Induction of tachycardia confined within a pulmonary vein by electrical cardioversion of atrial fibrillation: Is it proof of reentry?

Mauro Toniolo, MD,[*†] Jorge Figueroa, MD,[*] Sergio Castrejòn-Castrejòn, MD,[*] Jose Luis Merino, MD, PhD[*]

From the [*]Arrhythmia and Robotic Cardiac Electrophysiology Unit, IdiPaz, Hospital Universitario "La Paz", Madrid, Spain and [†]Division of Cardiology, University Hospital "S. Maria della Misericordia", Udine, Italy.

Introduction

Because paroxysmal atrial fibrillation (AF) is most often triggered by the sources inside the pulmonary veins (PVs), radiofrequency (RF) catheter ablation of AF is performed. In a lot of cases, after PVs isolation AF persists and often it can be necessary perform a direct current (DC) cardioversion.

Description

A 66-year-old man with no structural heart disease and daily episodes of paroxysmal atrial fibrillation (AF) was referred for pulmonary vein (PV) isolation. The patient had hypertension and no other clinical risk factor related to AF. A quadripolar catheter (Viking, Bard; Boston Scientific, Marlborough, MA), a 20-pole circular catheter (Optima; St Jude Medical, St. Paul, MN) and an irrigated-tip ablation catheter (Cool Flex; St Jude Medical) were introduced in the coronary sinus (CS), PVs, and left atrium (LA), respectively. The patient was in sinus rhythm at the beginning of the procedure but developed AF during catheter manipulation within the LA. Electrical cardioversion restored sinus rhythm, but the patient had early AF reinitiation triggered by ectopy from the left superior PV on several occasions. The ablation procedure was initiated during ongoing AF, and the left inferior PV was isolated by radiofrequency (RF) application at the vein ostium with no effect on AF. RF application at the left superior PV ostium resulted in AF termination with early reinitiation on several occasions. Finally, this vein was disconnected by additional RF applications, but the atria remained in AF (Figure 1). Regular spontaneous automaticity within the vein was demonstrated during ongoing AF. A 200-J biphasic shock terminated AF and restored sinus rhythm in the LA but induced a fast and irregular tachycardia confined within the left superior PV (Figure 2). The tachycardia was induced with no apparent premature atrial contraction. Tachycardia within the vein spontaneously terminated after 20 minutes and could be reproducibly induced by rapid pacing within the vein with no conduction to the LA and spontaneous termination (Figure 3). The patient was discharged with orders for flecainide therapy for 1 month and observed via outpatient visits at 1, 6, and 12 months. He also underwent 24-hour electrocardiogram Holter monitoring at 6-month follow-up. No AF recurrences were documented, and the patient was asymptomatic during the whole follow-up.

Ventricular fibrillation induction by direct current (DC) shocks is a well-known phenomenon.[1] Induction of AF by DC shocks in patients with ventricular arrhythmias has been also demonstrated in the past.[2] The present report shows a similar phenomenon of tachycardia induced by a DC shock within a great cardiac vein.

A lower limit of vulnerability exists for both ventricular and atrial myocardium:[3] this limit is the minimum voltage required by an electrical stimulus to induce fibrillation during the vulnerable period. It was noted in the 1960s that there was also an upper limit to the strengths of shocks delivered during the vulnerable period that induce ventricular fibrillation.[4] It was also observed that the strengths of these shocks at the upper limit of vulnerability were approximately equivalent to the shocks at the defibrillation threshold.[4]

Structural and electrical discontinuity has been widely invoked to explain the myocardial response to electrical shock.[5] It is acknowledged that defibrillating shocks applied to the surface of the heart would not depolarize a sufficient volume of tissue to achieve cardioversion if myocardium behaved as a continuum.[6]

There are several theories of the mechanism of defibrillation that are derived from ventricular defibrillation. It is thought that shocks defibrillate by altering the potential difference across the cell membrane, that is, the transmembrane potential.[3]

Shock-induced transmembrane potential change ("virtual electrode")[7] distant from the site of current injection may be induced by unequal anisotropy of intracellular and

KEYWORDS Atrial fibrillation; Catheter ablation; Electrical cardioversion; Pulmonary vein arrhythmogenicity; Reentry mechanism

ABBREVIATIONS **AF** = atrial fibrillation; **CS** = coronary sinus; **LA** = left atrium; **PV** = pulmonary vein; **RF** = radiofrequency

(Heart Rhythm Case Reports 2015;1:225–228)

Conflicts of interest: J.L.M. is a consultant to Magnetecs, and he receives honoraria for providing medical and technical training for St. Jude Medical.

