The Treatment of Recurrent Ventricular Tachycardia with Bilateral Cervico-Thoracic Sympathetic-Ganglionectomy

A Report of Two Cases

By Richard Lloyd, M.D., Robert Okada, M.D., James Stagg, M.D., Robert Anderson, M.D., Brack Hattler, M.D., and Frank Marcus, M.D.

Summary

Two patients had bilateral cervico-thoracic sympathectomy for treatment of disabling symptoms due to refractory ventricular tachycardia. In both patients treatment with antiarrhythmic drugs singly, or in combination, or together with pacemaker overdrive had been ineffective in controlling this arrhythmia. Both patients had normal coronary arteriograms.

Bilateral thoracic sympathectomy appears to facilitate medical management of ventricular tachycardia in selected patients who are refractory to the usual medical treatment.

Additional Indexing Words:
Refractory ventricular tachycardia
Thoracic sympathectomy

Recurrent ventricular tachycardia not associated with coronary disease is uncommon. There have been sporadic reports of ventricular tachycardia without other evidence of heart disease, and Lesch et al. collected 34 reports of such patients between ages 2 and 40.\(^\text{1}\) This report relates our recent experience with two patients who had normal coronary arteriograms and who had recurrent incapacitating ventricular tachycardia, which proved refractory to conventional measures. Medical management of their arrhythmias was facilitated by bilateral cervico-thoracic sympathetic ganglionectomy. Prior experience with sympathectomy in this situation is reviewed.

Case Reports

Case 1

Patient M.E., a 48-year-old Caucasian housewife, was first admitted to the Arizona Medical Center on July 24, 1973. The patient was in good health until one week prior to ad-

mission when she experienced her first syncopal episode that lasted approximately four minutes. The patient consulted her family physician who prescribed spironolactone and hydrochlorothiazide for mild hypertension known to be present since 1971. An electrocardiogram was obtained at that time (fig. 1). During the ensuing week prior to admission the patient experienced several similar syncopal episodes. The patient was hospitalized elsewhere after an electrocardiogram demonstrated multifocal premature ventricular contractions, with short runs of ventricular tachycardia. On admission, the serum potassium was 3.7, chloride 102, and sodium 135 mEq/L. She was initially treated with intravenous injections of lidocaine, 50 mg each, repeated four times. Treatment thereafter consisted of quinidine sulfate, 200 mg per day given orally, diphenylhydantoin, 200 mg per day given orally, and a continuous intravenous infusion of lidocaine in doses up to 4 mg per minute. On the second hospital day the patient was transferred to the Arizona Medical Center because of the onset of generalized seizures initially thought to be due to lidocaine toxicity.

The patient could not recall any unusual childhood illnesses. She was told she had a heart murmur while in high school and was cautioned against excessive exertion. There was no family history of cardiac disease or syncopal episodes.

On admission to the Arizona Medical Center the patient was comatose. Initial measurements included a temperature of 36.5\(^\circ\) Centigrade, a weight of 63 kilograms, and a blood pressure of 130/100 mm Hg. She had a regular sinus rhythm with a rate of 100 beats per minute. This regular rhythm was interrupted by frequent premature beats and a tachycardia at a rate of 190 beats per minute, lasting less than three seconds. The tachycardia had a left bundle branch block configuration with a QRS axis of +120, identical to the
premature ventricular beats seen in figure 1. The cardiovascular examination was normal as was the rest of the physical examination, except for hyperactive deep tendon reflexes and positive plantar reflexes bilaterally.

Initial laboratory data included a hemoglobin of 12.8 g/100 mg, a hematocrit of 37% and a white blood cell count of 18,400/mm³ with a normal differential. Serum potassium was 3.0, sodium 115, and chloride 77 mEq/L. Blood urea nitrogen was 3, and glucose 135 mg/100 ml. Urine osmolality was 536 mOsm/kg. Electrocardiogram was normal except for frequent multifocal premature ventricular contractions. Chest X-ray was normal. Spinal tap was normal.

The patient’s electrolyte abnormalities and convulsions were thought consistent with the syndrome of inappropriate secretion of antidiuretic hormone. The etiology of this syndrome could not be determined, and did not recur subsequently. Convulsions abated after appropriate correction of her serum electrolytes and the patient regained consciousness on the second hospital day.

