Reentry in a Pulmonary Vein as a Possible Mechanism of Focal Atrial Fibrillation

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Reentry in a Pulmonary Vein. The case of an 18-year-old woman with recurrent idiopathic catecholamine-sensitive paroxysmal atrial fibrillation is reported. Recordings of multiple initiations of atrial fibrillation at the proximal part of the right superior pulmonary vein suggested local reentry in the vein as the mechanism of atrial fibrillation. A single radiofrequency pulse delivered at this site resulted in definite cure of the arrhythmia. (J Cardiovasc Electrophysiol, Vol. 15, pp. 824-828, July 2004)

Introduction

Pioneering work by Jais et al.1 and Haissaguerre et al.2 have shown that most cases of atrial fibrillation (AF) have a focal origin in the pulmonary veins. This finding has had a dramatic impact on the nonpharmacologic management of AF with catheter ablation techniques. The mechanism of initiation of focal AF in humans is unknown. Triggered activity initially was considered to be the most likely mechanism of these focal discharges, although abnormal automaticity also may play a role.3 In the patient reported here, we suggest reentry in a pulmonary vein as the most likely mechanism of focal AF.

Case Report

An 18-year-old woman was referred for radiofrequency ablation because of recurrent, paroxysmal AF. She reported multiple arrhythmic episodes, mainly triggered by stress, during the year preceding her hospitalization. Result of echocardiography and thyroid function tests were normal. However, during exercise testing, the arrhythmia could be reproducibly provoked and consisted of bouts of AF lasting from a few seconds to 30 seconds. Maximal heart rate during AF was 190 beats/min. Exercise testing, the arrhythmia could be reproducibly provoked and consisted of bouts of AF lasting from a few seconds to 30 seconds. Maximal heart rate during AF was 190 beats/min.

Electrophysiologic evaluation was performed in November 2000, before Lasso mapping catheters became available in our laboratory. Decapolar electrode catheters were introduced in the right atrium and the coronary sinus. A quadripolar electrode catheter was positioned at the His-bundle area, and a 7-French ablation catheter (EP Technologies, Inc.) was introduced in the left atrium through a patent foramen ovale.

At baseline, sinus rate was 80 beats/min, and only rare atrial extrasystoles were present. No arrhythmias could be induced with rapid right atrial pacing. After infusion of isoproterenol that resulted in an increase of sinus rate to 110 beats/min, multiple episodes of atrial arrhythmias consisting of single extrasystoles up to short-lasting (<20 s) atrial tachycardia (AT) at a rate of 300 beats/min were observed. Activation mapping of the right and left atria suggested that the AT originated from the right superior pulmonary vein (RSPV).

During sinus rhythm, the electrogram recorded at the ostium of the RSPV (Fig. 1A, first complex) consistently showed two components: (1) a first potential (labeled 1) assumed to represent the far-field potential originating from the contiguous posterior wall of the right atrium; and (2) a second potential assumed to represent a split pulmonary vein potential (FVP) consisting of a first fast, large deflection (labeled 2) followed by a fragmented deflection of low amplitude (labeled 3). Occasionally, a third component, originally believed to represent a “PVP extrasystole” occurred (labeled 3′) at constant and short coupling intervals. These “PVP extrasystoles” invariably occurred when component 3 of the PVP failed to appear (due to block between components 2 and 3). The “PVP extrasystoles” were sometimes isolated (Fig. 1A) and not associated with an electrical activity in the right atrial electrograms (“concealed PVP”). In other instances, they were followed by two PVP activities (components 2′ and 1′) and constituted the first complex of a triplet that apparently gave origin to a single response in the right atrial electrogram (Fig. 2, top panel). The morphology and timing of these two PVP activities (2′ and 1′) suggested that they were retrograde activations of components 2 and 1 observed in sinus rhythm. Finally, in other instances, a repetitive activity in the RSPV constituted by a regular succession of triplets (components 3′, 2′, 1′) was recorded, with each triplet associated with a single atrial activity in the right atrial electrogram (Fig. 3). Six such episodes of focal AT (cycle length 180–200 ms) could be recorded, all lasting <20 seconds. During all six episodes, the “PVP extrasystole” preceded by 120 ms the earliest right atrial activity recorded in the high right atrium, and its occurrence invariably was preceded by abolition of component “3” of the RSPV electrogram.