Address reprint requests and correspondence: Dr. Mauro Toniolo c/o Dr. Jose L. Merino, Unidad de Arritmias y Electrofisiología Cardiaca Robotizada (1 planta), Hospital General La Paz, Paseo de la Castellana 261, 28046 Madrid, Spain. E-mail address: mautoniolo@libero.it.

http://dx.doi.org/10.1016/j.hrcr.2015.03.009

KEY TEACHING POINTS

- The definitive proof that reentry is the mechanism in at least a subset of PV tachycardias is still lacking. In this case report, the underlying mechanisms of induction of the observed tachycardia, add another piece to the puzzle of PV tachycardia mechanisms.
- When we perform an electrical cardioversion of AF, we can often see the restoration of sinus rhythm but a new onset of AF after only a few seconds. The mechanism of this arrhythmogenicity could be due to the induction of fibrillation within a PV following a DC shock.
- Fibrillation induction in the atria and the ventricles by DC shocks has been reported in the past. This is the first report showing fibrillation induction within a PV by a DC shock. Therefore this report support the inclusion of the thoracic veins as other cardiovascular structures with this proarrhythmic risk.

extracellular spaces.[8] myofiber curvature,[9] discontinuity associated with gap junctions,[10] and fiber narrowing.[11] All these factors promote reentry by providing areas of conduction block, and this attribute may support reentry as the underlying mechanism of arrhythmias induced by DC shocks. Moreover, induction of AF by DC shock has been associated with P-wave dispersion, which is also a factor known to be related to reentry.[12] In addition, muscular discontinuities and abrupt changes in fiber orientation were seen in most PV-LA segments, creating significant substrates for reentry.[13]

Tachycardias confined within a PV have been reported in the recent past,[14,15] but the mechanism of these arrhythmias and the critical structures required to sustain them are still unclear.

Induction of PV tachycardia by DC shock is not a definitive proof of reentry. However, the underlying mechanisms as the factors associated with the virtual electrode of DC shocks, the PV tachycardia induction by programmed electrical stimulation, and their association with reentry precondition (short refractory period, heterogeneity of refractoriness, and slow conduction) supports reentry as one of the most likely mechanisms of PV tachycardia.

Finally, demonstration of induction of fibrillation within a PV following DC cardioversion raises the questions of whether this is a rare phenomenon and whether it could be responsible for some AF episodes developed very soon after DC cardioversion.

