Periodic conduction and 2:1 block from the left atrium to the pulmonary vein during radiofrequency ablation

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Paroxysmal atrial fibrillation is often initiated by foci in the pulmonary veins (PVs); the junction between the PVs and the left atrium (LA) has become the target of radiofrequency (RF) ablation performed to isolate the PVs.

Ectopic atrial beats originating from the PVs propagate to the LA with a characteristically long conduction time, often with a conduction delay or block within the PV or at the PV-LA junction. However, details about the conduction properties within the PVs and across the PV-LA junction are still scanty.

We report a unique case of LA-PV decremental conduction caused by RF applications. New insights into the electroanatomical characteristics of the PV-LA junction are provided. The present report demonstrates, for the first time in humans, that decremental conduction may be related to the progressive damage of the conducting myocardial bundle due to RF energy applications.

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Introduction

Most cases of atrial fibrillation (AF) are triggered by repetitive ectopic beats or rapid tachycardia originating in the pulmonary veins (PVs) and conducted to the left atrium (LA)[1-4].

Catheter ablation of triggers originating from the PVs may successfully terminate paroxysmal AF[1,5]. Since ablation within the PVs may result in stenosis, the junctions between the PVs and the LA have become the target of radiofrequency (RF) ablation performed to achieve electrical isolation of the PVs[6,7].

Ectopic beats, originating from the PVs, may be propagated to the LA with a conduction delay or block within the PV or at the PV-LA junction[1,2] due to the complex arrangement of the myocardial fibers in the PV and/or in the PV-LA junction[8-10].

Hocini et al.[10] demonstrated zones of activation delay in canine PVs correlated with abrupt changes in fascicle orientation. However, the detailed conduction properties within the PVs and across the PV-LA junction are still subject of debate.

We report a unique case of LA-PV decremental conduction caused by RF energy applications that provides new insights into the electroanatomical characteristics of the PV-LA junction.

Case report

A 45-year-old woman with frequent episodes of drug-refractory, paroxysmal AF was referred to our Institution for electrical disconnection of the PVs.

The antiarrhythmic drug was discontinued for 5 half-lives before the study.

Having obtained the patient's informed written consent, a basic electrophysiological study was performed. The surface 12-lead ECG and intracardiac electrograms were acquired using a computerized digital system (Pruka Engineering, Houston, TX, USA). Three multipolar electrode catheters were placed in the antero-lateral right atrium, the His bundle area and the coronary sinus. A double transseptal puncture was performed, placing two sheaths within the LA. An intravenous bolus of heparin (70 U/kg) was then administered. Repeated dosages were given to maintain the activated clotting time in the range of 300 s.

The ostium of each PV was visualized at angiography.

An irrigated tip ablation catheter (Thermocool, Biosense Webster, Johnson & Johnson, Diamond Bar, CA, USA) and a decapolar circular catheter were passed through the transseptal sheaths (Lasso, Biosense Webster, Johnson & Johnson) and initially positioned in the left superior PV.

PV isolation was achieved during coronary sinus pacing by delivering RF energy at the ostial sites that exhibited the earliest PV activation[11].

RF energy was delivered at a maximum power of 30 W for 30 s. Saline (0.9%) solution was infused through the irrigated tip ablation catheter at a rate of 17 ml/min during RF application. A flow rate of 3 ml/min was used between applications.

The elimination of all ostial PV potentials and complete entrance and exit block were required for total electrical isolation. The baseline activation pattern and the changes caused by the first RF application were consistent with a single-input, wide connection located at the bottom of the ostium (Fig. 1). Unexpectedly, 2 RF applications, at a site close to the previous one, changed the conduction properties of the connection resulting in periodic conduction with a Wenckebach sequence 3:2 followed by stable 2:1 entrance block (Fig. 2A and 2B). A further RF application in the inferior segment of the ostium caused complete electrical isolation of the vein with the disappearance of all sequentially activated PV potentials (Fig. 2C).

Discussion

A better knowledge of the anatomical and electrophysiological characteristics of the PV muscle fibers and fascicle connecting the PVs to the atrial myocardium may help to understand the role of the PVs in initiating and maintaining AF[1].

