Clin. Cardiol. 20, 169–174 (1997)

Short Communications

Catheter Ablation of Ventricular Tachycardia in Chagasic Cardiomyopathy

FERNANDO ROSAS, M.D., VICTOR VELASCO, M.D., FELIPE ARBOLEDA, M.D., HERNANDO SANTOS, M.D., HERNANDO ORJUELA, M.D., NESTOR SANDOVAL, M.D., VICTOR CAICEDO, M.D., JUAN CORREA, M.D., GUY FONTAINE, M.D., PH.D.*

Department of Electrophysiology and Pacing, Colombian School of Medicine, University Clinic A. Shaio, Bogotà, Colombia; *Department of Rhythmology and Cardiac Stimulation, Hôpital Jean Rostand, Ivry sur Seine, France

Summary: There is a limited experience with catheter ablation for treatment of ventricular tachycardia (VT) in Chagasic cardiomyopathy. A 30-year-old woman experienced episodes of palpitations and syncope due to attacks of VT. A diagnosis of Chagas disease was established on a biological basis. Two-dimensional echo and contrast ventriculography showed an apical aneurysm with thrombus. Surgery was indicated to resect the aneurysm and ablate the VT. Ventricular tachycardia recurred 1 month later despite therapy, including amiodarone. Two clinical frequent and well-tolerated tachycardias were identified. The site of origin was located in the right ventricular apex and in the apical-lateral wall of the left ventricle, respectively. Catheter ablation was performed at two sites with DC shocks (total energy 600 J) after unsuccessful radiofrequency ablation. Holter recordings performed during the postoperative period showed only infrequent extrasystoles. After follow-up of 24 months the patient remains asymptomatic. Drug-refractory VT in Chagasic cardiomyopathy can be ablated by medium-energy DC shocks after failure of radiofrequency ablation.

Key words: Chagas disease, ventricular tachycardia, ablation

Introduction

The World Health Organization estimates that about 90 million people are at risk of infection with *Tripanosoma Cruzi*, the agent of Chagas disease.[1] In Latin America, 10 to 20 million people are infected and nearly 50,000 deaths occur every year, especially by Chagasic cardiomyopathy.[2] There is no effective medical treatment for this chronic infectious disease; only preventive measures in the rural areas have resulted in a decrease of its incidence. Acute clinical manifestations of the disease occur in 10% of patients, whereas 90% of these reach the chronic period without clinical manifestations. After a latent period of nearly 20 years, patients may develop clinical manifestations of visceral disease.[3] The development of chronic heart disease is estimated to occur in approximately 30–40% of patients.[4] Heart failure, embolic phenomena, arrhythmias, atypical chest pain, and sudden death are the cardiovascular manifestations of this disease.[5–10] Nearly 50% of the patients with ventricular arrhythmias have no marked cardiac dilatation; in fact, heart size can be normal on chest x-ray and two-dimensional (2-D) echo.[11–13] In endemic areas, 30% of autopsied Chagasic patients died suddenly,[14] and it is possible that episodes of ventricular tachycardia (VT) preceded ventricular fibrillation in these patients.[5] A combination of anatomopathologic substrate and an apparent parasympathetic denervation could trigger these events.[15] Since the introduction of catheter ablation for the treatment of arrhythmias,[16–19] this therapy has proven to be useful for patients with VT resistant to antiarrhythmic therapy when episodes are well tolerated. Few references have been published in the literature related to ablation of VT in Chagasic cardiomyopathy. In these patients, the site of origin of VT was located in the left ventricle; they had a low ejection fraction and one or several morphologies of monomorphic VT. Successful control of the arrhythmia has been obtained in nearly 50% of patients.[20,21]

We report one case of VT complicating Chagasic cardiomyopathy, successfully treated by DC ablation after failure of antiarrhythmic drugs and attempted radiofrequency (RF) ablation.

