Case Report

Reconstruction of the Right Ventricular Outflow Tract with a Transannular Patch for Ventricular Tachycardia Refractory to Radiofrequency Catheter Ablation in a Patient who Underwent Tetralogy of Fallot Surgery in Childhood

Akira Shimane MD*[1], Katsunori Okajima MD*[1], Kazuo Mizutani MD*[1], Masami Yoshida MD*[1], Kimitake Imamura MD*[1], Takatoshi Hayashi MD*[1], Yasuyo Taniguchi MD*[1], Shinichiro Yamada MD*[1], Sachiyo Iwata MD*[1], Masahiro Kumada MD*[1], Yasue Tsukishiro MD*[1], Kensuke Matsumoto MD*[1], Yusuke Kurogane MD*[1], Keiko Ryo MD*[1], Takumi Inoue MD*[1], Amane Kozuki MD*[1], Masahiro Tashiro MD*[1], Teishi Kajiya MD*[1], Masato Yoshida MD*[2], Kazuhiro Mizoguchi MD*[2], Katsuhiro Yamanaka MD*[2], Nobuhiko Mukohara MD*[2], Kouhei Yamashiro MD*[3]

*[1]Division of Cardiology, Himeji Cardiovascular Center
*[2]Division of Cardiovascular Surgery, Himeji Cardiovascular Center
*[3]Division of Cardiology, Toyohashi Heart Center

A 29-year-old male who underwent a complete tetralogy of Fallot repair at 2 years of age was referred to our hospital for treatment of sustained ventricular tachycardia (VT). The bipolar voltage map using an electroanatomical mapping system (CARTO, Biosense-Webster) during sinus rhythm revealed a low voltage area identical to the site of the right ventricular outflow tract (RVOT) patch on the anterior wall of the RVOT. During the tachycardia, the activation wavefront was found to revolve in a counterclockwise manner around the patch in the RVOT. Two radiofrequency catheter ablation (RFCA) sessions creating a line between the patch in the RVOT and pulmonary artery achieved only transient success. He underwent a pulmonary valve replacement and reconstruction of the RVOT with a transannular patch to treat the VT refractory to RFCA and severe pressure gradient in the RVOT. In postoperative electrophysiological study, a low voltage area in the RVOT connected to the pulmonary artery with the patch was observed, and produced conduction block in the reentry circuit of the VT. The patient has been free from any VT recurrence during 6 months of follow up.
(J Arrhythmia 2008; 24: 156–161)

Key words: Tetralogy of Fallot, ventricular tachycardia, surgical treatment

Received 12, February, 2008: accepted 31, July, 2008.
Address for correspondence: Akira Shimane MD, Himeji Cardiovascular Center, Saisyou Kou 520, Himeji, 670-0981, Japan. Phone: 079-293-3131 FAX: 079-295-8199

Introduction

Surgical repair of tetralogy of Fallot (TOF) has been reported with favorable outcomes in a majority of patients.[1–3] However, sustained ventricular tachycardia (VT) occurs in 0.4–4.2% of patients who undergo corrective surgery.[4,5] The majority of monomorphic VTs presumably are caused by macroreentry occurring around the right ventricular outflow tract (RVOT) scar and patch used to enlarge the RVOT and relieve the obstruction to flow.

Successful radiofrequency catheter ablation (RFCA) of monomorphic VT in patients after TOF repair has been reported in the last decade.[6–10] We report on a patient with TOF whose VT was refractory to RFCA successfully treated with a pulmonary valve replacement and transannular patch repair of the RVOT.

Case Report

This report describes a 29-year-old male who underwent a complete TOF repair at 2 years of age with a non-transannular RVOT patch. At the age of 29, he presented with palpitations and sustained VT. A 12-lead surface electrocardiogram (ECG) revealed a monomorphic VT with left bundle branch block, left axis deviation and a cycle length of 320 msec (**Figure 1A**). The tachycardia was terminated by an injection of procainamide. During sinus rhythm, the ECG exhibited a first-degree atrioventricular block, right bundle branch block and superior axis with a QRS duration of 160 msec (**Figure 1B**). A chest X-ray revealed cardiomegaly with a cardiothoracic ratio of 58% and normal pulmonary vascular markings. Two-dimensional echocardiography demonstrated right ventricular hypertrophy with a wall thickness of 5 mm, right ventricular enlargement, tricuspid regurgitation, pulmonary regurgitation, a mean pressure gradient of 40 mmHg from the RVOT to the main pulmonary artery and normal left ventricular function. The patient was referred to our hospital for further treatment of sustained VT.