By the third hospital day the patient’s serum electrolytes, urea nitrogen, fasting glucose, creatinine, and serum osmolality were normal. Urine sodium, potassium, and osmolality were also normal. Repeat hemogram included a white blood cell count of 8,800/mm³ with a normal differential count. Serum determinations of creatinine phosphokinase, lactic dehydrogenase, glutamic oxaloacetic transaminase, glutamic pyruvic transaminase, alkaline phosphatase, calcium, phosphorus, total and direct bilirubin, total protein, albumin, cholesterol, triglycerides, uric acid, thyroxin, triiodothyronine, iron, and iron-binding capacity were all normal. Coagulation studies were within normal limits. Determinations of antinuclear antibodies lupus erythematosus cell preparation, and rapid plasma reagin test for syphilis were negative. The erythrocyte sedimentation rate was slightly elevated at 16 mm/hour.

The patient’s tachycardia was thought to be ventricular in origin for the following reasons:

1) The configuration of individual QRS complexes of the tachycardia closely resembled those of the premature ventricular contractions.

2) The episodes of tachycardia were never initiated by a P wave.

3) The tachycardia was unresponsive to carotid sinus stimulation.

Initial therapy with sequential intravenous injections of procaine amide, 1 gm, lidocaine, 250 mg, and diphenylhydantoin, 700 mg, was unsuccessful in preventing the recurrent tachycardia. The patient was then treated with diphenylhydantoin, 400 mg per day given intravenously, and a continuous intravenous infusion of lidocaine in doses up to 2 mg/minute. Subsequent therapy included a variety of drugs singly or in combination as well as atrial pacing (table 1). These measures did not have any important therapeutic effect and the patient’s electrocardiogram demonstrated bigeminal and trigeminal rhythm with frequent coupling of premature beats during the first six hospital days.

On the thirteenth hospital day the patient again began to experience frequent premature ventricular beats with coupling of beats and occasional short runs of ventricular tachycardia (fig. 2). Increasing the oral quinidine sulfate dose to 2400 mg/day resulted in no change in the frequency of the ectopic beats. Propranolol was discontinued at this time because of hypotension. On the eighteenth hospital day all medications were discontinued because of nausea and vomiting, fever, diffuse myalgias, abnormal liver function tests and coagulation studies. The latter include a prolonged prothrombin time and partial thromboplastin time and mildly depressed platelet count. The serum quinidine level on that day was 3.1 µg/ml. The serum diphenylhydantoin level was 10 µg/ml. A five-day course of prednisone, 60 mg po q.d., was begun the twentieth hospital day and did not alter the arrhythmia. The frequency and duration of the ventricular tachycardia increased. By the 27th hospital day the patient was experiencing up to 100 episodes of ventricular tachycardia per hour, with individual attacks lasting less than three minutes. All episodes of ventricular tachycardia terminated spontaneously. During the day.

**Figure 1**
Electrocardiogram of patient M.E. taken 10 days before admission to the Arizona Medical Center. The Q-T interval of 0.40 is prolonged. There are frequent premature ventricular beats which have the same configuration as that seen during the ventricular tachycardia.

**Figure 2**
Representative strips from a monitor lead documenting four separate episodes of arrhythmia in patient M.E. The ventricular response ranges from 200 to 260 beats/minute.
Table 1

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<th>Therapy to Control the Recurrent Arrhythmias in Patient M.E.</th>
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preceeding 27 days the combination of therapeutic measures listed in Table 1 were unsuccessful in permanently decreasing the frequency of the ventricular tachycardia.

Bilateral stellate ganglion blocks were performed with a combination of 10 ml each of lidocaine ¼% and bupivacaine ¼% with epinephrine 1:400,000. For two hours before the procedure she had 14-16 episodes of ventricular tachycardia, whereas for the two hours after the procedure, ventricular tachycardia did not recur.

