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

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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 also been 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.7

However, induced transmembrane potential change ("virtual electrode")8 distant from the site of current injection may be induced by unequal anisotropy of intracellular and extracellular ions.9,10
extracellular spaces, myofiber curvature, discontinuity associated with gap junctions, and fiber narrowing.

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. In addition, muscular discontinuities and abrupt changes in fiber orientation were seen in most PV-LA segments, creating significant substrates for reentry.

Tachycardias confined within a PV have been reported in the recent past, 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 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.
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