Electrophysiological study and ablation procedure

After written informed consent was obtained, an electrophysiological study and catheter ablation was performed under local anesthesia. One decapolar catheter was introduced via the right jugular vein and advanced into the coronary sinus under fluoroscopic guidance. One decapolar and one quadoripolar catheter were introduced via the right femoral vein

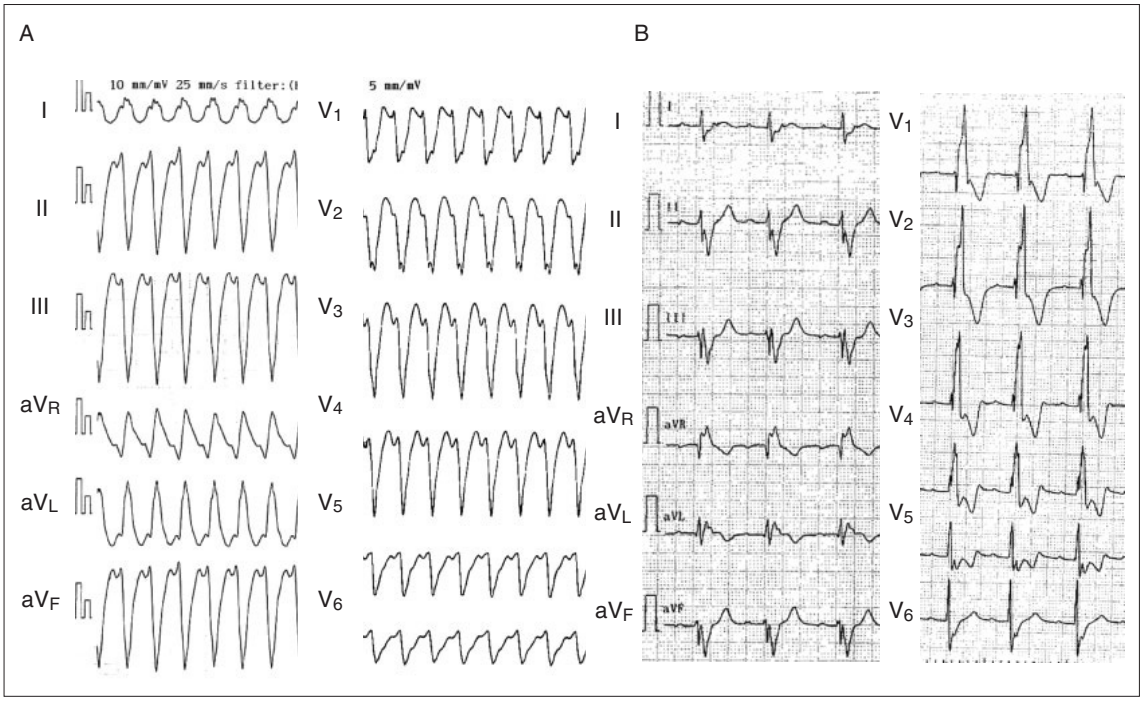

Figure 1
A: During the VT, the ECG revealed a left bundle branch block and left axis deviation morphology with a cycle length of 320 msec.
B: During sinus rhythm, the ECG revealed a first-degree atrioventricular block and right bundle branch block with a QRS duration of 160 msec.