Case Report

A 30-year-old woman was hospitalized for episodes of palpitations and syncope of 1 week's duration. She had a history

Address for reprints:

Docteur G. Fontaine
Hôpital Jean Rostand
39 rue Jean le Galleu
94200 Ivry, France

Received: September 13, 1995
Accepted with revision: January 5, 1996

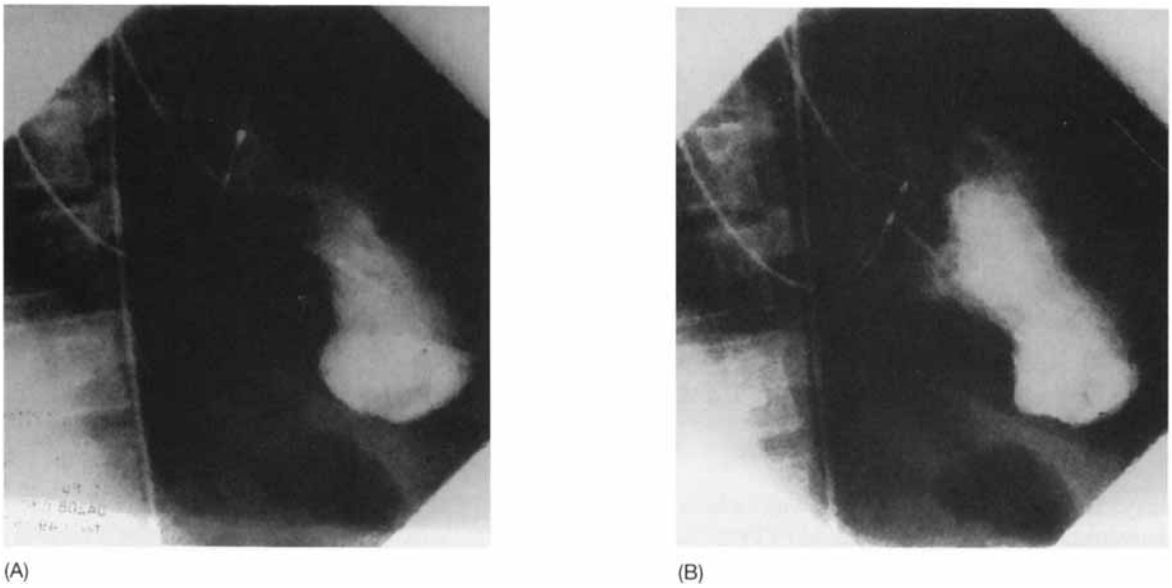


FIG. 1 Contrast ventriculography: Systolic (A) and diastolic (B) anteroposterior views showing an apical aneurysm of the left ventricle with an intracavitary thrombus. A transitory pacemaker electrode is seen in the right ventricle outflow.

of residing in an area of Colombia where Chagas disease is endemic. Five years later, the patient presented with syncope but did not undergo medical evaluation at that time. In the emergency unit, she was unconscious. She had VT with a rate of 270 beats/min, and blood pressure was 50/40 mmHg. Electrical cardioversion was performed. The electrocardiogram (ECG) in sinus rhythm showed abnormal repolarization in the inferolateral leads. The patient received a loading dose of amiodarone of 8 g in the first week and then a maintenance dose of 400 mg per day. Hemogram, blood chemistries, and coagulation tests were normal. *Tripanosoma Cruzi* antibodies by indirect immunofluorescence were positive on two occasions at a titer of 1/160. Chest x-ray showed mild enlargement. The patient had left ventricular dysfunction with an ejection fraction of 35% by 2-D echo and contrast ventriculography. An apical aneurysm containing mural thrombus was also seen (Fig.1). The coronary arteries were normal. Baseline electrophysiologic study was not performed because of hemodynamically unstable repetitive VT despite medical therapy. The patient underwent open heart surgery for resection of the aneurysm and for ablation of the arrhythmia. However, during sur-gery VT was not inducible by programmed ventricular stimulation during pacing with S1 of 600 and 400 ms, plus three extrastimuli with and without isoproterenol infusion. Resection of the aneurysm and intracavitary thrombus as well as geometric reconstruction of the left ventricle were performed. The procedure was well tolerated.

