Left Stellectomy in the Prevention of Ventricular Fibrillation Caused by Acute Myocardial Ischemia in Conscious Dogs with Anterior Myocardial Infarction

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SUMMARY The potential of left stellectomy in reducing the incidence of ventricular fibrillation associated with acute myocardial ischemia was investigated in a new animal model for sudden death. Thirty-two dogs had an anterior myocardial infarction produced by ligation of the left descending coronary artery. One week later they were randomly allocated to a control or to an experimental group that underwent left stellectomy. One month after ligation while the dogs were conscious, a balloon occluder previously positioned around the circumflex coronary artery was inflated and the ensuing coronary occlusion was maintained for 10 minutes. Ventricular fibrillation occurred in 11 of 17 (65%) control dogs, compared with five of 15 (33%) \((p < 0.05)\) in the experimental group. Among the survivors the incidence of arrhythmias was less in the experimental group compared with the control group. Infarct size \((21 \pm 2\% \text{ vs } 20 \pm 2\%)\), resting heart rate \((143 \text{ beats/min vs } 127 \text{ beats/min})\) and \(QT_c\) \((347 \pm 11 \text{ msec vs } 349 \pm 13 \text{ msec})\) were similar between control and experimental groups. The dogs that died had a greater increase in heart rate at 1 minute postocclusion than survivors and also had significantly longer QT intervals.

Our study indicates that left stellectomy exerts a major protective effect in reducing the incidence of ventricular fibrillation when conscious dogs with a previous anterior myocardial infarction undergo acute myocardial ischemia. This simple and safe surgical procedure may be considered for a clinical trial in subgroups of patients with ischemic heart disease at very high risk for sudden death.

SUDDEN DEATH is most often caused by ventricular fibrillation.\(^1\) The importance of neural mechanisms, particularly the sympathetic nervous system, in precipitating ventricular fibrillation has been shown experimentally, confirmed clinically and is generally accepted.\(^2,7\) Therefore, a rational approach toward the prevention of sudden death is represented by interventions that increase electrical stability and interfere with the effects of increases in sympathetic activity. We propose that left stellectomy may be one of these interventions.

We have shown that ablation of the left stellate ganglion results in a reduced incidence of arrhythmias either directly or indirectly, by changes in hemodynamic variables relevant to the genesis of cardiac arrhythmias. Briefly, left stellectomy reduces the incidence of arrhythmias associated with transient coronary occlusion,\(^8\) markedly increases the threshold for ventricular fibrillation,\(^9\) prolongs the ventricular refractory period,\(^10\) and increases the capability of the coronary bed to dilate at rest\(^11\) and during exercise.\(^12\)

Two considerations have guided the design of our experimental model: (1) Patients with a previous anterior myocardial infarction, when challenged by a new episode of acute ischemia, represent one of the groups at very high risk for sudden death. (2) If the results are to be transposed into the clinical setting, the experimental intervention has to be appropriately timed in consideration of the two cardiac events that created the risk factor and that may induce sudden death.

Therefore, we studied the incidence of ventricular fibrillation during acute myocardial ischemia in two groups of conscious dogs in which an anterior myocardial infarction had been produced 4 weeks earlier; the experimental group of dogs had a left stellectomy performed 1 week after the myocardial infarction. Preliminary results have been reported.\(^13,14\)

Methods

Surgical Procedure and Protocol

Sixty-four mongrel dogs, ranging in weight from 17–25 kg were anesthetized with sodium pentothal \((30 \text{ mg/kg i.v.) and intubated. The level of anesthesia was maintained with a mixture of oxygen, nitrous oxide and halothane. The heart was exposed through the fourth intercostal space. The left circumflex and descending coronary arteries were dissected free from surrounding tissue for approximately 1.0 cm, carefully avoiding damage to the pericoronary nerves. A hydraulic balloon occluder was positioned around the circumflex coronary artery and secured in place. A catheter attached to the balloon was brought out of the chest and tunneled to the dorsal surface of the neck where it exited from the skin.\]

The left stellate ganglion, the ansa subclavia and the rami communicantes from \(T_1\) to \(T_4\) were identified. A nylon monofilament suture was placed around the caudal portion of the ganglion near the point where the \(T_3\) ramus enters the sympathetic chain and a second suture was placed around the ansa subclavia at the
cranial portion of the ganglion. Both sutures were buried subcutaneously near the vertebrae on the left side. Care was taken to make sure that the sutures remained loose around the ganglion to prevent undue tension.