In addition, two episodes of nonsustained focal AT that had the same rate and activation sequence as the AT described earlier were documented (Fig. 4). In both instances, the AT was triggered by a single atrial extrasystole that apparently originated close to the right high atrium. The electrical activity in the RSPV that resulted from this atrial extrasystole was identical to that previously observed, namely, the occurrence of the “PVP extrasystole” invariably was associated with abolition of component “3” of the RSPV electrogram.

A single radiofrequency pulse (25–30 W, mean temperature 41°C) was administered for 35 seconds during sinus rhythm at the ostium of the RSPV where components 1′+2′+3’ were recorded. Abolition of the component “3” potential was noted after ablation (Fig. 1B). During a 40-minute follow-up period, no atrial tachyarrhythmias or ectopic activity in the RSPV could be observed before and after rapid atrial pacing following high doses of isoproterenol (sinus rate up to 180 beats/min). A few single atrial extrasystoles were recorded after the ablation catheter.
Figure 1. A: “Concealed extrasystoles” originating from the right superior pulmonary vein (RSPV). During sinus rhythm, the electrogram recorded at the baseline state is the first complex in the panel. This electrogram shows a constant morphology and consists of two components: (1) a first potential (“1”) assumed to represent the far-field potential originating from the contiguous right posterior wall of the high right atrium; and (2) a second potential assumed to represent a split pulmonary vein potential (PVP) consisting of a first fast deflection having a large amplitude (component 2) followed by a fragmented deflection of low amplitude (component 3). “PVP extrasystoles” (component 3’) occur at constant and short coupling intervals on the three subsequent complexes. These “PVP extrasystoles” invariably occur each time component 3 of the PVP does not appear. When occurring in an isolated manner, they are not associated with atrial activity in the right atrial electrogram. ABLb, ABLu = bipolar and unipolar recordings from the ablation catheter in the ostium of the RSPV; RA1 = electrogram from the high right atrium. B: RSPV electrogram after successful ablation. After delivery of a single radiofrequency pulse at the ostium of the RSPV, components 1 and 2 remain unchanged while component 3 is abolished.

was removed from the RSPV. These extrasystoles had an activation sequence identical to that of the atrial extrasystole previously assumed to originate close to the high part of the right atrium (arrow in Fig. 4).

During 36-month follow-up, the patient has remained asymptomatic without any medication. Only rare atrial extrasystoles have been documented during repeated Holter monitoring and maximal exercise testing. The P morphology of the latter is consistent with a high right atrial origin. AT has not been observed.

Discussion

All the episodes of AT recorded in our patient had the typical features of repetitive “focal AF” described by the Bordeaux group.1,2 (1) Despite a surface ECG suggesting AF, the tachycardia recorded in the RSPV was, in fact, a regular AT of very rapid rate (300 beats/min). (2) All instances of AT were initiated by a so-called “PVP extrasystole.”2 (3) “Concealed PVP extrasystoles,” i.e., PVP not conducted to the atrium, were observed. However, three findings suggested that the electrical complexes originally assumed to represent a “PVP extrasystole” (component 3’) as well as the “focal AF” in our patient were, in fact, due to reentry within the RSPV. (1) Slow conduction was present in the RSPV as suggested by delayed conduction between components 1 and 2 as well as between component 2 and 3. (2) Component 3’ occurred only when conduction block within the PVP (between the 2 and 3 components) occurred. (3) The short conduction time between components 2 and 3 and the long conduction time between components 2 and 3’ suggested longitudinal dissociation into a fast and a slow pathway, respectively, within the RSPV.

To actually prove reentry, pacing during tachycardia should have demonstrated entrainment of AT. However, the latter could not be achieved because AT was invariably short-lasting. Also, initiation of AT with pacing (extrastimulation) that would have provided further support for reentry was not performed. However, in two instances, we were lucky to record the initiation of AT within the RSPV by single atrial extrasystoles originating outside the RSPV (presumably from the right atrium). This “spontaneous extrastimulation” strongly argues in favor of a reentrant mechanism.