Histologic study of the excised aneurysm and myocardium showed extensive interstitial fibrosis, chronic mononuclear cellular infiltration, and hypertrophy of myocardial fibers with alteration of the usual parallel orientation (Fig. 2). One month later, VT recurred despite medical therapy including amiodarone, metoprolol, and enalapril. The VT had two morphologies (Fig.3). These episodes were hemodynamically well tolerated, but quite symptomatic. They were almost incessantly recurring about 10 times a day, lasting 10 to 45 min. They could not always be terminated by intravenous procainamide (10 mg/kg). An electrophysiologic study was performed with #6 or #7-F bipolar and quadripolar catheters introduced into the femoral artery and vein and advanced to the right atrium, right ventricular apex, His bundle and left ventricle. The ECG and intracavitary electrograms were recorded with the Midas 2000 system (PPG Biomedical System). The bipolar intracardiac electrograms were filtered between 30 and 500 Hz.

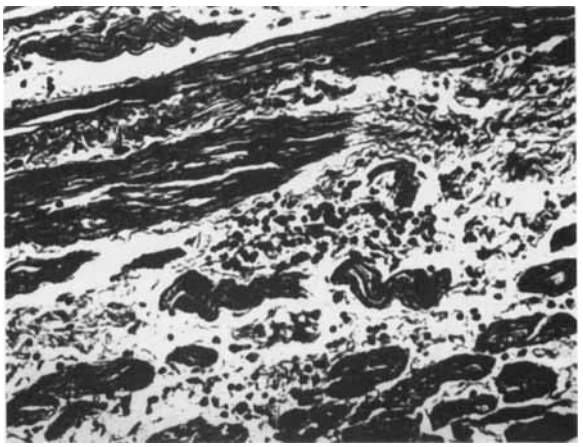


FIG. 2 Histology of the aneurysm showing extensive fibrosis and monocellular lymphoplasmocyte infiltrates with surviving myocardial fibers. Hematoxyline eosin stain (G × 280).

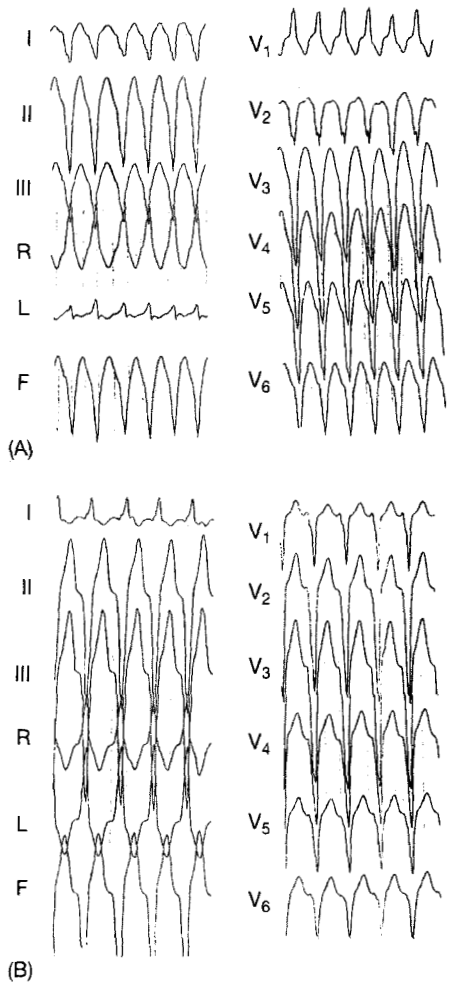


FIG. 3 Two different morphologies of clinical ventricular tachycardias (A, B), both with left axis deviation.