The myocardial sleeves are more extensive around the superior than around the inferior PVs, as suggested by the distribution of the rapid firing foci responsible for the spontaneous initiation of AF[3]. The myocardial sleeves are thicker at the veno-atrial junctions and become circumferential or spirally oriented, overlapping the media of the venous wall when traced distally. Also, the presence of fibrotic and adipose areas together with gaps in the sleeves themselves may modify the arrangement and, consequently, the electrophysiological properties of myocytes. Indeed, myocardial fibrosis may cause heterogeneity in the conduction properties and refractory periods among the fascicles within the myocardial sleeves resulting in reentry and slow conduction[12].

It has been shown that the muscle fascicles, that generate PV potentials, display decremental conduction during premature stimulation. Decremental conduction also occurs in the LA, as shown by a progressive increase in the latency of the stimulus of the atrial electrograms recorded in the PVs during premature stimulation. However, the magnitude of slowing during premature stimulation was greater in the myocardial sleeves than in the LA, resulting in an increased separation of the atrial and PV potentials recorded in the PVs[13].

This finding is in agreement with the concept, suggested by Ho et al.[14], that a differential thickness of the muscle sleeves could also account for the variable characteristics of propagation across the PV-LA junction.

Our case may contribute to the understanding of the properties of veno-atrial conduction and demon-

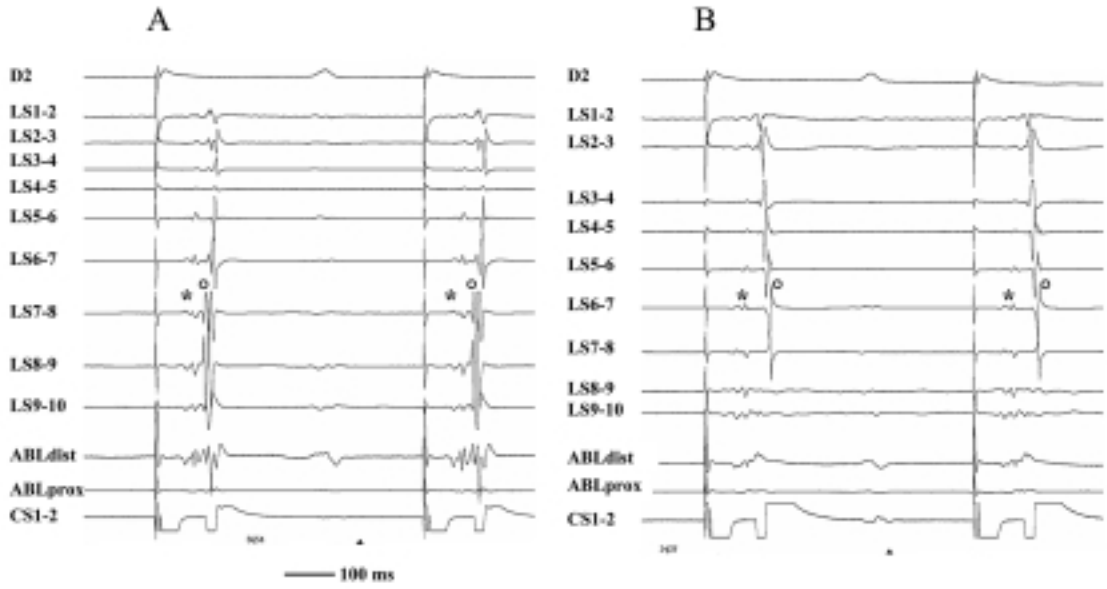

Figure 1. *Activation sequence of the left superior pulmonary vein potentials during distal coronary sinus pacing (CS) (cycle length 600 ms) at baseline* (A) *and after the first radiofrequency application* (B). From the top: *ECG lead II, bipolar electrograms recorded using the Lasso catheter (LS), the ablation catheter (ABL distal and proximal) and the CS catheter (distal).* A: *the earliest pulmonary vein activation was recorded at the bottom of the pulmonary vein ostium (LS8-9, LS9-10 and ABLdist in the same area), the local conduction time between the left atrium (*) and the pulmonary vein (**o**) was 46 ms.* B: *radiofrequency application between the LS8-9 and 9-10 eliminated the pulmonary vein potentials at these sites and changed the activation sequence such that the earliest pulmonary vein potential was recorded by LS1-2 (local conduction time 53 ms), located close to LS9-10 in the inferior segment of the pulmonary vein ostium.*

