

Inadvertent atrial dissociation following catheter ablation: A demonstration of cardiac anisotropy and functional block



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Introduction

Atrial dissociation (AD) is defined as an occurrence in which the right and left atria exhibit two distinct rhythms. It has been described in a few individual case reports and was first reviewed by Deitz and colleagues¹ in 1957. Recently in the era of aggressive atrial fibrillation (AF) management, multiple ablation procedures have been commonly used for treating persistent AF and postablation macroreentrant atrial tachycardias.

To really understand this phenomenon, one must first recognize the normal physiology behind interatrial conduction and the biatrial input to the atrioventricular (AV) node. In the literature, 3 distinct physiological pathways called internodal tracts have been described to provide conduction from the sinoatrial (SA) to AV node. A posterior pathway extends from the inferior SA node, past the crista terminalis and Eustachian ridge. A middle internodal tract stems from the superior SA node and travels posterior to the superior vena cava (SVC) and subsequently down the intraatrial septum. The third and most complex internodal tract courses anterior to the SVC where it bifurcates; 1 branch descends along the interatrial septum, while the other travels subepicardially across the interatrial groove, subsequently extending to the rightward and leftward branches. The rightward branch extends to the AV node. The leftward branch, known as Bachmann's bundle, crosses the interatrial groove; that is the primary interatrial conduction pathway. Two other physiological pathways have been described for interatrial conduction: 1 is located across the interatrial septum near the

fossa ovalis while the other lies near the proximal coronary sinus (CS).^{2,3}

Atrial myocardial input to the AV node occurs via 2 distinct cellular types. Transitional cells, having characteristics of both the atrial myocytes and the compact AV node, extend from the fossa ovalis to the compact AV node after passing the tendon of Todaro. This cellular group is localized to the left atrial septum. The second group of cells forms the inferior nodal extension, which lies superior to the tricuspid annulus and merges with the compact AV node. The transitional cells and inferior nodal extension form the fast and slow pathways of the AV node, respectively.^{4,5}

AD has historically been associated with conditions such as digitalis toxicity, congestive heart failure,¹ and corrective surgery for transposition of the great vessels.⁶ More recently AD has occurred following cardiac ablation procedures, in which there was an interruption noted across sites of interatrial electrical connection.⁷⁻⁹ We present a case of AD that occurred inadvertently during a radiofrequency ablation procedure for symptomatic atrial flutter (AFL).

Case report

A 70-year-old man with a history of atrial septal defect (ASD), recurrent AFL, and AF presented for a repeat ablation procedure. Many years prior to the presentation, the patient had undergone an ASD patch repair. Five years prior to presentation, he underwent AF ablation at an outside hospital. Three years later he experienced recurrent AFL and AF and presented for a repeat ablation procedure. Left atrial entry was achieved via 2 transseptal punctures utilizing a radiofrequency needle because of thick septum. Left atrial voltage mapping was performed, and large low-voltage areas were observed, consistent with endocardial scar. Pulmonary vein isolation (PVI) was performed utilizing a 4 mm irrigated-tip CARTO RMT ablation catheter (Biosense Webster, Diamond Bar, CA) navigated by a Stereotaxis magnetic navigation system (Stereotaxis, St. Louis, MO). After PVI he was inducible for upper loop reentry AFL and therefore linear ablation lesions were performed extending from the

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KEY TEACHING POINTS

- This case report illustrates the various parts of the atrial conduction system and how it is interconnected with each other.
- This case report carefully illustrates the various pathways and how electricity flows from right to left atrium. Disruption of these pathways can dissociate 2 atria.
- This case demonstrates electrical disconnection of both the atria and the principles behind it.
- This care report details the electrophysiological fundamentals of tissue anisotropy and functional and physiological blocks.
- Effects of heart surgery and multiple ablation procedures altering atrial conduction behavior are displayed.

SVC to the upper portion of the ASD patch. The patient remained in sinus rhythm for approximately 2 years; however, he had recurrence of AFL episodes.

The presenting electrocardiogram (ECG) showed AFL with variable ventricular rate of 84 beats/min. Intracardiac electrograms showed AFL with right-to-left CS activation suggestive of typical macroreentry right-sided AFL. Electroanatomic contact mapping was attained with the Biosense mapping system in a similar fashion as described above.