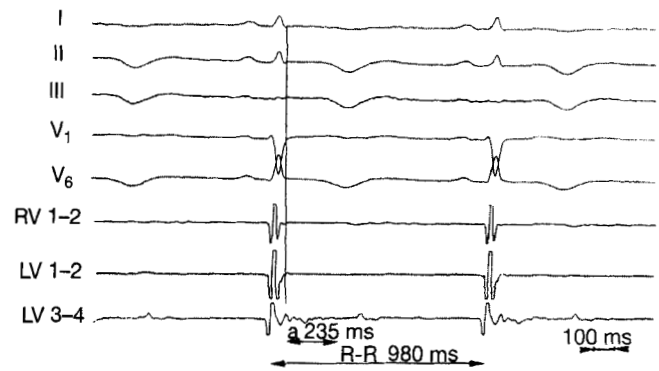
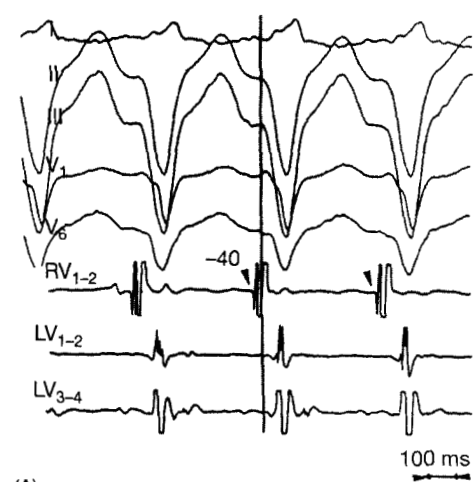


FIG. 4 Endocardial signals recorded inside the left lateral apical ventricular wall showing (a) fragmented potentials extending up to 235 ms after the end of the surface QRS complex. RV = right ventricle; LV = left ventricle.



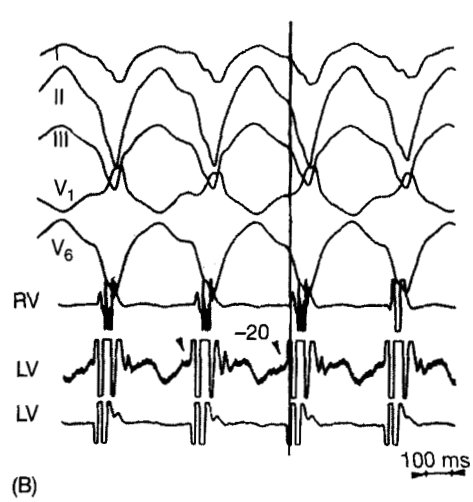


FIG. 5 Intracavitary recording during sustained ventricular tachycardia: (A) at the apex of the right ventricle, with a presystolic interval of −40 ms (arrow), (B) at the left lateral apical ventricular wall with a presystolic interval of −20 ms (arrow). RV = right ventricle, LV = left ventricle.

In the left ventricular lateral apical wall, several low-amplitude and low-frequency potentials were found during sinus rhythm, 235 ms after the end of the surface QRS complex (Fig.4). The clinical VTs were induced by programmed ventricular stimulation. Endocardial mapping was performed with a #7-F quadripolar catheter with a 4 mm long distal electrode (Mansfield Webster Catheter). The site of origin was located at the apex of the right ventricle, the other was in the apical one-third of the lateral wall of the left ventricle. The intracavitary potentials during VT showed early activity in relation to the surface QRS of −40 ms and −20 ms, respectively (Fig. 5). Pacemapping in these sites during sinus rhythm reproduced the morphology in 10 of 12 surface leads for the VT originating in the right ventricle and in 9 of 12 for VT originating in the left ventricle (Fig.6). A total of 20 applications of radiofrequency energy of 20–30 W for 30–60 s was delivered using a Radionics RFG-3DRF generator (Burlington, Mass.), without temperature control in the right and left ventricles between an adhesive patch in the back and distal electrode of the ablation catheter, and all were unsuccessful. Ablation by DC