The left descending coronary artery was occluded, by means of the Harris two-stage procedure, approximately 1.5–2.0 cm from its origin. The ECG was monitored before and after occlusion for 1 hour. If too many ventricular ectopic complexes occurred, lidocaine (5 mg i.v.) was usually given. In the last group of dogs, we also often applied lidocaine topically on the left stellate ganglion. One hour after coronary artery occlusion the chest was closed and the dogs were returned to their cages.

The dogs alive 1 week after the production of the anterior myocardial infarction were randomly allocated to an experimental (LSGx) group or to a control group. The dogs in the experimental group were lightly anesthetized with sodium pentothal (30 mg/kg i.v.), and through an incision in the skin, the nylon sutures passed around the left stellate ganglion were suddenly pulled, resulting in the destruction of the ganglion itself. The final part of the study was performed 3–4 weeks after myocardial infarction was produced. The dogs were conscious while the balloon occluder positioned around the circumflex coronary artery was inflated, producing acute ischemia. The occlusion was maintained for 10 minutes and then released.

A postmortem examination was performed in all dogs. The heart was removed and the clearly demarcated infarct was excised from normal tissue, weighed and expressed as a percentage of the total heart weight.

Measurements

The ECG (lead 2) was recorded on an eight-channel Beckman direct-writing oscillograph and was used to trigger a cardiomultiplet for measurement of the heart rate. The signals were also recorded on a magnetic tape (Ampex FR-1300) for later analysis.

The QT interval was measured and corrected for heart rate using the classic Bazett's formula \( QT_c = QT / \sqrt{RR} \). QT was calculated from five cardiac cycles in each dog and a mean value was then obtained. The data were analyzed by means of Student's paired- and unpaired-sample t-tests and by chi-square test. All values are expressed as mean ± SEM.

### Results

An anterior myocardial infarction was produced in 64 dogs. Thirteen dogs died on the surgical table within 2 hours of total occlusion of the anterior descending coronary artery. Thirteen dogs died postoperatively within 1 week after surgery; they were all found dead in the cage and were, with one exception, in fair condition the day before their death. One dog died just after induction of anesthesia with sodium pentothal when the sutures around the left stellate ganglion had to be pulled. Five dogs were eliminated from the study, three because of a small infarct (<10% of total heart weight), one because the balloon occluder around the circumflex artery ruptured at the time of occlusion and one because of persistent ventricular tachycardia up to 46 days after myocardial infarction.

Thus, the data were obtained from 32 dogs (50% of the initial number) that met our criteria; 17 were in the control group and 15 in the LSGx group. The overall data are shown in table 1.