Right atrial contact activation map showed a possible macroreentry circuit. Subsequent entrainment mapping showed concealed entrainment from the proximal CS with the same CS intracardiac activation pattern. However, the postpacing interval was longer from the distal CS with manifest intracardiac fusion. Therefore, right atrial macroreentrant AFL was suspected and the integrity of the previously ablated right atrial isthmus line was checked, and that ablation line seemed to have a gap during activation mapping. A 20-pole Lasso catheter was placed on the anterolateral wall of the right atrium (RA). Entrainment from lateral RA also had longer postpacing interval suspicious for left-sided macroreentry. However, the decision was made to ablate the conduction gap in the isthmus line first before going to the left atrium (LA). The 4-mm-tip CARTO RMT ablation catheter was placed in the cavotricuspid isthmus region. Ablation lesions were performed along the medial aspect of the established right atrial isthmus line that was thought to be a conducting area. Postablation, intracardiac electrograms showed sole conversion of the RA to sinus rhythm. The LA continued in AFL with the same activation pattern, uncovering a previously suspected left atrial macroreentry circuit with passive activation of the RA (Figure 1). The prior upper loop reentry flutter line (which might have damaged Bachmann's bundle) and ASD patch repair (damaged septal conduction with prior surgery), along with a new complete medial isthmus block, may have completely disconnected both atria. Electroanatomic mapping showed the macroreentry circuit involving the right inferior and superior pulmonary veins passing through a roof scar near the right superior vein. Linear ablation was then carried out along the roof of the LA from the right superior pulmonary vein to the left superior pulmonary vein

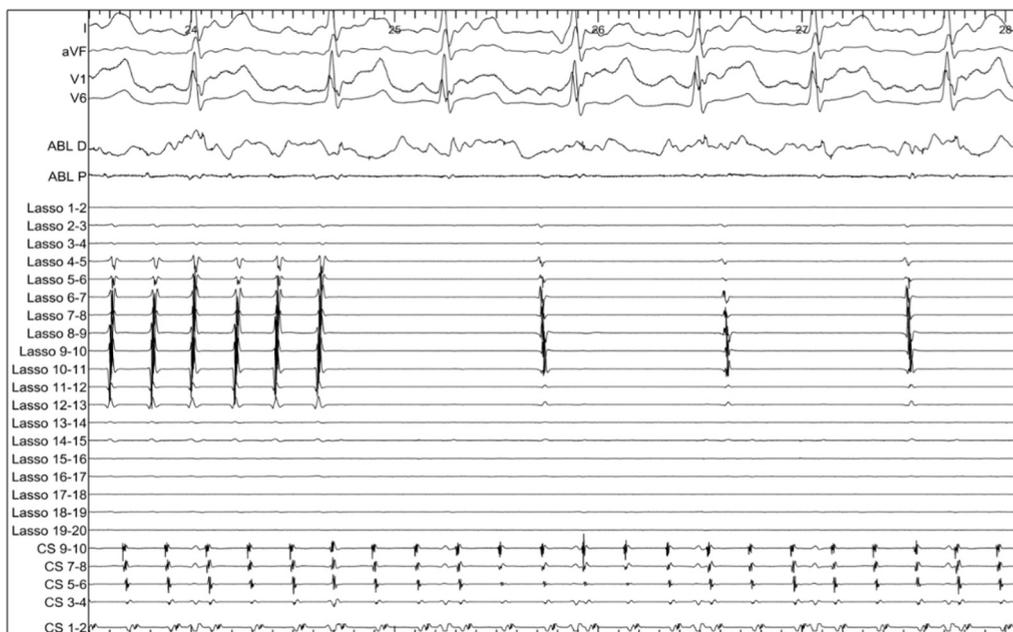


Figure 1 Intracardiac electrograms demonstrating atrial dissociation after right atrial isthmus ablation. Lasso catheter shows sinus rhythm of right atrium while coronary sinus (CS) leads show a persistent flutter circuit. Surface electrocardiogram leads reveal an irregular rhythm consistent with preferential conduction of left atrium to atrioventricular node. ABL = ablation catheter.