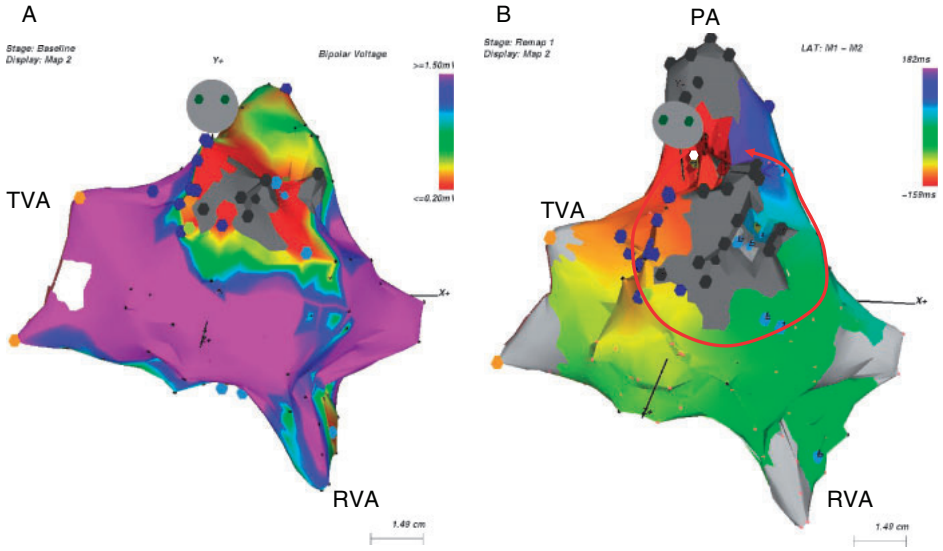

Figure 2
A: The bipolar voltage map during sinus rhythm revealed a low voltage area identical to the site of the RVOT patch on the anterior wall of the RVOT. **B**: The activation map during the tachycardia. The activation wavefront was found to revolve in a counterclockwise manner around the scar in the RVOT. The colors represent early (red) to late (purple) activation. White tag indicates the site where the mid-diastolic potential was recorded. Brown tags indicate the ablation points.
PA: pulmonary artery, RVA: right ventricular apex, TVA: tricuspid valve annulus

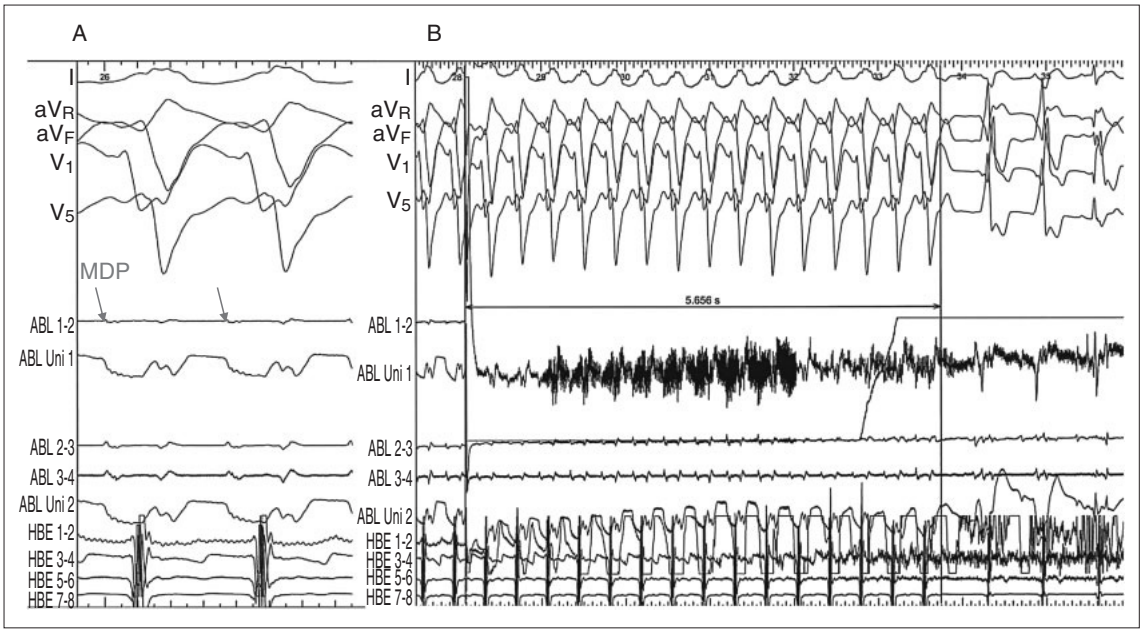

Figure 3
A: The local bipolar electrogram along the isthmus between the RVOT patch and pulmonary artery demonstrated a fractionated mid-diastolic potential (MDP). **B**: Intra-cardiac electrogram during the RF delivery. The RF current interrupted the tachycardia.

and positioned in the His-bundle region and right ventricular apex (RVA), respectively. One 4 mm tip deflectable 7Fr catheter (Navistar, Biosense-Webster) was used for mapping and ablation.