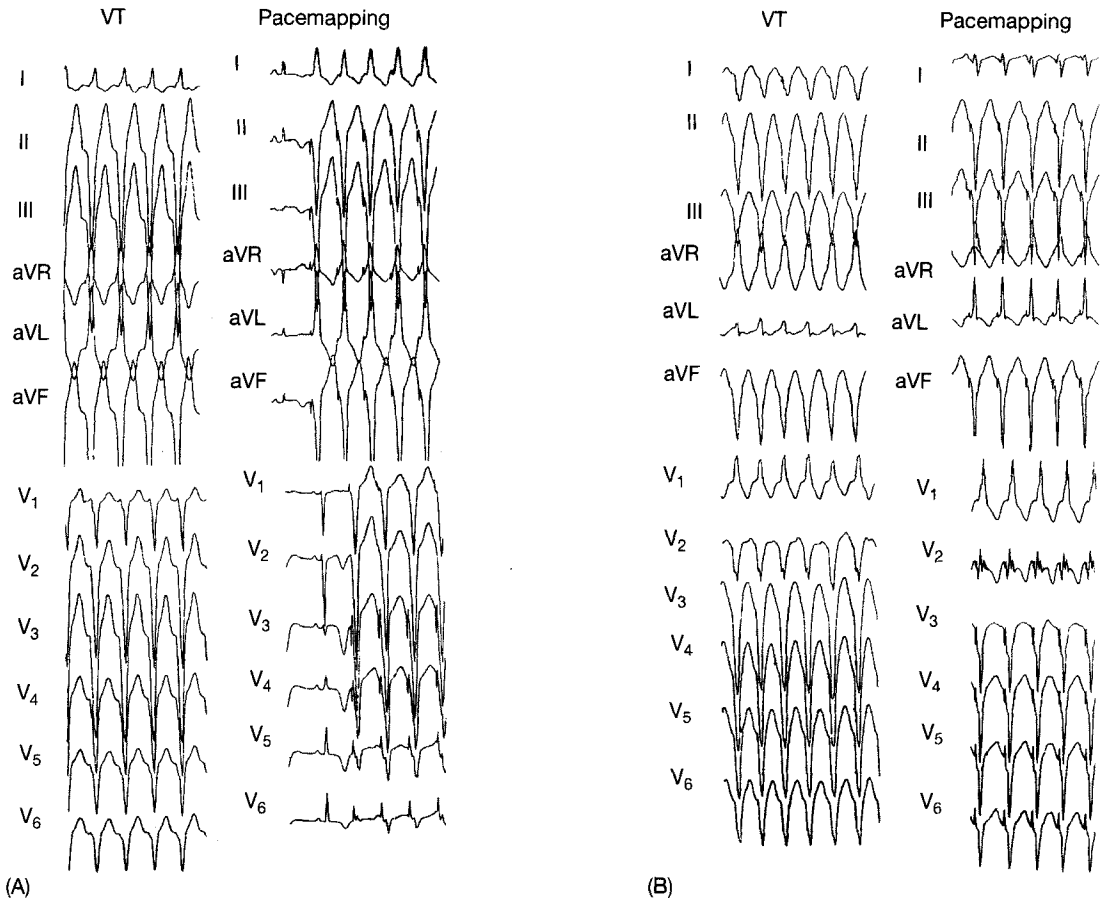


FIG. 6 (A) Pacemapping obtained with a score of 10/12 during pacing at the right ventricular apex. Left: clinical right ventricular tachycardia; right: during pacing. (B) Pacemapping obtained from the apical portion of the left lateral apical ventricular wall with a score of 9/12. Left: clinical left ventricular tachycardia; right: during pacing.

synchronized shock (Therecard Defibrillator 400, Siemens Medical, Iselin, N.J.) was performed after general anesthesia between the distal cathodal electrode and the defibrillator anodal patch in the left anterior wall. Catheter ablation was performed with the same catheter which was previously used for RF. Two cathodal shocks of 100 J and one shock of 200 J were delivered at the sites of origin of the VT in the right and left ventricles respectively. The peak value of CPK-MB fraction was 40 IU (normal value < 25 IU). One week later, the same morphologic right ventricular VT recurred and another catheter ablation was performed. DC energy was delivered to the right ventricular apex with two cathodal shocks of 100 J, again after failure of RF ablation. The CPK-MB fraction rose to 36 IU. The 2-D echo-Doppler control showed no new abnormality induced by ablation. After ablation, the patient refused a follow-up electrophysiologic study; therefore, two 24-h Holter were performed that showed no new episodes of VT. After a 24-month period following ablation, the patient is asymptomatic. She continues to take amiodarone, metoprolol, and enalapril because of the risk of relapse because of the extensive histologic myocardial disease. An AAI-R pacemaker was incidently implanted for sinus node dysfunction.