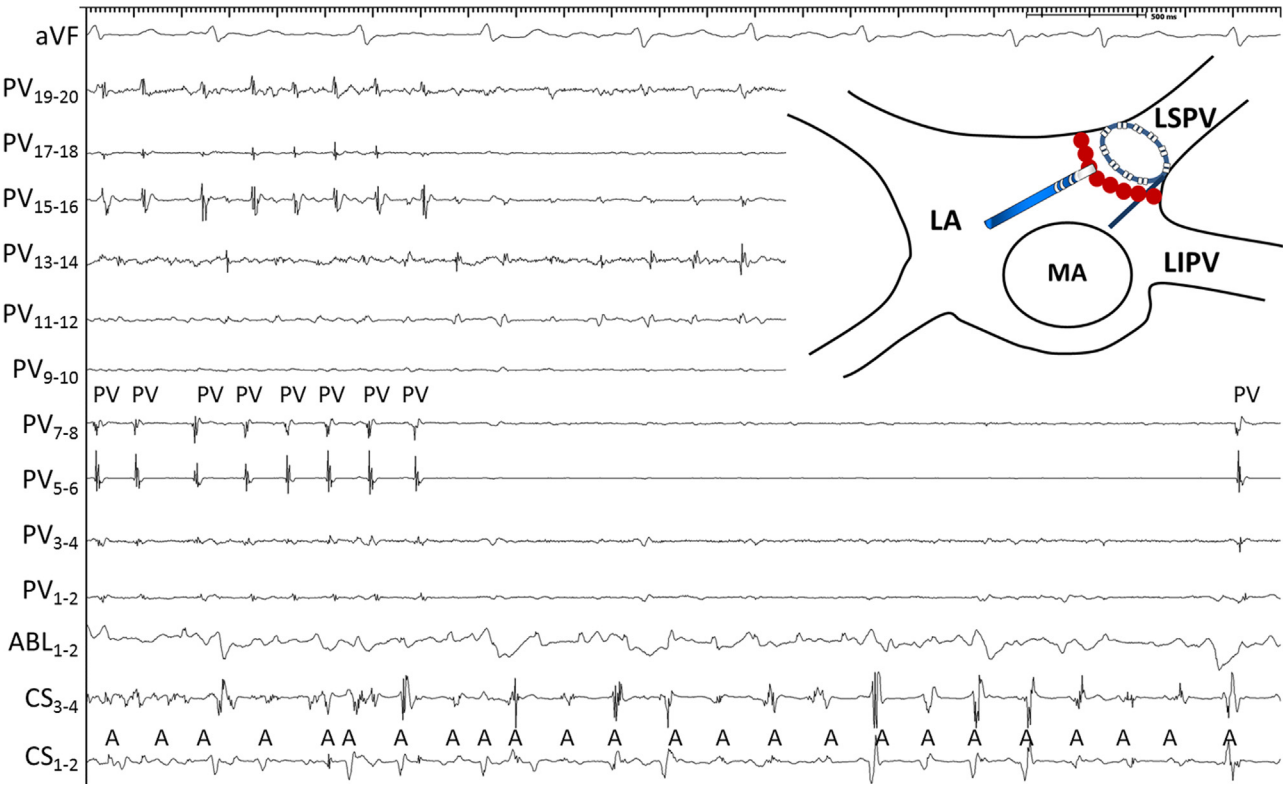

Figure 1 Electrical disconnection of the left superior pulmonary vein (PV) by radiofrequency (RF) application. From top to bottom, the figure shows electrocardiogram lead aVF and intracardiac recordings at 100 mm/s from the ablation catheter (ABL_{1-2}) placed at the left superior PV ostium, a circular 20-pole catheter (PV_{1-2} to PV_{19-20}) placed within the left superior PV, and a tetrapolar catheter (CS_{1-2} and CS_{3-4}) placed in the coronary sinus. RF application during ongoing atrial fibrillation (AF) results in the sudden disappearance of the PV electrogram (apparent on the recordings from all bipoles except PV_{11-12} and PV_{13-14}) with AF persistence in the left atrium (LA) on the coronary sinus more apparent as far-field electrograms on the recordings from bipoles PV_{11-12} and PV_{13-14}. The figure also shows a PV ectopic beat at the end of the tracing and a schematic showing the catheter position. A = atrium; LIPV = left inferior PV; LSPV = left superior PV; MA = mitral annulus.