### Table 1. Summary of Data in 32 Dogs with Anterior Myocardial Infarction

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>VF (%)</th>
<th>Mortality</th>
<th>Infarct size</th>
<th>Initial HR (beats/min)</th>
<th>HR at 1 min. postocclusion</th>
<th>QTc (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controls</td>
<td>17</td>
<td>11 (65%)</td>
<td>12 (71%)</td>
<td>21 ± 2%</td>
<td>143 ± 8</td>
<td>201 ± 9</td>
<td>347 ± 11</td>
</tr>
<tr>
<td>LSGx</td>
<td>15</td>
<td>5 (33%)</td>
<td>6 (40%)</td>
<td>20 ± 2%</td>
<td>127 ± 6</td>
<td>193 ± 7</td>
<td>349 ± 19</td>
</tr>
<tr>
<td>Survivors</td>
<td>14</td>
<td>—</td>
<td>—</td>
<td>18 ± 2%</td>
<td>130 ± 5</td>
<td>178 ± 5</td>
<td>318 ± 13</td>
</tr>
<tr>
<td>Deceased</td>
<td>18</td>
<td>87%</td>
<td>100%</td>
<td>22 ± 1%</td>
<td>139 ± 8</td>
<td>210 ± 7</td>
<td>371 ± 13</td>
</tr>
<tr>
<td>C surv</td>
<td>5</td>
<td>—</td>
<td>—</td>
<td>18 ± 5%</td>
<td>126 ± 9</td>
<td>172 ± 14</td>
<td>311 ± 14</td>
</tr>
<tr>
<td>C dec</td>
<td>12</td>
<td>92%</td>
<td>100%</td>
<td>22 ± 2%</td>
<td>149 ± 10</td>
<td>210 ± 9</td>
<td>363 ± 12</td>
</tr>
<tr>
<td>LSGx surv</td>
<td>9</td>
<td>—</td>
<td>—</td>
<td>18 ± 3%</td>
<td>132 ± 7</td>
<td>181 ± 4</td>
<td>322 ± 20</td>
</tr>
<tr>
<td>LSGx dec</td>
<td>6</td>
<td>83%</td>
<td>100%</td>
<td>22 ± 1%</td>
<td>118 ± 8</td>
<td>211 ± 14</td>
<td>356 ± 31</td>
</tr>
</tbody>
</table>

* \( p < 0.05 \)

** ** \( p < 0.01 \)

Abbreviations: C = controls; surv = survived; dec = deceased; LSGx = left stellatectomy; VF = ventricular fibrillation.
Arrhythmias

Before the 10-minute occlusion of the circumflex artery, the dogs were quietly lying on the table; their heart rates, however, were higher than those in conscious dogs without myocardial infarction. When the circumflex coronary artery was occluded, heart rate increased rapidly and 18 of 32 dogs (56%) died, almost always as a consequence of ventricular fibrillation, usually within 4 minutes. Figure 1 shows two examples of the electrocardiographic changes that immediately followed the occlusion in two control dogs that died in ventricular fibrillation.

Ventricular fibrillation occurred in 11 of 17 (65%) control dogs and in five of 15 (33%) left stellectomized dogs ($p < 0.05$). Cardiac arrest caused by asystole occurred once in both groups (7%). Thus, the overall mortality rate was 12 of 17 (71%) in the control group and six of 15 (40%) in the left stellectomized group ($p < 0.05$).

Ventricular arrhythmias were very frequent after complete ligation of the left descending coronary artery and several dogs died in this early phase. Intravenous lidocaine was usually used in the attempt to prevent ventricular fibrillation, but with variable success. In four dogs with life-threatening arrhythmias resistant to 5 mg i.v. lidocaine, we applied lidocaine topically on the left stellate ganglion. In all of them the arrhythmias ceased completely within 1 minute and did not recur during the next 30-40 minutes.

The short-acting anesthetic (sodium pentothal) used for the left stellectomy was often associated with arrhythmias, most often with ventricular bigeminy. Prompt performance of the left stellectomy by means of traction on the ligature immediately abolished ventricular bigeminy and sinus rhythm returned in all but one instance. In this case, there was delay in finding the ligature and the dog died in ventricular fibrillation (fig. 2).

Premature ventricular complexes (PVCs) were counted in the dogs that survived the 10-minute occlusion of the circumflex coronary artery (fig. 3). In control dogs, most PVCs occurred between the second and fourth minutes, and a second surge of arrhythmias appeared during the last minute of occlusion. In contrast, left stellectomized dogs had fewer PVCs.
Figure 2. Three dogs with a 1-week old anterior myocardial infarction, 1 minute after induction of anesthesia with sodium pentothal. Ventricular bigeminy is evident in all three dogs. The arrows indicate the moment when the ligatures placed around the stellate ganglion were suddenly pulled, resulting in a left stellectomy. (A) is few seconds before (B). (C) Just after left stellectomy, a short run of atrioventricular nodal tachycardia is evident. (D) Before the ligatures buried under the skin could be identified and left stellectomy performed, ventricular fibrillation occurred.