The bipolar voltage map using an electroanatomical mapping system (CARTO, Biosense-Webster) during sinus rhythm revealed a low voltage area identical to the site of the RVOT patch on the anterior wall of the RVOT (**Figure 2A**). Clinical VT was induced by programmed electorical stimulation

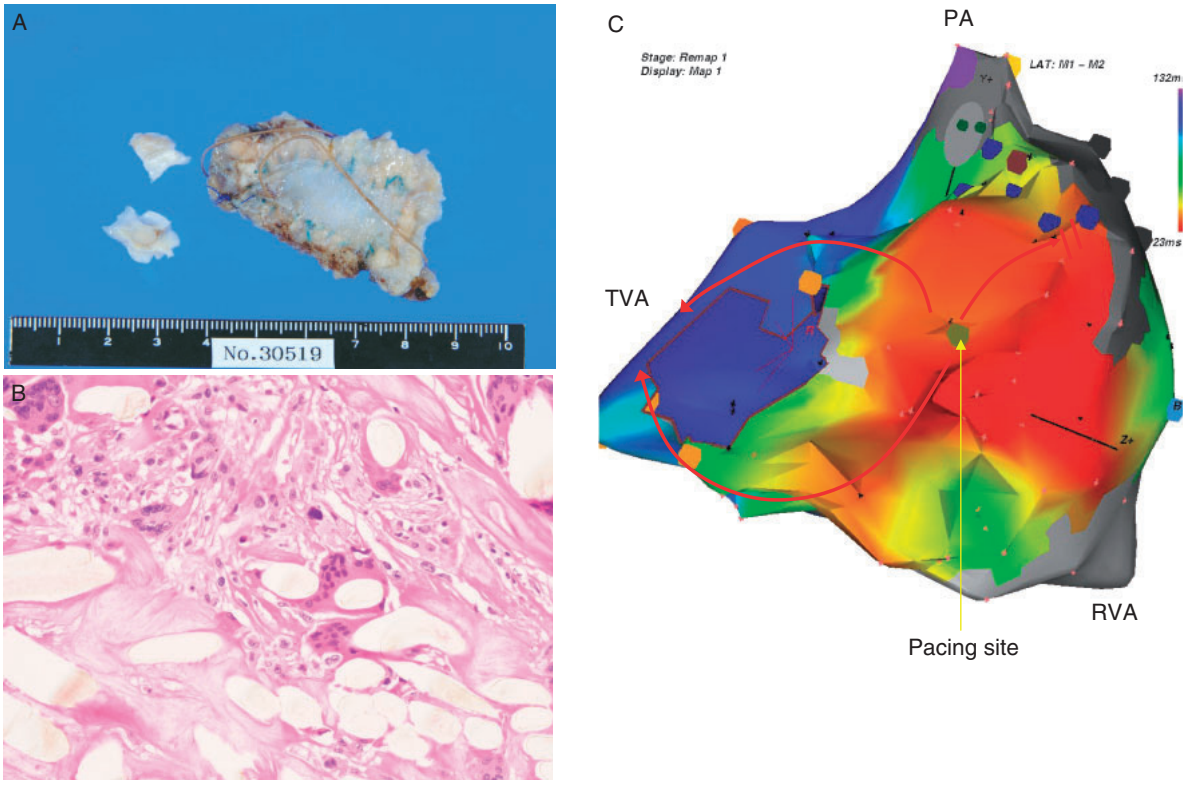

Figure 4
A: The old patch and pulmonary valve removed during the operation. **B**: Scar tissue with fibrosis and infiltration by polynuclear giant cells was observed. C: The activation map during RV pacing after the operation. The scar at the RVOT was connected to the pulmonary artery with a patch and conduction through the cryoablation line was observed.

from the RVA. During the tachycardia, the activation wavefront was found to revolve in a counterclockwise manner around the patch in the RVOT (**Figure 2B**). The local bipolar electrogram along the isthmus between the RVOT patch and pulmonary artery demonstrated a fractionated mid-diastolic potential (MDP), entrainment with concealed fusion and post pacing interval at the site equal to the tachycardia cycle length (**Figure 3A**). RF current was delivered at a power of 30 to 50 W with a target temperature of 55 °C at the site. RF application in a line between the RVOT patch and pulmonary artery interrupted the tachycardia. No VT was induced by programmed ventricular stimulation, and conduction block through the ablation line was confirmed by the activation map during RV pacing after the ablation. However, VT recurred 6 months after the procedure. During the 2nd electrophysiological study, the same VT that was previously treated and a non-clinical VT in which the activation wavefront revolved in a clockwise manner around the RVOT patch were induced. An RF application along the same line as that in the 1st procedure was performed with an 8 mm tip ablation catheter. After the ablation, non-inducibility and conduction block through the ablation line were reconfirmed. However, a second recurrence occurred 2 months after the 2nd session.

