defined here the term anterior infarction includes anteroseptal, anterior, anterolateral, or lateral myocardial infarction. Of the five remaining patients without ischemic heart disease, one had sarcoid involvement of the lungs and myocardium. Rheumatic heart disease was the primary pathology in two patients, one of whom had undergone operation recently for the insertion of mitral and tricuspid valve prostheses. Lupus erythematosus coexisted with rheumatic mitral insufficiency in one case. One patient had a cardiomyopathy of undetermined etiology. Six patients were taking 0.25 mg of digoxin daily, but none of the ventricular tachycardias could be ascribed to digitalis

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Received August 11, 1972; revision accepted for publication October 2, 1972.

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toxicity. All patients had failed to respond to an intravenous infusion of lidocaine with doses up to 8 mg/min (usually 4 mg/min), supplemented by repeated bolus injections of 1–2 mg/kg body weight. Diphenyhydantoin sodium, quinidine sulfate, procainamide, and propranolol in various combinations had also been used unsuccessfully in all cases.

The initial dose of bretylium was 2.8–12 mg/kg body weight diluted in 50 cc of 5% dextrose in water and given intravenously (i.v.) over a 10–15-min period. The initial dose, or some fraction of it, was repeated every 30–60 min intramuscularly (i.m.) or i.v. until the rhythm disturbance improved or the systolic arterial pressure dropped below 100 mm Hg. When either occurred, treatment intervals were lengthened to every 4–6 hours. Maintenance medication was administered i.m., approximately 16 mg/kg/day. When bretylium was discontinued, oral procainamide was instituted for variable periods of time as a prophylactic agent, usually 2 g/day in divided doses.

We diagnosed the origin of the tachycardia as left ventricular when the QRS complexes displayed terminal S waves in leads I, aV_{1}, or V_{6}, on the one hand, and, on the other, were completely upright, or with large terminal R waves in V_{1} (fig. 1). A right ventricular origin of the tachycardia was indicated by QRS complexes that were mainly or completely upright with no Q waves in the left-sided chest leads, and mainly or completely inverted in V_{1}. Sinus rhythm and right ventricular tachycardia occurring in the same patient (C.N.) are shown in figures 2 and 3.

We made a distinction between “repetitive” and “recurrent” ventricular tachycardia. “Repetitive” ventricular tachycardia (fig. 4) denotes, for our purposes,

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**Figure 1**

Patient R.H. Sinus rhythm (rate 90 beats/min) with premature ventricular beats causing ventricular bigeminy (top) changes to ventricular tachycardia (rate 125 beats/min) with incomplete atrioventricular dissociation of the recurrent type (bottom). Ectopic QRS complexes upright in V_{1}, and with slurred terminal S waves in V_{6} indicate a left ventricular origin. F = ventricular fusion beats.
numerous brief runs of ventricular beats interspersed among sinus beats. Recurrent ventricular tachycardia refers to paroxysmal ventricular tachycardia of longer duration (fig. 1), terminated either by pharmacologic or electrical intervention, and reappearing spontaneously within minutes to hours. In some instances, “repetitive” and “recurrent” ventricular tachycardia occurred in the same patient.

The patient’s response to bretylium was classified into one of the following categories: (1) good response, survived (tables 1 and 2,A) indicates abolition of rapid ventricular tachycardia and survival of the patient for this hospitalization; (2) good response, expired (table 2,B) indicates unresponsiveness of the ventricular tachycardia, with death resulting from arrhythmia.

Twelve-lead electrocardiograms were recorded in all patients at least once before the administration of bretylium and daily during the course of therapy. Patients were continuously monitored, and selected electrocardiographic leads were recorded at least every hour. Complete blood count, urinalysis, BUN, serum creatinine, SGOT, SGPT, LDH, bilirubin, and serum sodium, potassium, chloride, and CO₂ content were determined in all patients before and during bretylium therapy. In some patients, serum magnesium, calcium, phosphorus, and arterial pCO₂, pO₂, and pH were also obtained.

**Figure 2**

Patient C.N. Sinus tachycardia, left atrial enlargement, and left and possibly biventricular hypertrophy. The patient was on no medication at this time and his serum potassium was normal.
**ECG Characteristics**

Right ventricular tachycardia occurred in five patients, and bretylium produced a good response in all of them. The beneficial effects in this group of patients occurred with the first dose, and all survived their hospitalization (table 1). These patients had acute inferior or inferior and anterior myocardial infarction, or conditions that affected the right ventricle, such as mitral plus tricuspid valve replacement (patient S. J.) or sarcoidosis of both ventricles and the lungs (patient C. N., determined at autopsy 6 months later).