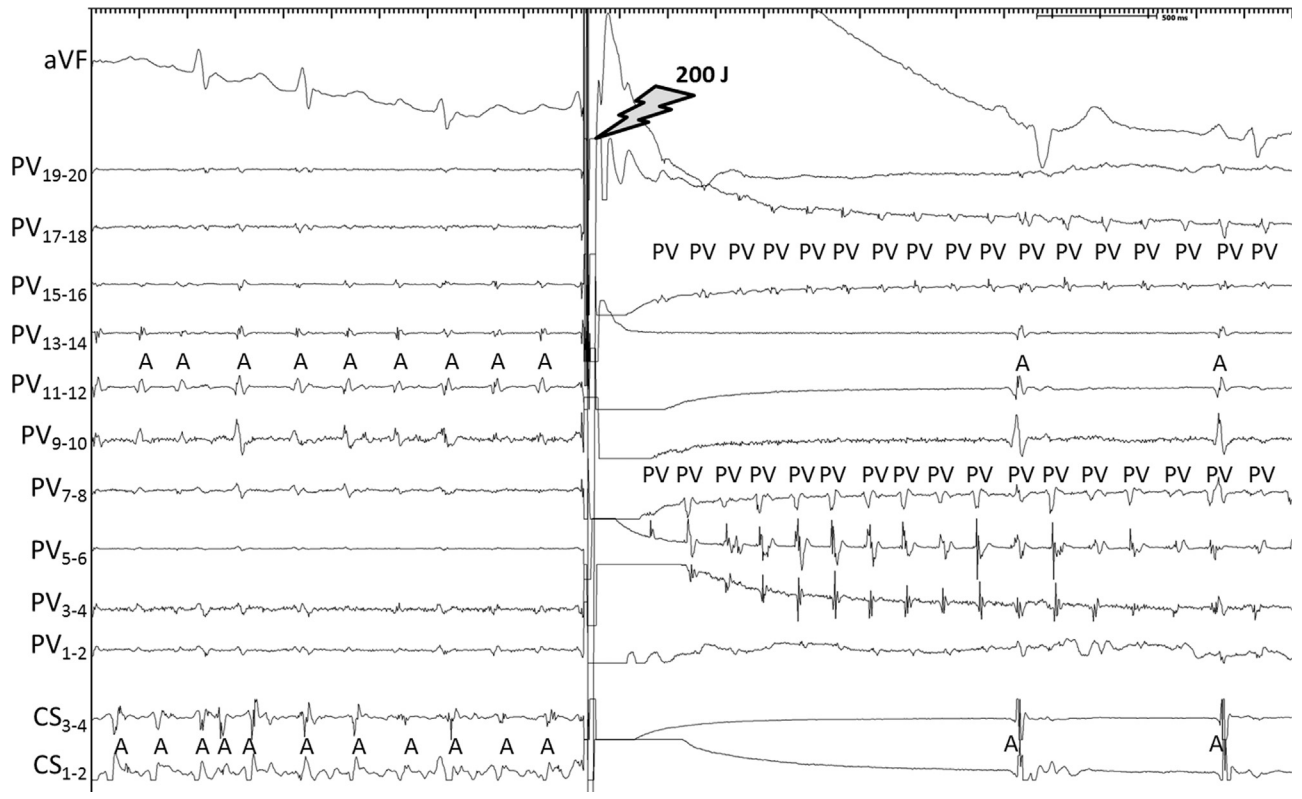

Figure 2 Tachycardia induction in the left superior pulmonary vein following direct current cardioversion. Atrial fibrillatory activity is replaced by sinus rhythm activity after cardioversion (arrow) on the recordings obtained from the coronary sinus and from bipoles PV_{9-10} to PV_{13-14} of the pulmonary vein catheter (far-field activation). Small far-field atrial fibrillatory activity is replaced by fibrillatory activity within the vein after cardioversion on the recordings from all bipoles but PV_{11-12} and PV_{13-14} (more apparent on bipoles PV_{3-4}, PV_{5-6}, and PV_{7-8}). Abbreviations as defined in Figure 1.