Two-dimensional echocardiography after the second recurrence demonstrated a mean pressure gradient of 49 mmHg from the RVOT to the main pulmonary artery. Cardiac catheterization revealed a pulmonary artery pressure of 27/8 mmHg, right ventricular pressure of 96/−2 mmHg, and right atrial pressure of 6 mmHg.

Surgical procedure and findings

The patient underwent a pulmonary valve replacement and reconstruction of the RVOT to treat the VT refractory to RFCA and severe pressure gradient in the RVOT. The RVOT patch was removed. The endocardial site of the RVOT patch was completely covered with scar tissue. The pulmonary valve was so severely thickened that it had the appearance of a bicuspid valve. After the pulmonary valve replacement using a Mosaic bioprosthesis, an extension of the RVOT with a patch extending from the pulmonary artery bifurcation to the right ventricular infundibulum was performed. Ventricular cryoablation was performed for 3 minutes at −70 °C along a

line between the ventriculotomy site and tricuspid annulus. The old patch removed during the operation included scar tissue with fibrosis and infiltration by polynuclear giant cells. (**Figure 4A**, **B**) In the postoperative electrophysiological study, the scar in the RVOT was connected to the pulmonary artery with the transannular patch which produced conduction block within the reentry circuit of the VT; however, conduction through the cryoablation line was observed (**Figure 4C**). Programmed ventricular stimulation of up to triple extra stimuli confirmed the noninducibility of the VT. The patient has been free from any VT recurrence during 6 months of follow up.

Discussion

Macroreentry near the ventriculotomy scar in the RVOT is thought to be one of the mechanisms of VT occurring after TOF repairs. In such cases, an RF application in a line between the ventriculotomy scar and tricuspid annulus or pulmonary artery has been used to successfully treat tachycardia.[7,8)]

Misaki et al. identified a clockwise macroreentrant circuit around a prior ventriculotomy scar by epicardial mapping, and reported a favorable outcome of the resection of the origin of the VT foci in the RVOT and cryoablation. In the resected RVOT wall around the scarred suture line of the previous operation, the surviving myocytes formed islet-like groups that were connected to each other with a few small myocyte branches and were irregularly scattered in the middle layer.[11)] Polynuclear giant cells observed in the removed patch suggested that an allergic reaction to the Teflon patch had occurred.

In the present case, the VT was due to macroreentry around the RVOT patch. However, VT was not eliminated with RF application along a line between the patch and pulmonary artery. During the RF application, a maximum power of about 30 W was achieved and the local bipolar electrogram amplitude became reduced by about 50%. Fibrous tissue and infiltration by polynuclear giant cells were observed on the RVOT patch which was removed during the reoperation. RF energy adequate to create permanent conduction block may not have reached the myocardium surrounded by such tissue.

Oechslin et al. reported 60 cases that underwent a reoperation after a previous TOF repair. The prevalence of sustained VT decreased from 33% to 7% after the reoperation, and the additional effects of cryoablation were not clear in that study.[12)]

Therrien et al. reported that the incidence of VT decreased from 22% to 9% after a pulmonary valve replacement for pulmonary regurgitation in 70 cases. In their study, none of their 9 patients who underwent concomitant ventricular cryoablation had recurrent VT, whereas 2 of 6 who had not undergone ventricular cryoablation at the time of surgery had recurrent VT.[13)] Because the VT recurred in spite of two RFCA sessions along a line between the RVOT patch and pulmonary artery, we performed cryoablation between the RVOT patch and tricuspid annulus, which was another isthmus of the reentry circuit. In the postoperative electrophysiological study, no effects of the cryoablation were observed. Right ventricular hypertrophy may be one reason why conduction block was not achieved by cryoablation. Careful observation for any new tachycardia due to that incomplete cryoablation line would be warranted.