During longer episodes of ventricular tachycardia the patient, while supine, complained of substernal chest ache, nausea, and light headedness. The patient's electrocardiogram demonstrated T wave inversion in the right atrial appendage. The free end of the electrode wire was threaded through the diaphragm and implanted in the subcutaneous tissues to be used for pacing if needed. The patient was free of ventricular arrhythmias for the first six days following surgery. Frequent premature ventricular beats developed on the seventh postoperative day. Short episodes of ventricular tachycardia lasting less than two seconds developed on the ninth postoperative day. These arrhythmias were well controlled with quinidine sulfate, 800 mg per day given orally. One week later the quinidine sulfate was discontinued because of nausea. Episodes of ventricular tachycardia did not recur and the patient was discharged from the hospital on September 10, 1973, without receiving any antiarrhythmic medications.

Ten days after discharge from the hospital the patient experienced a brief syncopal episode. Quinidine sulfate, 800 mg per day given orally, was again started and discontinued one week later because of nausea. The patient was treated with small doses of procaine amide given orally. She was subsequently referred to the care of Dr. Marvin Dunn, Chief of Cardiology at the University of Kansas. She was hospitalized at the University of Kansas Medical Center one month after discharge because of recurrent ventricular tachycardia. The rhythm was controlled with dipyridamyl, 300 mg daily. Eight months after surgery, she had occasional episodes of premature ventricular contractions and bigeminal rhythm.

Electrocardiograms obtained prior to sympathectomy and shortly before initiation of antiarrhythmic agents were forwarded to us by the patient's family physician and by the referring physician. Q-T intervals of 0.40 and 0.380 seconds (table 2) were consistently higher than the upper limits of normal reported by Ashman and Hull. Q-T intervals after surgery were 0.36 sec. These intervals were consistently lower than the upper limits of normal reported by Ashman and Hull (table 2). The patient was not receiving any antiarrhythmic agents at the time that any of the Q-T measurements were made.

**Case 2**

V.L., a 57-year-old aircraft mechanic, had the first of multiple episodes of ventricular tachycardia in 1960. His physical examination was then within normal limits. Electrocardiogram showed left bundle branch block and short
bursts of ventricular tachycardia that terminated spontaneously. He was treated with procaine amide 250 mg q.i.d. and hydroxyzine, 25 mg q.i.d. He had four symptomatic episodes of tachyarrhythmia in 1960 and 1961. In 1964 a Master’s Two-Step was done. An episode of ventricular tachycardia occurred immediately after exercise, and reverted spontaneously. Digitalis leaf, 0.1 gram daily, was begun. From 1964 until 1969 he was aware of intermittent tachycardia despite continuous medication. In December, 1969, the patient was hospitalized because of syncope and prolonged tachycardia, which was terminated by DC cardioversion. He was treated with increasing doses of propranolol up to 480 mg daily. Digitalis was continued. In February of 1970 medication was changed to propranolol, 320 mg daily, and diphenylhydantoin sodium, 400 mg daily, because of frequent ventricular tachycardia until May, 1970, when he was admitted to the Tucson Veterans Hospital with ventricular tachycardia at a rate of 250 beats per minute terminated by DC cardioversion.

Past medical history was negative for excess alcoholic intake, smoking, rheumatic fever, diphtheria, hypertension, diabetes. There was no family history of heart disease.

Examination of the heart in May, 1970, showed that the cardiac apical impulse was diffuse with no dyskinetic area palpable. An atrial gallop sound was present. No murmurs were heard. Chest X-ray showed that the heart was normal in size and shape. The lungs were clear. ECG showed left bundle branch block. (fig. 3).

The patient’s hospital course was marked by 12 episodes of ventricular tachycardia lasting from 10–30 minutes and associated with hypotension. A short episode of this arrhythmia is shown in figure 4. The ventricular tachycardia had a left bundle branch block configuration with a QRS axis of −80° (fig. 5). DC cardioversion was required on six occasions. The recurrent ventricular tachycardia was unaffected by administration of a variety of drugs alone or in combination including lidocaine up to 4 mg per min, quinidine polygalacturonate, 3.5 grams per day; propranolol, 400 mg daily, diphenylhydantoin, 400 mg daily and digoxin, 0.25 mg daily. He was given bretylium* 600–1200 mg daily, both parenterally and orally, without benefit. Pacemaker overdrive was not consistently successful in preventing or interrupting recurrent ventricular tachycardia. An atrial electrogram revealed P wave activity, independent of the ventricular complexes (fig. 6). Coronary arteriography revealed no significant coronary atherosclerosis. Left ventricular contraction was described as hypokinetiic; however, a left ventricular aneurysm was not present. On August 31, 1970, a two stage sympathectomy was begun. A left dorsal sympathectomy was performed with excision of the stellate and T1–T5 ganglia. Following sympathectomy the patient was treated with quinidine polygalacturonate, 3.5 grams daily, and propranolol, 240 mg daily. On this regimen he had between 1–5 premature ventricular beats per minute. Three weeks after operation, exercise with a bicycle ergometer up to 200 kilogram meters for five minutes produced no significant increase in pulse rate and there was no increase in ectopic beats. He was discharged September 25, 1970.