Discussion

In Brazil, Venezuela, and Argentina, Chagas disease is an important cause of morbidity and mortality, especially in rural areas.[2] In Colombia, there are endemic areas of this disease. The patient, who spent her childhood in one of these areas, was exposed to the insect vector. Evidence for the diagnosis of Chagasic cardiomyopathy was (1) residence in an endemic area 20 years previously, (2) apical left ventricular aneurysm with intracavitary thrombus, (3) ventricular tachycardia, (4) positive serologic test, and (5) histologic findings.

Ventricular tachycardia (VT) was the first clinical presentation of the disease[5, 11, 12] after a long asymptomatic period.[22] Class III antiarrhythmic medications are the most effective therapy for VT since other antiarrhythmic drugs such as quinidine have been implicated in cases of proarrhythmic death.[5] The lack of response to amiodarone has also been described.[23]

Endocardial mapping during sinus rhythm showed late potentials that may reflect the histologic findings of myocardial fibers embedded in extensive interstitial chronic mononuclear cells and fibrous tissue.[24, 25] The electrophysiologic presentation of these histologic findings suggests nonhomogeneous

anisotropy[26, 27] that consists of a preferential transverse transmission of the electrical impulse of the myocardial fibers with abnormal slow conduction. These conditions are compatible with reentrant circuits if other electrophysiologic factors are met.[11, 28]

Most authors consider that at least 10 leads and preferably 11 or 12 need to match in order to conclude that the mapping catheter is close enough to the site of origin of the VT to make ablation possible. In our case, during electrophysiologic study, the absence of mid-diastolic potentials during VT and the imperfect pacemapping during sinus rhythm suggested that the catheter was not located at the best site for ablation. However, the effectiveness of the DC ablation procedure suggests that ablation was performed close enough to the site of origin to prevent reentry. Therefore, a perfect pacemapping was not an obligatory prerequisite to achieve a satisfactory result.[29–31]

We think that the absence of response to radiofrequency energy could be due to (1) distance of catheter ablation from the site of origin of VT, (2) the fact that the lesions made by this form of energy were too small to ablate the critical slow pathway,[32, 33] and (3) a possible intramyocardial origin of this form of VT.[20, 21] The good response to DC energy is consistent with the larger lesion generated by its electric and barotraumatic effects that induced sufficient modifications of the reentrant circuits.[34] In addition, medium-energy DC (100–200 J cathodal shocks) carries less risk than high-energy DC.[35]

Transcoronary chemical ablation of incessant VT in two patients with chronic Chagas disease has also been described, with successful control of VT.[36, 37]

Sick sinus syndrome was either secondary to medical therapy or could have been due to the clinical manifestation of underlying heart disease or a combination of both. Amiodarone was continued because of the risk of relapse due to the extensive histologic myocardial disease.[12] In addition, we administered an angiotensin-converting enzyme inhibitor to preserve cardiac function.[38] The patient has not developed any new episodes of VT during a follow-up period of 24 months.

Conclusion

This case illustrates the problems of treatment of VT associated with Chagas disease. Medium-energy DC catheter ablation can be an effective treatment for patients with Chagasic cardiomyopathy and VT refractory to medical therapy and radiofrequency ablation. In this situation, the arrhythmogenic substrate is highly fibrotic, and in this setting VT is more difficult to ablate than after myocardial infarction. However, in this case, a more precise endocardial mapping or higher energies could have led to successful radiofrequency ablation.

Acknowledgments

The authors thank Mrs. Nicole Proust, Mrs. Sylvie Aaddaj, and Mrs. Martha Calixto for their invaluable assistance in the preparation of the manuscript.