Left ventricular tachycardia, seen in a total of 20 patients, responded to bretylium in 11 instances (table 2, A and B). In two of these, however, hypotension supervened so that bretylium could not be continued despite the abolition of ventricular tachycardia, and they died. One of these had sustained an acute anterior myocardial infarction.

Four patients with left ventricular tachycardia did not respond to bretylium but survived (table 3, A). Three of these had acute anterior wall infarction or ischemia. The remaining five patients with left ventricular tachycardia did not respond to bretylium and expired (table 3, B). Four of these
had recent anterior myocardial infarction or ischemia and one had an old anterior infarction.

Thus, nine of 20 patients with left ventricular tachycardia did not respond to bretylium, and two whose arrhythmia did respond went on to die, nevertheless. Eight of these 11 had recent anterior myocardial infarction or ischemia. On the other hand, of nine patients with left ventricular tachycardia who responded to bretylium and survived, only two had a recent anterior wall infarction. The others had acute inferior infarction in three instances, old infarctions in three, or another disease in one.

The rate of ventricular tachycardia in the 25 patients varied between 118 and 214 beats/min, and the width of the QRS complexes during ventricular tachycardia ranged from 0.12 to 0.24 sec. No significant difference was apparent with respect to these variables between right and left ventricular tachycardia or between those patients who showed a response to bretylium and those who did not. In addition, no difference in responsiveness

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**Table 1**

**Right Ventricular Tachycardia: Good Response, Survived**

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age (yr), sex</th>
<th>Wt (kg)</th>
<th>Diagnosis</th>
<th>Predominant rhythm before bretylium</th>
<th>Duration and treatment of arrhythmias before bretylium</th>
<th>Initial dose; cumulative dose to produce response (mg/kg)</th>
<th>Duration of treatment; rhythm after bretylium</th>
</tr>
</thead>
<tbody>
<tr>
<td>W.B.</td>
<td>66</td>
<td>M 95</td>
<td>AMI, inf wall</td>
<td>Recurrent VT</td>
<td>12 hr lid; DCS(M)</td>
<td>8.0; 8.0</td>
<td>7 days; SR</td>
</tr>
<tr>
<td>H.H.</td>
<td>51</td>
<td>M 70</td>
<td>AMI, inf wall</td>
<td>Repetitive VT</td>
<td>1 hr, 45 min lid</td>
<td>4.3; 4.3</td>
<td>7 days; SR</td>
</tr>
<tr>
<td>E.S.</td>
<td>33</td>
<td>M 69.5</td>
<td>AMI, inf and ant intramural</td>
<td>Repetitive VT</td>
<td>7 hr lid; DCS(M)</td>
<td>5.0; 5.0</td>
<td>3 days; SR</td>
</tr>
<tr>
<td>S.J.</td>
<td>52</td>
<td>F 55</td>
<td>RHD, postop mitral and tricuspid valve replacement</td>
<td>Repetitive VT; multiple PVCs; episodes of VF</td>
<td>6 hr lid; DCS</td>
<td>5.4; 5.4</td>
<td>2 days; AF</td>
</tr>
<tr>
<td>C.N.</td>
<td>36</td>
<td>F 55</td>
<td>Myocardial sarcoidosis</td>
<td>Multiple PVCs; recurrent VT</td>
<td>12 hr lid</td>
<td>8.0; 8.0</td>
<td>4 days; SR with PVCs</td>
</tr>
</tbody>
</table>

Abbreviations: AMI = acute myocardial infarction; RHD = rheumatic heart disease; VT = ventricular tachycardia; PVC = premature ventricular contraction; VF = ventricular fibrillation; lid = lidocaine; DCS(M) = direct current countershock (multiple); SR = sinus rhythm; AF = atrial fibrillation.