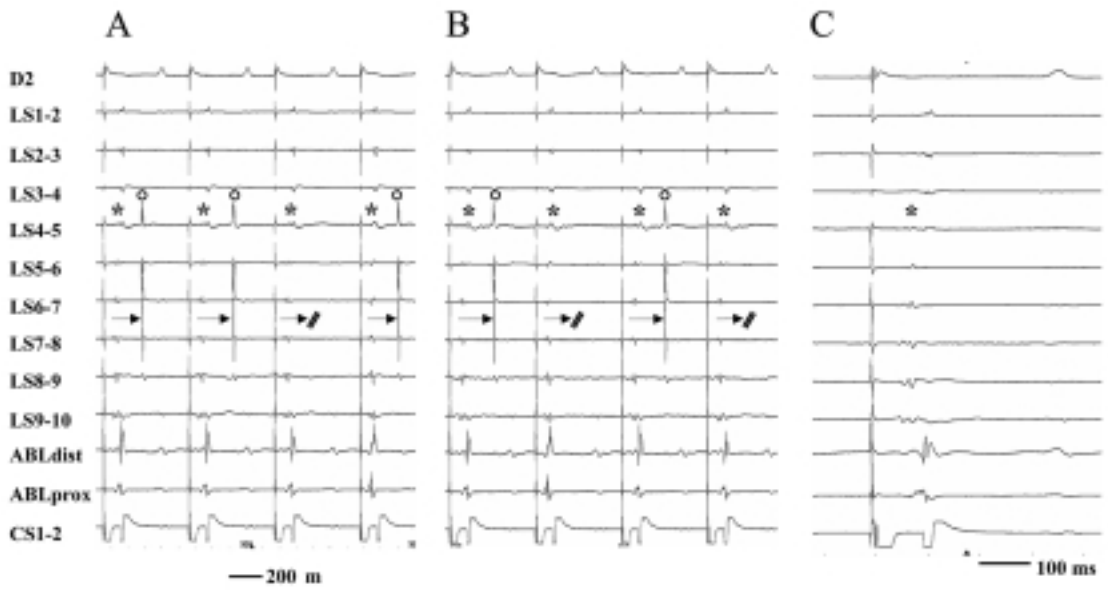

Figure 2. *Tracing format and symbols as in figure 1. Two radiofrequency applications at sites corresponding to LS1-2 and LS2-3 caused the elimination of the local pulmonary vein potentials and marked prolongation of the conduction time from the left atrium to the pulmonary vein, resulting in regular entrance block with a Wenckebach sequence 3:2* (broken arrow) (A), *followed by a stable 2:1 block* (B). *Note the low-amplitude, continuous activity between the left atrium and pulmonary vein potential recorded by LS4-5 electrodes suggesting that this was an area of slow conduction. A further radiofrequency application in this area eliminated all sequentially activated pulmonary vein potentials* (C).

strates, for the first time in humans, that the occurrence of periodic conduction may be related to progressive damage of the conducting myocardial bundle due to RF energy applications. Furthermore, it allows speculation concerning the possible mechanisms of periodic conduction due to progressive myocardial damage.

The acute myocardial lesion caused by RF energy shows coagulation necrosis, contraction band necrosis, interstitial hemorrhage and edema with more extensive damage found in the central area of the lesion and less damage in the marginal areas[15]. Specifically, it is likely that interstitial hemorrhage and edema may be the histopathological changes mainly responsible for the impairment of conduction across the connecting fascicle during RF applications.

Another attractive hypothesis is that the histopathological changes produced by RF energy actually decrease the width of the conducting fascicle resulting in a shift from 1:1 to periodic conduction. An incisional atrial reentrant tachycardia canine model demonstrated that the width of the isthmus determines the conduction properties through the isthmus itself: indeed, decremental conduction was observed only for a critical width of viable atrial myocardium[16]. These findings apparently support the hypothesis of a relationship between the dimensions of the viable connecting bundle and the conduction characteristics.

Moreover, the study suggests that whenever PV potentials appear dissociated from atrial potentials after RF ablation, the persistence of conduction with decremental properties should be ruled out to avoid late conduction recovery and the recurrence of AF.

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