Figure 3. Number of premature ventricular complexes (PVBs) occurring during each of the 10 minutes of occlusion of the circumflex coronary artery among the five controls and the nine experimental dogs that survived.
throughout the entire occlusion ($p < 0.05$). The comparison was made only in the surviving dogs, so the greater incidence of deaths in the control group does not affect this observation. Thus, left stellectomy reduced the incidence not only of ventricular fibrillation, but also of PVCs in the dogs that survived the 10 minutes of circumflex coronary artery occlusion.

Infarct Size

Infarct size was similar in the two groups, $21 \pm 2\%$ in the control group and $20 \pm 2\%$ in the LSGx group. The difference in infarct size between the survivors ($18 \pm 2\%$) and nonsurvivors ($22\% \pm 1$) was not significant.

Heart Rate

Heart rate before occlusion of the circumflex coronary artery was higher in the control group than in the experimental group ($143 \pm 8$ beats/min vs $127 \pm 5$ beats/min) (NS). Heart rate in the dogs with myocardial infarction was considerably higher than that observed in the same laboratory in previous studies of normal conscious dogs ($91 \pm 7$ beats/min).

At 1 minute after the beginning of occlusion, heart rate increased significantly ($p < 0.001$) in both groups ($201 \pm 9$ beats/min in the control group and $193 \pm 7$ beats/min in the LSGx group). This is at variance with that observed by Thames et al., in anesthetized dogs without myocardial infarction. A large and significant difference ($p < 0.05$) was found when the

![Figure 4](http://circ.ahajournals.org/)

**Figure 4.** Control dog 4 weeks after the production of an anterior myocardial infarction just before occlusion of the circumflex coronary artery. (B) Three minutes after occlusion the heart rate (HR) markedly decreased. No arrhythmias occurred and the dog survived. (C) Two days later, under the same conditions as in (A). (D) Two minutes later the HR once again was clearly reduced; few premature ventricular complexes occurred, but the dog survived the 10 minutes of occlusion. (E) Two days later, in the same condition as (A) and (C), but after atropine (200 $\mu$g/kg I.v.), the heart rate is markedly elevated. (F) Marked ischemic changes are evident within 30 seconds of occlusion. (G) At 2 minutes of occlusion, runs of ventricular tachycardia appear. (H) At 8 minutes of occlusion the heart rate increased and ventricular fibrillation ensued.
changes in heart rate in the first minute of occlusion were compared between survivors (from 130 ± 5 to 178 ± 5 beats/min) and nonsurvivors (from 139 ± 8 to 210 ± 7 beats/min). The heart rate in the dogs that died reached the same level in both groups (210 ± 9 in the control group and 211 ± 14 beats/min in the LSGx group). Detailed information on heart rate before and at 1 minute of occlusion is presented in table 1.

In four dogs, the heart rate decreased after the occlusion of the circumflex artery. Two of these dogs died in asystole and two survived. (The figures on heart rate reported above do not include them.) Figure 4 shows the relationship between vagal reflexes, heart rate and survival in one of the latter two. This dog survived two occlusions that lasted 10 minutes each, always associated with a decreased heart rate; after pretreatment with atropine the coupling of myocardial ischemia with increased heart rate resulted in ventricular fibrillation.

**Discussion**

For almost 50 years, the possibility of improving survival after acute myocardial infarction was produced. Therefore, to have reference values for this study, QTc was calculated in 10 conscious intact dogs enrolled in a different investigation and was found to be 304 ± 9 msec. An upper limit of normal values in dogs was tentatively determined using the same arbitrary criterion usually accepted in man (mean ± 1½ SD). Because the SD was 30 msec, we arbitrarily decided to use 350 msec as the upper limit for normal values in dogs. In this series of experiments the QTc was measured just before the occlusion of the circumflex artery, so the values reported are those found in conscious dogs with a prior anterior myocardial infarction. Because of either a flat T wave or noise in the ECG signal, the QT interval could not be measured in four control dogs and three LSGx dogs (fig. 5).

The data for all groups are reported in table 1. Our method of defining infarct size may underestimate the true extent of myocardial cell damage, but at a minimum defines the necrotic area. With these limitations in mind, the value of QTc was independent of infarct size as shown by the poor correlation (r = 0.24) between these two variables.