References

1. Chagas C: *Nova Tripanozomiae humana. Mem Inst Oswaldo Cruz* 1909;160–216
2. Control of Chagas' disease. Report of a WHO Expert Committee World Health Organization, Geneva 1991; 1
3. Tanowitz H, Kirchhoff L, Simon D, Morris S, Weis L, Wittner M: Chagas' disease. *Clin Microbiol Rev* 1992;5:400–419
4. Filho-MM, Sosa E, Nishioka S, Scanavaccam M, Belloti G, Pileggi F: Clinical and electrophysiologic features of syncope in chronic heart disease. *J Cardiovasc Electrophysiol* 1994;5:563–570
5. Elizari M, Chiale P: Cardiac arrhythmias in Chagas' heart disease. *J Cardiovasc Electrophysiol* 1993;4:596–608
6. Bestetti RB, Ramos CP, Godoy RA, Olivera JSM: Chronic Chagas' heart disease in the elderly: A clinical pathological study. *Cardiology* 1987;74:344–351
7. Araujo RC, Bestetti RB, Godoy RA, Olivera JSM: Chronic Chagas' heart disease in children and adolescents: A clinico-pathologic study. *Int J Cardiol* 1985;9:439–449
8. Laranja FS, Dias E, Nobrega G, Miranda A: Chagas' disease: A clinical epidemiologic and pathologic study. *Circulation* 1956; 14:1035–1060
9. Giniger AG, Retyk EO, Laino RA, Sananes EG, Lapuente AR: Ventricular tachycardia in Chagas' disease. *Am J Cardiol* 1992;70: 459–462
10. Chiale PA, Halpern S, Nau GJ, Przybylsky J, Tambussi M, Lazzari JO, Elizari MV, Rosenbaum MB: Malignant ventricular arrhythmias in chronic Chagasic myocarditis. *PACE* 1982;5:162–172
11. Bestetti R, Santos C, Machado O, Arioli MT, Carmo J, Costa N, Olivera RB: Clinical profile of patients with Chagas' disease before and during sustained ventricular tachycardia. *Int J Cardiol* 1990; 29:39–46
12. Milei J, Storino R, Alonso GF, Beigelman R, Vanzulli S, Ferrans V: Endomyocardial biopsies in chronic Chagasic cardiomyopathy. Inmunohistochemical and ultrastructural findings. *Cardiology* 1992;80:424–437
13. Koberle F: Chagas' disease and Chagas' syndromes: The pathology of American trypanosomiasis. *Adv Parasitol* 1968;6:63–116
14. Bestetti RB, Freitas OC, Muccillot G, Oliveira SM: Clinical and morphological characteristics associated with sudden cardiac death in patients with Chagas' disease. *Eur Heart J* 1993;14:1610–1614
15. Davila D, Bellabarbra G, Donis JH, Torres A, Rossell J, Figueroa O, Amaro M, Vasquez J: Cardiac autonomic control mechanisms in Chagas' heart disease. Therapeutic implications. *Med Hypoth* 1993;40:33–37
16. Vedel J, Frank R, Fontaine G, Fournial JF, Grosgogeat Y: Bloc auriculo-ventriculaire intra-hisien definitif induit au cours d'une exploration endocavitaire droite. *Arch Mal Coeur* 1979;72:107–112
17. Gonzales I, Scheinman M, Margaretten W, Rubinstein M: Closed chest electrode catheter technique for His bundle ablation in dogs. *Am J Physiol* 1981; 241:283–287
18. Scheinman MM, Morady F, Hess DS, Gonzalez R: Catheter induced ablation of the atrioventricular junction to control refractory supraventricular arrhythmias. *J Am Med Assoc* 1982;248:851–855
19. Fisher JD, Brodman R, Kins SG, Matos JA, Brodman LE, Wallerson D, Waspel E: Attempted nonsurgical electrical ablation of accessory pathway via the coronary sinus in the Wolff-Parkinson-White syndrome. *J Am Coll Cardiol* 1984;4:685–694
20. Galvao S, Medeiros J, Santos R, Maeve S, Decio P: Treatment of recurrent ventricular tachycardia by endocardial catheter fulguration in patients with Chagas' cardiomyopathy (abstr). *Eur J CPE* 1992;2:153
21. Sosa E, Scalabrini A, Rati M, Belloti G, Pileggi F: Successful catheter ablation of the "origin" of recurrent ventricular tachycardia in chronic Chagasic heart disease. *J Electrophysiol* 1987;1:58–61
22. Case records of the Massachusetts General Hospital. Weekly clinicopathological exercises; Case 32-1993. A native of Salvador with tachycardia and syncope. *N Engl J Med* 1993;329:488–496