_Circulation, Volume XLVII, February 1973_
### Table 2

#### Left Ventricular Tachycardia: Good Response

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age (yr), sex</th>
<th>Wt (kg)</th>
<th>Diagnosis</th>
<th>Predominant rhythm before bretylum</th>
<th>Duration of treatment of arrhythmias before bretylum</th>
<th>Initial dose; cumulative dose to produce response (mg/kg)</th>
<th>Duration of treatment; rhythm after bretylum</th>
</tr>
</thead>
<tbody>
<tr>
<td>J.R.</td>
<td>44 M</td>
<td>65</td>
<td>AMI, anterolat wall</td>
<td>Recurrent VT</td>
<td>6 days prop; dil; proc; lid; DCS(M)</td>
<td>7.0; 14.0</td>
<td>9 days; SR</td>
</tr>
<tr>
<td>E.B.</td>
<td>49 M</td>
<td>72</td>
<td>OMI, anterolat wall</td>
<td>Multiple PVCs; repetitive VT</td>
<td>48 hr proc; lid</td>
<td>8.3; 8.3</td>
<td>2 days; SR</td>
</tr>
<tr>
<td>O.S.</td>
<td>62 M</td>
<td>74</td>
<td>OMI, ant wall</td>
<td>Recurrent VT</td>
<td>3 days lid; proc; DCS(M)</td>
<td>5.0; 5.0</td>
<td>3 days; SR</td>
</tr>
<tr>
<td>R.H.</td>
<td>62 M</td>
<td>69</td>
<td>AMI, lat wall</td>
<td>PVCs causing bigeminy; recurrent VT</td>
<td>2 hr, 30 min lid; DCS(M)</td>
<td>5.0; 25.0</td>
<td>6 days; SR</td>
</tr>
<tr>
<td>D.B.</td>
<td>64 M</td>
<td>70</td>
<td>OMI, ant and inf infarction</td>
<td>Repetitive VT</td>
<td>24 hr lid; prop</td>
<td>5.0; 5.0</td>
<td>60 hr; SR</td>
</tr>
<tr>
<td>M.M.</td>
<td>54 F</td>
<td>55</td>
<td>RHD; SLE</td>
<td>PVCs causing bigeminy; repetitive VT</td>
<td>29 hr quin; lid</td>
<td>5.0; 5.0</td>
<td>8 days; SR</td>
</tr>
<tr>
<td>P.F.</td>
<td>63 M</td>
<td>64</td>
<td>AMI, inf</td>
<td>Repetitive VT</td>
<td>2 days quin; lid</td>
<td>6.0; 6.0</td>
<td>18 days; SR and slow VT with few PVCs</td>
</tr>
<tr>
<td>B.L.</td>
<td>56 M</td>
<td>86</td>
<td>AMI, inf</td>
<td>Recurrent VT; multiple PVCs; VF</td>
<td>1 day lid</td>
<td>7.0; 7.0</td>
<td>3 days; SR with PVCs</td>
</tr>
<tr>
<td>B.G.</td>
<td>66 M</td>
<td>85</td>
<td>AMI, inf</td>
<td>Recurrent VT</td>
<td>35 hr proc; lid</td>
<td>3.5; 4.7</td>
<td>18 days; SR with few PVCs</td>
</tr>
</tbody>
</table>

#### B. Expired (developed hypotension)

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age (yr), sex</th>
<th>Wt (kg)</th>
<th>Diagnosis</th>
<th>Predominant rhythm before bretylum</th>
<th>Duration of treatment of arrhythmias before bretylum</th>
<th>Initial dose; cumulative dose to produce response (mg/kg)</th>
<th>Duration of treatment; rhythm after bretylum</th>
</tr>
</thead>
<tbody>
<tr>
<td>E.C.</td>
<td>63 M</td>
<td>70</td>
<td>AMI, anteroseptal; postop laminectomy and orchiectomy; Prostatic carcinoma with metastasis</td>
<td>Recurrent VT</td>
<td>1 hr, 45 min lid; DCS(M)</td>
<td>7.5; 7.5</td>
<td>24 hr; SR with few PVCs</td>
</tr>
<tr>
<td>C.C.</td>
<td>30 F</td>
<td>60</td>
<td>RHD; severe mitral insufficiency; cerebral embolism</td>
<td>Multiple PVCs; single episode of VT</td>
<td>14 hr lid; prop; DCS</td>
<td>7.5; 7.5</td>
<td>3 doses in 3 hr; SR with few PVCs</td>
</tr>
</tbody>
</table>

Abbreviations: OMI = old myocardial infarction; SLE = systemic lupus erythematosus; prop = propranolol; dil = diphenhydantoin; proc = procainamide; other abbreviations as in table 1.
**Table 3**