Using 350 msec as the upper limit of normal and lumping together controls and LSGx dogs, 83% of the dogs with a QTc longer than 350 msec died; 100% of control dogs with a prolonged QTc died, but only 60% of the LSGx dogs died.

**FIGURE 5. Comparison of mean QTc value (mean ± SEM) between the groups.** The horizontal line at 350 msec represents the upper limit of normal values of QTc for conscious dogs (see Results). The first bar indicates the QTc of 10 normal intact dogs. The second and third bars show the average value of QTc among the controls (n = 13) and the experimental (LSGx) (n = 12) groups, 4 weeks after myocardial infarction (M1) and just before the occlusion of the circumflex coronary artery; in seven dogs QTc could not be properly measured (see Methods); it is evident that no difference exists between the two groups. The fourth and fifth bars show that a significant (p < 0.01) difference exists when the QTc of the survivors is compared with that of the dogs that died. The last two columns show the individual data of QTc for the control and experimental groups (open circles = survivors; closed circles = deceased) and indicate that more extreme values were present among the experimental dogs and that dogs with QTc greater than 350 msec had 83% mortality, compared with 31% in dogs with a normal QTc.
clinical interest in sympathectomy, usually performed to relieve anginal pain, has progressively decreased, for two main reasons: (1) bilateral thoracic sympathectomy constitutes rather extensive surgery; and (2) the advent of β-adrenergic blocking agents, which were thought to duplicate the effects of surgical sympathectomy, seemed to supersede surgical denervation.

Recent experimental evidence, however, has shifted the focus of interest from bilateral thoracic sympathectomy to unilateral left stellectomy.

Critique of the Experimental Model

Large differences still exist between the experimental models for sudden cardiac death and the clinical situation; a major one is that the diseased, largely atherosclerotic and ischemic hearts of most patients who die suddenly37, 38 are not comparable to the intact hearts of the animals used in laboratory experiments. Previous studies, although often very carefully conducted36 or analyzed,21, 22 suffer because coronary artery occlusion was performed (1) during anesthesia, (2) at the same time or too shortly after denervation, (3) in otherwise completely intact hearts, and/or (4) after a complete, and therefore clinically unrealistic, denervation.

Our interest in the potential clinical importance of our results guided the design of our protocol. The experiments had to be performed in conscious animals. To study the effects of denervation in a heart that was completely normal until the very moment of coronary occlusion is of modest clinical interest because a surgical denervation would, of course, be inconceivable in patients with a normal heart, but may be considered for patients at high risk of sudden death. Patients with anterior myocardial infarction constitute one of these high-risk groups, so we decided to study dogs with a previous anterior myocardial infarction during an episode of acute ischemia. Furthermore, a patient would be considered for surgery only some time after the myocardial infarction, yet early enough to protect him from life-threatening arrhythmias that are more likely to occur during the first 6 months after discharge from the hospital; accordingly, we scheduled the left stellectomy 1 week after infarction and the episode of acute ischemia 3 weeks later. The duration of the occlusion of the circumflex coronary artery, (10 minutes) was decided arbitrarily in an attempt to simulate the occurrence of a major ischemic insult; we realize, however, that this 10-minute occlusion, coupled with the previous ligature of the left anterior descending coronary artery, may have been overwhelming for many dogs. This may have affected our results by decreasing the relative importance of the sympathetic nervous system compared with pump function; if so, our data may actually underestimate the protective effect of left stellectomy.

Sudden Death

The high mortality (71%), mostly the result of ventricular fibrillation (65%), indicates that our model is indeed very effective in producing sudden cardiac death in control dogs.

Our main finding is that left stellectomy, performed 1 week after an anterior myocardial infarction and 3 weeks before a 10-minute occlusion of the left circumflex coronary artery, significantly reduces the incidence of ventricular fibrillation in conscious dogs. The minor difference in heart rate before the occlusion and the similar infarct size stress the fact that the only important difference between the two groups was the presence or absence of the left stellate ganglion.