There was already a moderate pressure gradient in the RVOT before the 1st ablation session but it increased after the 2nd session. In an experimental study, the pathological examination after the RFCA has revealed coagulation necrosis.[14)] By 8 weeks, the necrotic zone has been replaced by fatty tissue, cartilage, and fibrosis.[15)] The chronic lesion shows evidence of significant contraction and volume loss.[16)] The total RF energy delivered in the RVOT during the 1st and 2nd sessions was 93464J. The repeated procedures may have contributed to the increase in the pressure gradient of the RVOT. During the surgery, a ventricular resection was not performed, and therefore a pathological examination of the RF lesion was not possible.

Conclusion

We presented a patient with VT refractory to RFCA after TOF repair. The mechanism of the VT was macroreentry around the RVOT patch. The VT was successfully treated by making an anatomical conduction block in the reentry circuit with a transannular patch.

References

1) Murphy JG, Gersh BJ, Mair DD, et al: Long-term outcome in patients undergoing surgical repair of tetralogy of Fallot. N Engl J Med 1993; 329: 593–599
2) Nollert G, Fischlein T, Bouterwek S, et al: Long-term survival in patients with repair of tetralogy of Fallot: 36-year follow-up of 490 survivors of the first year after surgical repair. J Am Coll Cardiol 1997; 30: 1374–1383
3) Waien SA, Liu PP, Ross BL, et al: Serial follow-up of adults with repaired tetralogy of Fallot. J Am Coll Cardiol 1992; 20: 295–300
4) Nakazawa M, Shinohara T, Sasaki A, et al: Arrhythmias

late after repair of tetralogy of Fallot: A Japanese multicenter study. Circ J 2004; 68: 126–130
5) Gatzoulis MA, Balaji S, Webber SA, et al: Risk factors for arrhythmia and sudden cardiac death late after repair of tetralogy of Fallot: a multicentre study. Lancet 2000; 356: 975–81
6) Rostock T, Willems S, Ventura R, et al: Radiofrequency catheter ablation of a macroreentrant ventricular tachycardia late after surgical repair of tetralogy of Fallot using the electroanatomic mapping (CARTO). PACE 2004; 27[Pt. I]: 801–804
7) Chinushi M, Aizawa Y, Kitazawa H, et al: Successful radiofrequency catheter ablation for macroreentrant ventricular tachycardias in a patient with tetralogy of Fallot after corrective surgery. PACE 1995; 18[Pt. I]: 1713–1716
8) Horton PR, Canby RC, Kessler DJ, et al: Ablation of ventricular tachycardia associated with tetralogy of Fallot: Demonstration of bidirectional block. J Cardiovasc Electrophysiol 1997; 8: 432–435
9) Papagiannis J, Kanter RJ, Wharton M: Radiofrequency catheter ablation of multiple haemodynamically unstable ventricular tachycardias in a patient with surgically repaired tetralogy of Fallot. Cardiol Young 1998; 8: 379–382
10) Biblo LA, Carlson MD: Transcatheter radiofrequency ablation of ventricular tachycardia following surgical correction of tetralogy of Fallot. PACE 1994; 17: 1556–1560
11) Misaki T, Tsubota M, Watanabe G, et al: Surgical treatment of ventricular tachycardia after surgical repair of tetralogy of Fallot: Relation between intraoperative mapping and histological findings. Circulation 1994; 90: 264–271
12) Oechslin EN, Harrison DA, Harris L, et al: Reoperation in adults with repair of tetralogy of Fallot: Indications and outcomes. J Thorac Cardiovasc Surg 1999; 118: 245–51
13) Therrien J, Siu SC, Harris L, et al: Impact of pulmonary valve replacement on arrhythmia propensity late after repair of tetralogy of Fallot. Circulation 2001; 103: 2489–2494
14) Huang SK, Bharati S, Graham AR, et al: Closed chest catheter desiccation of the atrioventricular junction using radiofrequency energy: A new method of catheter ablation. J Am Coll Cardiol 1987; 9: 349–358
15) Huang SK, Bharati S, Lev M, et al: Electrophysiologic and histologic observations of chronic atrioventricular block induced by closed-chest catheter desiccation with radiofrequency energy. PACE 1987; 10: 805–816
16) Haines DE: The biophysics and pathophysiology of lesion formation during radiofrequency catheter ablation. In Zipes DP, Jalife J, eds: Cardiac Electrophysiology: From cell to bedside, 4th edition. Elsevier Inc, Philadelphia, 2004, p.1022