The patient returned one week later with recurrent ventricular tachycardia at a rate of 170 beats per minute necessitating DC cardioversion. Quinidine polygalacturonate was discontinued and procaine amide was given in doses of 4-8 grams per day; however, recurrent ventricular tachycardia continued. On October 14, 1970, right dorsal sympathectomy was performed with removal of the stellate and T1–T4 ganglia. Following operation the patient was treated with quinidine polygalacturonate, 3.3 grams per day, and propranolol, 160 mg per day. He was discharged on October 26, 1970, but returned on November 5, 1970.

*investigational drug
with ventricular tachycardia at the slower rate of 150 per minute. He was treated with DC cardioversion. He was discharged with the same drug regimen of quinidine and propranolol with the addition of hydrochlorothiazide 50 mg every other day because of a slight increase in heart size.

On December 21, 1970, ventricular tachycardia again occurred at a slower rate of 140-150 beats per minute. The tachycardia lasted for one hour before being successfully treated with DC cardioversion, 30 watt seconds. Once again he was discharged and remained asymptomatic until April, 1971, when he was hospitalized in an attempt to reduce the dose of antiarrhythmic drugs. He was given a stress exercise test and ectopic ventricular beats were induced at a slow heart rate. Twenty-four hours after stopping quinidine and propranolol, multiple premature ventricular beats occurred. Quinidine polygalacturonate was restarted at 2.2 grams per day. On April 30th, ventricular tachycardia occurred which converted with lidocaine. Propranolol 120 mg was reinstituted. In November, 1971, quinidine polygalacturonate was increased to 3.3 grams per day because of increased frequency of premature ventricular beats. These drugs were continued until August, 1972, when propranolol was decreased to 30 mg a day because of dizziness and bradycardia. Quinidine polygalacturonate was decreased to 2.2 grams per day and this drug regimen has been continued until the present time with little symptomatic indication of ventricular arrhythmia.

Since his bilateral sympathectomy the patient has been able to undertake part-time work as a hotel clerk. In October, 1973, a 24-hour continuous ECG showed that premature ventricular beats up to 14 per minute were present, with occasional bursts of asymptomatic ventricular tachycardia.

**Figure 5**  
Electrocardiogram of patient V.L. during ventricular tachycardia.

**Discussion**

Both patients demonstrated intractable ventricular tachycardia for which no etiology was found. In patient V.L. the diagnosis of ventricular tachycardia was based upon the presence of a tachyarrhythmia at the rate of 250 beats per minute with a QRS duration greater than 0.12 seconds and with a QRS configuration differing from the left bundle branch block pattern present during normal sinus rhythm. Atrial lead electrocardiography demonstrated independent atrial and ventricular activity. The diagnosis of ventricular tachycardia in patient M.E. was based upon the presence of ventricular premature beats preceding and following the paroxysmal tachycardia, which had QRS morphology similar to that during the arrhythmia. Vagotonic maneuvers or drugs had no effect upon the arrhythmia. However, capture, fusion, or independent atrial beats was not observed, and although the diagnosis of ventricular tachycardia was likely, it was not rigorously proven. We recognize that, in the absence of His bundle electrocardiography, strict criteria for the diagnosis of ventricular tachycardia are not fulfilled.