Arrhythmias

Among the survivors, the control dogs had more arrhythmias than the LSGx dogs, again confirming the antiarrhythmic effect of left stellectomy.9 The burst of arrhythmias during the last minute of occlusion in the control dogs suggests that in this group the mortality might have been higher if the occlusion was maintained for 1–2 more minutes; in contrast, in the LSGx group the few PVCs during the first part of the occlusion had almost completely subsided by the seventh minute.

The observation that topical application of lidocaine on the left stellate ganglion suppressed, within seconds, ventricular arrhythmias resistant to full doses of lidocaine given intravenously, points again to the critical role played by the nerve fibers traversing the left stellate ganglion in the genesis of the ventricular arrhythmias that occur during the early phase of myocardial ischemia. In 1955, Milch et al.22 found that the incidence of sudden death after ligation of the left anterior descending coronary artery in anesthetized dogs was reduced by bilateral thoracic sympathetic ganglionectomy to 39%, (compared with 62% in control dogs), and to 16% by injecting procaine in the left stellate ganglion.

Heart Rate

High heart rates in the early stage of acute myocardial ischemia may precipitate ventricular fibrillation.29-31 The sympathetic control of heart rate is primarily exerted by the right stellate ganglion12, 32 and important differences between control and LSGx dogs would not be expected. The resting heart rates were indeed similar and did not seem to play an important role, being slightly higher (16 beats/min) in the controls compared with the LSGx dogs. The main and most significant difference was the peak value reached at the first minute of occlusion in the dogs that survived compared with those that did not. The fact that the dogs that died reached the same heart rate at 1 minute postocclusion (210 beats/min), irrespective of whether they belonged to control or LSGx groups, suggests that very high heart rates may overcome the protective effect of left stellectomy. Hope et al.33 similarly showed that the antiarrhythmic effect of propranolol was lost if the hearts of dogs with coronary artery occlusion were paced to 184 beats/min. However, major and dangerous increases in rate occurred much more frequently among control dogs. Why left stellectomized dogs had a reduced tendency to increase heart rate to a major extent during acute ischemia is not clear, but a tentative explanation is
possible. The heart rate response to ischemia is complex and depends on many factors, one of them being a component of the cardio-cardiac sympathetic reflex with the efferent limb mostly through the right stellate ganglion and the afferent limb probably through both right and left sympathetic afferent fibers. Left stellate ganglion would eliminate part of the afferent information, thus limiting the full expression of this reflex.

In dogs with an intact heart, occlusion of the circumflex coronary artery is often associated with bradyarrhythmia, dependent upon activation of vagal afferent fibers. In only four of our dogs with a previous myocardial infarction did heart rate decrease. The role of vagal reflexes during acute myocardial ischemia is controversial and both protective and detrimental effects have been claimed. Some enhancement in vagal tone may be beneficial by preventing excessive tachycardia, thus preserving underperfusion tissue from advancing ischemia. However, when the increase in vagal tone is excessive, it may produce hypotension, further reducing coronary flow to the ischemic areas and eventually resulting in either ventricular fibrillation or asystole. Why some dogs show such an overwhelming vagal activity is still unclear. Yet, it seems reasonable to postulate that the critical factor may be the existing level of sympathetic tone: if it is high enough, the vagal reflex may prevent excessively high heart rates without the detrimental effects that occur if the increased vagal activity is left completely unopposed.

From what is known about the neural pathways through which the sympathetic nervous system exerts its control on heart rate, it is evident that the sympathetic nerves that are critically important in counteracting vagal reflexes in the setting of acute ischemia are those that originate from the right stellate ganglion. Their function would be annulled by bilateral stellatectomy or β-adrenergic blocking agents but would remain unimpaired after left stellatectomy. The balance between sympathetic and parasympathetic efferent activity may be a key to survival during acute myocardial ischemia.

**Rationale for Left Stellatectomy**

The rationale for evaluating the potential of left stellatectomy in reducing the incidence of sudden death due to ventricular fibrillation lies mainly in the notions concerning (1) the sympathetic reflexes elicited by ischemia, (2) the dominant role played by left-sided nerves in the genesis of life-threatening arrhythmias, and (3) some recent findings related to the neural control of coronary circulation.