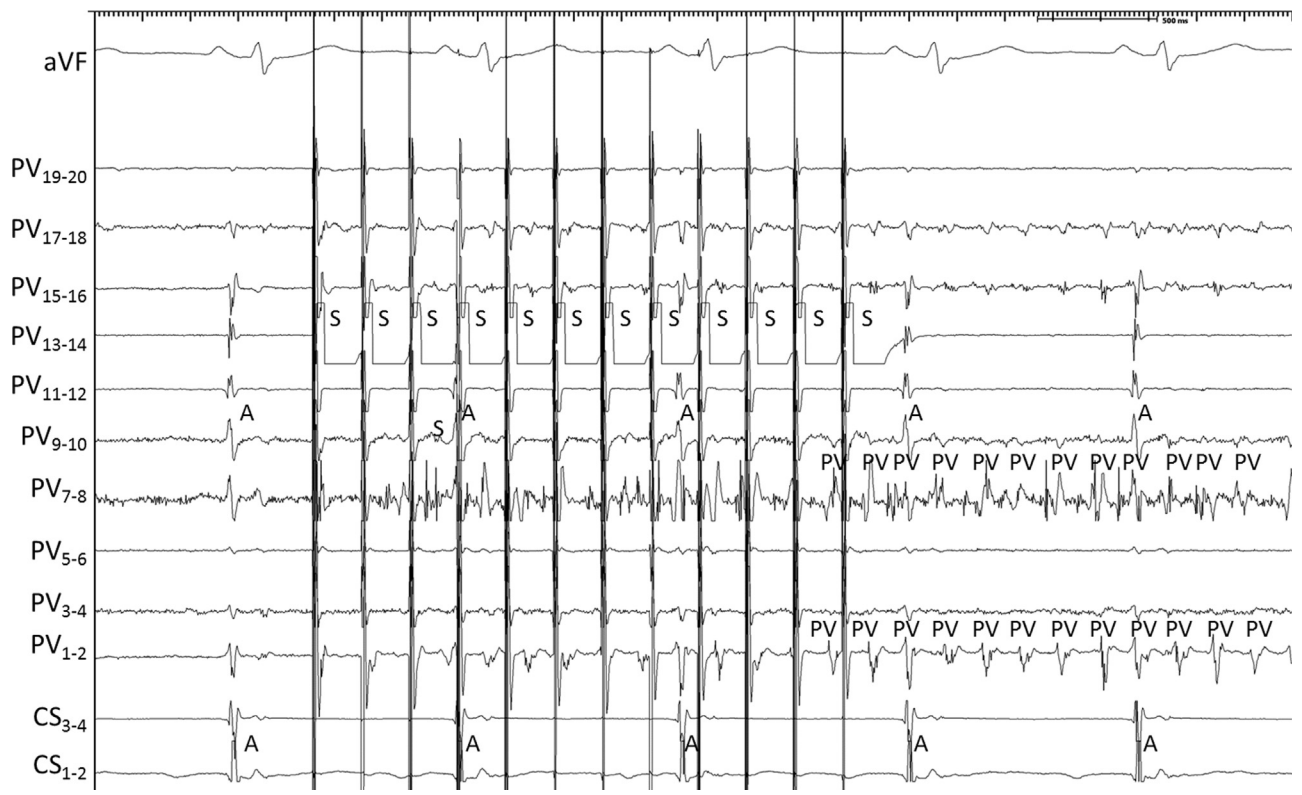

Figure 3 Tachycardia induction within the left superior pulmonary vein, with no conduction to the left atrium, by rapid pacing. Following pacing, sinus rhythm far-field activity is recorded from bipoles PV_{9-10}, PV_{11-12}, PV_{13-14} , and PV_{15-16} concurrently with fibrillatory activity within the pulmonary vein from bipoles PV_{1-2}, PV_{15-16}, and PV_{17-18}. Abbreviations as defined in Figure 1.

References

1. Lauer MR, Young C, Liem LB, Hoboni L, Peterson J, Goold P, Sung RJ. Ventricular fibrillation induced by low-energy shocks from programmable implantable cardioverter-defibrillators in patients with coronary artery disease. Am J Cardiol 1994;73:559–563.
2. Florin TJ, Weiss DN, Peters RW, Shorofsky SR, Gold MR. Induction of atrial fibrillation with low-energy defibrillator shocks in patients with implantable cardioverter defibrillators. Am J Cardiol 1997;80:960–962.
3. Ideker RE, Chattipakorn N, Gray RA. Defibrillation mechanisms: the parable of the blind men and the elephant. J Cardiovasc Electrophysiol 2000;11: 1008–1013.
4. Fabiato A, Coumel P, Gourgon R, Saumont R. Le seuil de réponse shynchrone des fibres myocardiques. Application à la comparaison expérimentale de l'efficacité des differéntes formes de chocs électriques de défibrillation. Arch Mal Coeur 1967;60:527–544.
5. Fast VG, Rohr S, Gillis AM, Klèber AG. Activation of cardiac tissue by extracellular electrical shocks: formation of "secondary sources" at intercellular clefts in monolayers of cultured myocytes. Circ Res 1998;82:375–385.
6. Trayanova N. Far-field stimulation of cardiac tissue. Herz Elektrophysiol 1999;10:137–148.
7. Wikswo JP Jr, Lin SF, Abbas RA. Virtual electrodes in cardiac tissue: a common mechanism for anodal and cathodal stimulation. Biophys J 1995;69:2195–2210.
8. Sepulveda NG, Wikswo JP Jr. Electric and magnetic fields from two-dimensional anisotropic bisyncytia. Biophys J 1987;51:557–568.
9. Trayanova N, Skouibine K. Modelling defibrillator: effects of fiber curvature. J Electrophysiol 1998;31:23–29.
10. Trayanova N. Discrete versus syncytial tissue behaviour in a model of cardiac stimulation, II: results of simulation. IEEE Trans Biomed Eng 1996;43: 1141–1150.
11. Sobie EA, Susil RC, Tung L. Generalized activating function for predicting virtual electrodes in cardiac tissue. Biophys J 1997;73:1410–1423.
12. Ozdemir O, Soylu M, Demir AD, et al. Does p-wave dispersion predict the atrial fibrillation occurrence after direct-current shock therapy? Angiology 2006;57:93–98.
13. Kumagai K, Ogawa M, Noguchi H, Yasuda T, Nakashima H, Saku K. Electrophysiologic properties of pulmonary veins assessed using a multielectrode basket catheter. J Am Coll Cardiol 2004;43:2281–2289.
14. Knight BP, Oral H, Morady F. Paroxysmal fibrillation within an isolated pulmonary vein. Circulation 2002;106:1426–1427.
15. Rosso R, Kistler PM. Sinus rhythm with isolated pulmonary vein fibrillation. Heart Rhythm 2010;96:322.