*Left Ventricular Tachycardia: No Response*

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age (yr), sex</th>
<th>Wt (kg)</th>
<th>Diagnosis</th>
<th>Predominant rhythm before bretylium</th>
<th>Duration and treatment of arrhythmias before bretylium</th>
<th>Initial dose; total dose (mg/kg)</th>
<th>Duration of treatment; rhythm after bretylium</th>
</tr>
</thead>
<tbody>
<tr>
<td>L.B.</td>
<td>74 M</td>
<td>75</td>
<td>AMI, anterolat intramural</td>
<td>Repetitive VT; multiple PVCs</td>
<td>10 hr lid; proc</td>
<td>4.0; 36.0</td>
<td>3 days; VT (responded to dil)</td>
</tr>
<tr>
<td>C.R.</td>
<td>61 M</td>
<td>85</td>
<td>OMI, inf; recent ant ischemia</td>
<td>Recurrent and repetitive VT</td>
<td>2 hr lid; proc; DCS</td>
<td>2.2; 55.0</td>
<td>7 days; VT (responded to ventric pacing)</td>
</tr>
<tr>
<td>C.B.</td>
<td>46 F</td>
<td>52</td>
<td>Cardiomyopathy of undetermined etiology</td>
<td>Repetitive VT; recurrent VF</td>
<td>9 days quin; proc; lid</td>
<td>12.0; 192.0</td>
<td>7 days; VT; VF (responded to prednisone)</td>
</tr>
<tr>
<td>L.S.</td>
<td>58 F</td>
<td>73</td>
<td>Ant ischemia</td>
<td>Repetitive VT</td>
<td>17 days lid; proc; quin; dil; prop</td>
<td>2.8; 90.0</td>
<td>5 days; repetitive VT (no response, discharged with repetitive VT)</td>
</tr>
</tbody>
</table>

A. **Survived**

B. **Expired**

| O.G.| 52 M          | 70      | AMI, ant wall                | Recurrent VT                        | 24 hr lid; prop; DCS(M)                             | 5.0; 21.0                     | 3 doses in 2 hr; VT; VF                      |
| H.C.| 78 M          | 70      | Ant wall ischemia            | Recurrent VT                        | 48 hr lid; quin; DCS(M)                              | 5.0; 75.0                     | 3 days, then 2 hr; SR; PVCs; VT; VF          |
| M.T.| 77 F          | 55      | OMI, anteroseptal wall       | Multiple PVCs; repetitive VT         | 48 hr lid                                           | 5.0; 75.0                     | 4 days; VT; VF                               |
| M.G.| 58 M          | 78      | OMI, anteroseptal; recent anterolat ischemia | Repetitive VT; VF | 20 hr lid | 3.8; 45.5 | 3 days; VF |
| G.G.| 65 M          | 115     | AMI, anterolat              | Recurrent VT; VF                    | 17 min lid; DCS                                     | 5.2; 120.0                    | 4 days; VT; VF                               |

Abbreviations as in tables 1 and 2.
to bretylium was observed between patients with recurrent and patients with repetitive ventricular tachycardia.

The sinus rate after successful treatment with bretylium was lower than in the period before treatment, often after an initial increase. Patient S. J. had atrial fibrillation before and after the development of the ventricular arrhythmias, with no significant change in ventricular rate as a result of bretylium treatment. During the administration of bretylium, the nonresponders showed no significant change in rate of ventricular tachycardia, except for patient P. F. who was considered to have had a good response to bretylium when rapid ventricular tachycardia changed to sinus rhythm interspersed with occasional episodes of a ventricular rhythm at a rate of 64. Bretylium did not produce alterations in the contour of the scalar electrocardiograms except for the changes in QRS complexes resulting from the disappearance of ventricular tachycardia.

Responders and nonresponders to bretylium were equally divided among the six patients taking digoxin. No adverse effects were recognized when bretylium was given in the presence of digoxin.

**Dosage**

The duration of the ventricular tachycardias before the institution of bretylium ranged from 75 min to 18 days in the 16 responders, and from 2 hours to 17 days in the nine nonresponders. Twelve patients responded to bretylium beginning with the initial dose; four others responded only after additional doses. The cumulative dose required to produce a favorable response in the 16 patients ranged from 4.3 to 25 mg/kg body weight, the latter within a 6-hour period. The cumulative dose of bretylium given the patients who failed to receive a beneficial effect ranged from 21 to 192 mg/kg, given within 2 hours to 7 days, respectively.

**Side Effects**

The arterial pressure increased in four patients and decreased in 21. Bretylium had to be discontinued in two instances (patients E. C. and C. C.) because the fall in arterial pressure could not be reversed by administration of fluid or vasopressor drugs, including norepinephrine. Otherwise, symptoms attributed to hypotension occurred only with abrupt changes in posture. Six patients experienced a drop in recumbent systolic arterial pressure below 100 mm Hg while taking the drug. Nausea and vomiting were occasionally present after i.v. but not after im administration. Parotid pain was not produced by the administration of bretylium nor did unexpected changes occur in the laboratory data studied. Patients who had sustained a recent myocardial infarction showed characteristic elevations of serum enzymes.