In hospitalized patients with ventricular tachycardia, it is estimated that 10–12% will have no other evident heart disease. In a recent review, Lesch described 35 such patients under 40 years of age to eliminate those with coronary artery disease. Although our patients were over 40 years of age, neither had a clinical history of coronary artery disease, and both had normal coronary arteriograms. Left ventricular angiograms did not show evidence of aneurysm. We could find no cause for the ventricular arrhythmia such as rheumatic heart disease, sarcoidosis, myxedema, collagen vascular disease, viral infections or myocarditis. A myocardial biopsy from patient M.E. taken at the time of pacemaker implantation did not show myocarditis. Steroid therapy in patient M.E. was prescribed before the biopsy was obtained on the presumptive diagnosis of myocarditis. There was no apparent decrease in the frequency of the arrhythmia as a result of steroid therapy. Variation of the frequency of the arrhythmia in relation to menses was not observed in patient M.E. This lack of correlation was documented since an increased frequency of paroxysmal ventricular tachycardia in relation to menstrual cycles has been reported in a 46-year-old female with improvement attributed to oral contraceptive therapy. However, later discontinua-
stellite removal in dogs produced a prolonged Q-T interval. This mimics the electrocardiogram seen in the spontaneously occurring syndrome in man and suggests that sympathetic stimulation or imbalance may be involved in its genesis. Further evidence for cardiac sympathetic participation in the induction of this syndrome is provided by the successful use of left cervico-thoracic sympathectomy in returning the Q-T interval to normal and in preventing recurrent arrhythmias. In this patient, unilateral left stellate block prior to surgery resulted in shortening of the Q-T interval, while right stellate block accentuated Q-T prolongation and led to increased ventricular irritability. The origin of the abnormally augmented sympathetic activity is unknown; however, it has been postulated to arise from foci within the central nervous system. The improvement of patient M.E. following sympathectomy adds support to prior suggestions that interruption of cardiac sympathetics may be of value in preventing syncope, ventricular arrhythmias, and death in this syndrome. That one can predict the likelihood of improvement with sympathectomy was suggested by the response of the patient of Moss and McDonald to unilateral stellate blockade. Our patient, M.E., had a brief response following simultaneous bilateral stellate blocks, which produced a bilateral Horner's syndrome. Unilateral block was not performed and unfortunately no observations of changes in her Q-T interval was made. Unilateral stellate blockade may produce changes in the Q-T interval which might be predictive of the response to sympathectomy.

Trials of "medical sympathectomy" with propranolol alone or combined with quinidine were unsuccessful. Ventricular arrhythmias following experimental myocardial infarction in animals are diminished both by propranolol and by sympathectomy. While the protection from ventricular arrhythmias afforded by beta-blockade and sympathectomy in experimentally induced myocardial infarction was found to be qualitatively similar, it is the impression of some investigators that beta-blockade is quantitatively less effective (A. G. Wallace, personal communication). We cannot explain the disparity in the results of treatment between propranolol and sympathectomy in our patients. However, it appears that failure to respond to beta-blockade does not preclude a beneficial response to sympathectomy.

The role of sympathectomy in therapy of refractory ventricular tachycardia without identifiable cause remains to be defined. A recent report described the results of thoracic sympathectomy in five patients with recurrent ventricular tachycardia. Of the four patients with ischemic heart disease, two had no response, two had temporary relief and one had a
good result. The one patient with cardiomyopathy maintained sinus rhythm eight years after surgery. It would appear that thoracic sympathectomy would not be expected to be helpful in controlling recurrent ventricular tachycardia in the majority of patients with ischemic heart disease. While the temporal association between sympathectomy and control of ventricular tachycardia is obvious in our patients, it must be recognized that the natural history of this disorder is likely to be variable. Thus in patient V.L. the initial episodes of ventricular tachycardia were followed by an eight year period which was free of symptomatic arrhythmias. In view of this variability, caution is indicated in attributing causality to therapeutic maneuvers. However, it would appear that in certain selected patients with symptomatic ventricular arrhythmias refractory to medical therapy, bilateral cervico-thoracic sympathectomy may be of benefit.

Acknowledgments

We wish to thank Dr. Oscar Thorup for reviewing the manuscript; Dr. Deward J. Moody of Nogales, Arizona, and Dr. Sam Maben, Raytown, Missouri, for providing the electrocardiograms; and Dee Beckman for secretarial assistance. We gratefully acknowledge the assistance of Medical Audio-Visual Services at the University of Arizona Medical Center and the Tucson Veterans Administration Hospital for photographic assistance.

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Circulation. 1974;50:382-388
doi: 10.1161/01.CIR.50.2.382

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

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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