Two reentrant circuits can be hypothesized to explain the mechanism of AF in our patient (Fig. 2). The first one involves “macroreentry” within the circumference of the RSPV ostium (middle panel). The other one involves “microreentry” along the proximal aspect of the RSPV (bottom panel).
A detailed explanation is given in the legend of Figure 2. Without more detailed mapping of the RSPV (as it would have been obtained with a Lasso catheter), it is difficult to distinguish between the two. Interestingly, definite cure of the arrhythmia in our patient was achieved after a single radiofrequency pulse that resulted in conduction block between components 2 and 3 and not between components 1 and 2, as is generally observed when a PVP is disconnected from the left atrium with radiofrequency ablation. This suggests that the segment between 2 and 3 represents a critical common pathway of the reentrant circuit and/or the only electrical connection between the left atrium and the RSPV.

Despite the huge amount of work performed in the cure of AF with catheter ablation techniques since the pioneering work of the Bordeaux group, the mechanism of arrhythmogenicity in the pulmonary veins in man is still unknown. Hocini et al. studied electrical conduction in canine pulmonary veins. They found that electrograms recorded in their animal model were similar to those recorded in patients with lone AF.
Figure 3. Spontaneous occurrence of rapid atrial tachycardia. During sinus rhythm, a repetitive activity consisting of a regular succession of triplets (components $3', 2', 1'$) is recorded in the RSPV, with each triplet associated with single atrial activity in the right atrial electrogram. A focal atrial tachycardia (cycle length 180-200 ms) results from this repetitive firing in the pulmonary vein (a similar event of sequence was recorded at five additional instances). Note again that occurrence of component $3'$ is dependent on abolition of component “3′” of the right superior pulmonary vein (RSPV) electrogram. ABLu = unipolar recording from the ablation catheter in the ostium of the RSPV. Electrograms from the right atrium (RA2, RA3, RA4) and the atrioventricular (AV) junction area also are shown. Other abbreviations as in Figure 1.

Figure 4. Initiation of atrial tachycardia due to spontaneous atrial extrasystoles. An episode of atrial tachycardia identical to that recorded in Figure 3 is triggered by a single atrial extrasystole (arrow), which apparently originates close to the high right atrium. The electrical activity in the right superior pulmonary vein (RSPV) that results from this atrial extrasystole consists of only two components (1+2). Note again that occurrence of the “pulmonary vein potential (PVP) extrasystole” is associated with abolition of component 3 of the RSPV electrogram. Abbreviations as in previous figures.
They concluded that the architecture of muscular sleeves in the pulmonary veins might facilitate reentry and arrhythmias. Using high-resolution optical mapping, Arora et al.\(^6\) studied coronary-perfused, isolated whole-atrial preparations from normal dogs. They found that the normal pulmonary vein seems to have the necessary substrate to support reentry as well as focal activity. Conduction was found to be significantly slower at the proximal pulmonary vein than in the rest of the left atrium, with decremental conduction and variable entrance block observed at faster atrial pacing rates. In man, Shah et al.\(^7\) obtained strong evidence in support of circus movement reentry within the superior vena cava as the arrhythmia mechanism in their patient. They concluded that a similar mechanism may exist in the pulmonary veins. More recently, Takahashi et al.\(^8\) investigated the inducibility and the mechanism of sustained pulmonary vein tachycardia after achievement of pulmonary vein isolation. Their results suggest that reentry is one mechanism of pulmonary vein arrhythmogenicity. However, by study design, they did not assess the mechanism of AF initiation in pulmonary veins before ablation. In our patient, the limited mapping data available with our single ablation catheter made it impossible to prove that reentry and not abnormalities in automaticity or triggered rhythms was responsible for the initiation of AF. Nonetheless, we believe that our patient is the first human case suggesting a reentrant mechanism in a pulmonary vein as a mechanism of focal AF.

References