**Discussion**

Although bretylium has been shown to be effective in the control of life-threatening ventricular arrhythmias, our study further defines those clinical situations most likely to respond to this medication. Thus, in our patients whose arrhythmias were potentially lethal and resistant to lidocaine and other antiarrhythmic drugs, we analyzed the electrocardiogram to determine the major site of myocardial disease and to define whether the arrhythmia originated from a focus in the right or left ventricle.

Right ventricular tachycardia was associated with inferior myocardial infarction or right ventricular myocardial disease, and a good response to bretylium. Patients with left ventricular tachycardia in the presence of only inferior infarction also responded to bretylium and survived. On the other hand, left ventricular tachycardia that was associated mainly with anterior infarction, mitral regurgitation, and cardiomyopathy exhibited relatively poor responsiveness and a low survival rate.

In our patients, therefore, the site of the ectopic focus during ventricular tachycardia or the location of the myocardial disease, or both, appeared to bear a significant relationship to the patient's course. Since no cases of anterior infarction were associated with right ventricular tachycardia, our data are also compatible with the postulation that the site of infarction determined both the types of tachycardia and the response to bretylium.

Many hemodynamic events have previously been investigated in patients with myocardial infarction. The changes in certain ones such as prejection period and left ventricular ejection time and the development of cardiogenic shock have not been dependent upon the location of infarction.

In contrast, the origin of ectopic ventricular beats has been shown previously to be associated with the location of myocardial infarction, and the location of infarction has been related to the incidence of life-threatening ventricular arrhythmias. Benignity of right ventricular ectopic beats as compared to left-sided ones has been suggested in the past. These differences in prognosis, taken together with the apparent differences in the responsiveness to
bretylium, may reflect an underlying physiologic difference between functions of right- and left-sided specific myocardial tissue. It has been shown, for example, that digitalis increases the automaticity of left-sided Purkinje tissue preferentially, compared to that from the right side.

The failure of bretylium to reverse experimentally produced ventricular tachycardia noted by Allen et al. may be related to the fact that they ligated the left anterior descending coronary artery in dogs. Such ligation presumably caused anterior myocardial infarction; they failed to investigate the responsiveness of ventricular tachycardia to bretylium therapy when other coronary arteries were obstructed experimentally.

The mechanism of the antiarrhythmic action of bretylium is unclear. Whereas procainamide, quindine sulfate, diphenhydantoin, propranolol, and lidocaine depress automaticity and significantly prolong the effective refractory period of Purkinje fibers relative to the action-potential duration, bretylium does not produce the same effects. Although it prolongs the action-potential duration, it lengthens the refractory period proportionately, without slowing conduction. Bretylium has been shown to cause a prompt increase in resting transmembrane voltage and, consequently, a rise in amplitude of phase 0, as well as the maximum rate of depolarization ($V_{\text{max}}$). These changes would tend to abolish reentrant arrhythmias by altering the wavelength in the reentry pathway. Although bretylium initially releases norepinephrine, it then blocks the further release of norepinephrine, which may contribute to the antiarrhythmic action of this drug, though contrary evidence has been reported.

Hypotension noted in patients receiving bretylium can be attributed to the adrenergic blocking action of the drug. This effect can usually be controlled with volume replacement and vasoactive amines. These patients are sensitive to circulating catecholamines, perhaps because bretylium inhibits monoamine oxidase activity or because it blocks uptake of catecholamines into adrenergic neurons. Norepinephrine is therefore usually effective in reversing hypotension produced by bretylium. The increase in arterial pressure encountered in some of our patients may be explained by the initial release of norepinephrine from the sympathetic nerve endings before it manifests its blocking action.

In our experience, factors that were not related to the patients' response to bretylium included: (1) duration of the tachyarrhythmia before administration of the drug, and (2) the width and rate of the ectopic QRS complexes during the ventricular tachycardia.

Our studies do not allow us to distinguish between ventricular tachycardias resulting from rapid diastolic depolarization versus those resulting from a reentry mechanism. Yet, regardless of the underlying mechanism, this study suggests that ventricular tachycardia apparently originating in the right ventricle or associated with inferior infarction responds well to bretylium; left ventricular tachycardia associated with acute anterior myocardial ischemia or infarction does not. In our view, the question of the relationship of drug response to the site of ectopic impulse formation and to the location of myocardial disease merits further clinical and experimental investigation.

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Circulation, Volume XLVII, February 1973
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doi: 10.1161/01.CIR.47.2.331

*Circulation* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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