23. Chiale PA, Halpern MS, Nau GJ, Tambassi AM, Pryzzbylski J, Lazzari JO, Elizari MV, Rosenbaum MB: Efficacy of amiodarone during long-term treatment of malignant ventricular arrhythmias in patients with chronic Chagasic myocarditis. *Am Heart J* 1984;107: 656–665
24. Laranja FS, Dias E, Nobrega G, Miranda A: Chagas' disease. A clinical, epidemiologic, and pathologic study. *Circulation* 1956; 14:1035–1060
25. Rosenbaum M: Chagasic myocardiopathy. *Prog Cardiovasc Dis* 1964;7:199–225
26. Spach MS, Dolver PC: Relating extracellular potentials and their derivates to anisotropic propagation at a microscopic level in human cardiac muscle. Evidence for electrical uncoupling of side-to-side fiber connections with increasing age. *Circ Res* 1986;58: 356–371
27. Spach MS, Miller WT, Dolver PC, Kootsey M, Sommer JR, Mosher CE: The functional role of structural complexities in the propagation of depolarization in the atrium of the dog: Cardiac conduction disturbances due to discontinuities of effective axial resistivity. *Circ Res* 1982;50:175–191
28. Wit AL, Dillon SM: Reentry caused by the anisotropic conduction properties of cardiac muscle. In *Tachycardias: Mechanisms and Management* (Eds. Josephson ME, Wellens HJJ). Futura Publishing Company, Inc., 1993:13–31
29. Morady F, Kadish S, Rosenheck H, Calkins H, Kou WH, De Buitleir M, Sousa J: Concealed entrainment as a guide for catheter ablation of ventricular tachycardia in patients with prior myocardial infarction. *J Am Coll Cardiol* 1991;17:678–689
30. Stevenson WG, Weiss JN, Weiner I, Wohlgelernter D, Yeatman L: Localization of slow conduction in a ventricular tachycardia circuit by entrainment: Implications for catheter ablation. *Am Heart J* 1987;114:1253–1258
31. Morady F, Scheinman MM, Diu Carlo LA Jr, Davis JC, Herre JM, Griffin JC, Winston SA, De Buitleir M, Hantler CB, Wahr JA, Kou WH, Nelson SD: Catheter ablation of ventricular tachycardia with intracardiac shocks: Results in 33 patients. *Circulation* 1987;75: 1037–1049
32. Langberg JJ, Lee MA, Chin MC, Rosenqvist M: Radiofrequency catheter ablation: The effect of electrode size on lesion volume in vivo. *PACE* 1990;13:1242–1248
33. Huang SK: Advances in applications of radiofrequency current to catheter ablation therapy. *PACE* 1991;14:28–42
34. Fontaine G, Umemura J, Iwa T, Aldakar M, Grosgogeat Y: Aspects physiques et biophysiques des décharges électriques intracardiaques à haute energie. IV-Effets des bulles de fulguration en milieu diphasique isotrope. *Ann Cardiol Angeiol* 1991;40:515–625
35. Fontaine G, Frank R, Gallais Y, Rosas Andrade F, Tonet J, Lascault G, Aouate P, Poulain F: La radiofréquence dans le traitement de la tachycardie ventriculaire. *Arch Mal Coeur* 1996;89:99–107
36. Sternic EB, Sobrino AL, Lisboa JC, Barbosa MR, Fantini F, Gontijo-Filho B, Vrandecic MO: Transcoronary chemical ablation of ventricular tachycardia in a patient with chronic Chagas cardiomyopathy. *Arq Bras Cardiol* 1992;58:307–310
37. Paola AA, Gomes JA, Miyamoto MH, Fo EE: Transcoronary chemical ablation of ventricular tachycardia in chronic Chagasic myocarditis. *J Am Coll Cardiol* 1992;20:480–482
38. The SOLVD investigators: Effect of enalapril on survival in patients with reduced left ventricular ejection fractions and congestive heart failure. *N Engl J Med* 1991;325:293–302