Cardiac sympathetic sensory endings with myelinated and unmyelinated fibers are excited by ischemia and are capable of eliciting a cardio-cardiac sympathetic reflex. This sympathetic reflex, which takes place within a few seconds after the onset of ischemia, plays an important role in the genesis of the early ventricular arrhythmias. The excitation of cardiac sympathetic afferent fibers can also reflexly inhibit the activity of efferent vagal cardiac fibers, with the potential of impairing the maintenance of an optimal heart rate, thus facilitating the occurrence of a dangerous tachycardia. The afferent limbs of most cardio-cardiac sympathetic reflexes seem to be preferentially distributed through left-sided nerves, which makes these reflexes dependent to a major extent on an intact left stellate ganglion.

As indicated by prior work, left stellatectomy would not only reduce the effects of the tonic sympathetic effects on the heart, but would also largely prevent the occurrence of cardiac sympathetic reflexes.

Recent studies dealing with unilateral stellatectomy have shown some unexpected results and have indicated that left-sided sympathetic nerves play an unexpected role in the genesis of life-threatening arrhythmias. When left cardiac sympathetic activity is potentiated by right sympathectomy and a condition of imbalance with left dominance is created, the heart becomes more vulnerable to ventricular arrhythmias, during control states ischemia, or psychologic stress or exercise.

**QT Interval**

In a study among patients with a myocardial infarction who had an ECG recorded every 2 months for 7 years, prolongation of the QT interval (QTc > 440 msec) was found to be more frequent (57% vs 18%) among those who died compared with those who survived, and 77% of patients with prolonged QT interval died suddenly. These findings have been confirmed.

As in humans, dogs with myocardial infarction had a greater QTc than control dogs; the most interesting finding, however, was that the QTc was remarkably longer in dogs that died than in dogs that survived, as in the clinical study. We have no ready explanation for the cause of QT prolongation. It is tempting to speculate that, because right sympathetic nerves are mainly distributed to the anterior ventricular surface, an anterior myocardial infarction would damage or destroy adrenergic terminals mostly innervated by right-sided nerves, thus mimicking, to some extent, the results of right stellatectomy, which is associated with prolongation of the QT interval and ventricular arrhythmias. The dogs with the greater involvement of right-sided neural terminals would be those with the longer QT and the greater vulnerability to ventricular fibrillation.
of the border zone, and decrease the potential for life-
threatening arrhythmias, and may contribute to the
preservation of ventricular function.

Left stellectomy, probably because it does not produce a major catecholamine depletion, is not followed by post-denervation supersensitivity.

Clinical Implications

Although experimental results must be interpreted cautiously, this study suggests that left stellectomy (in man, high thoracic left sympathectomy) may reduce the incidence of sudden cardiac death in patients with ischemic heart disease. This procedure, which is performed extrapleurally and carries practically no risk, has been very successfully used in 23 patients affected by the long QT syndrome who were refractory to medical treatment. It is relevant that cardiac performance is not affected by left stellectomy, as shown by findings during exercise studies both with and without infarction in dogs and in man. The observation that the QT interval is prolonged just before the occurrence of coronary spasm, which suggests a unilateral or dominant left sympathetic discharge, coupled with our findings of a vasoconstrictor activity primarily dependent on the left stellate ganglion, leads us to speculate that left stellectomy might be appropriate in preventing coronary spasm and its dangerous complications in patients with variant angina.

Considering the simplicity and the safety of this surgical intervention, we believe that its potential for reducing the incidence of ventricular fibrillation is worthy of further investigation.

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References

1. Lown B: Sudden cardiac death: the major challenge confronting contemporary cardiology. Am J Cardiol 43: 313, 1979
26. Fowlis RAF, Sang CTM, Lundy PM, Ahuja SP, Colhoun H: Experimental coronary artery ligation in conscious dogs six months after bilateral cardiac sympathectomy. Am Heart J 88: 748, 1974
52. Crampton RS: Preeminence of the left stellate ganglion in the long Q-T syndrome. Circulation 59: 769, 1979
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