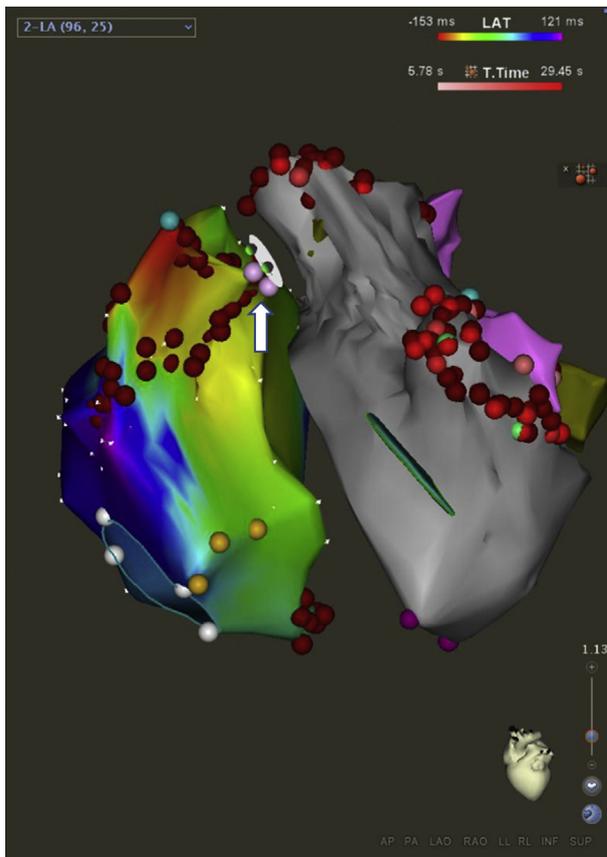


Figure 2 CARTO map (Biosense Webster, Diamond Bar, CA) of right atrium and left atrium. *Red dots* near superior vena cava represent linear ablation points for upper loop reentry flutter circuit from a prior ablation procedure. Ablation points disrupt Bachmann's bundle, which is represented by *white arrow*. LAT = local activation time.

vein, which terminated the flutter. A mitral line was also performed from the mitral annulus to the left inferior pulmonary vein to eliminate the chance of a mitral flutter circuit. The arrhythmia was not reproducible after administration of isoproterenol and atrial stimulation. After termination of the flutter, the atria were noted to be at the same rate (Supplemental Figure 1).

Discussion

To our knowledge, there are few reported cases of AD resulting from an ablation procedure. Similar to previous cases, left atrial isolation occurred once all sites of interatrial connection were interrupted. In our case, the interatrial septal connection near the fossa ovalis was disrupted by prior surgical patch repair of the ASD. Bachmann's bundle was likely affected from the prior linear ablation extending from the SVC to the ASD patch (Figure 2). During the current procedure, conduction through the CS ostium was affected during cavotricuspid isthmus ablation (Figure 3).

In contrast to prior reports, AD was only transiently apparent during the procedure. To best understand this phenomenon, one must consider each step of the ablation procedure and recognize its respective result.

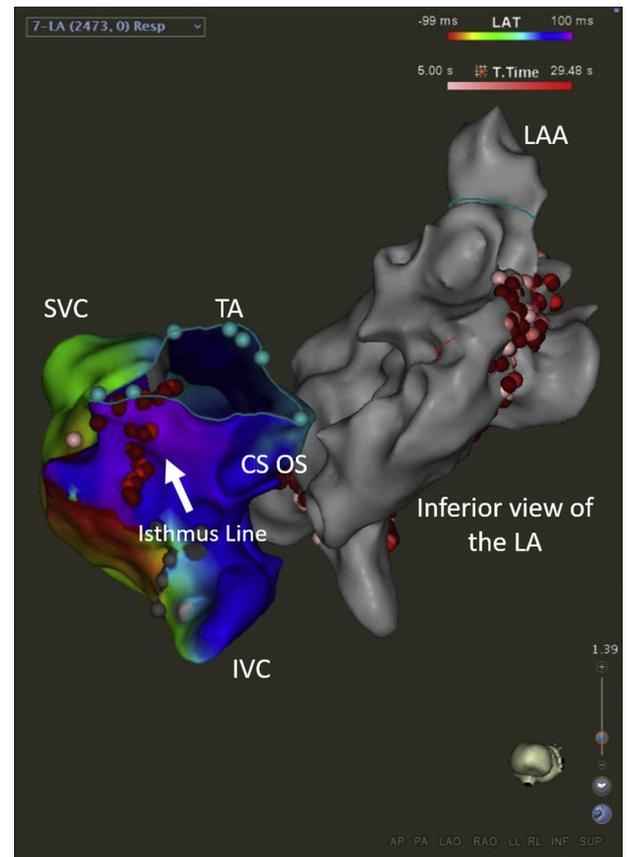


Figure 3 CARTO map (Biosense Webster, Diamond Bar, CA) of right atrium and left atrium. *Red dots* posterior to tricuspid annulus represent right atrial isthmus ablation. Ablation points transect interatrial conduction via coronary sinus ostium; physiological pathway is represented by *white arrow*. CS OS = coronary sinus ostium; IVC = inferior vena cava; LA = left atrium; LAA = left atrial appendage; LAT = local activation time; SVC = superior vena cava; TA = tricuspid annulus.

The few ablation points within the cavotricuspid isthmus caused interruption of only surviving interatrial conduction in this patient (as the septal pathway was blocked by the ASD patch and Bachmann's bundle was blocked by prior upper loop reentry AFL line) and allowed the RA to exhibit sinus rhythm. Such an occurrence exhibited complete electrical dissociation of the 2 atria. However, following ablation of the left-sided flutter circuit, the LA exhibited sinus rhythm; there was no "standstill" state that would be expected with complete left atrial isolation. Therefore, discordance of right and left atria cannot be attributed to a fixed anatomical block, but rather to a possible functional block.⁴ In addition, the concept of blocked conduction during AFL from left to RA and resolution during the sinus rhythm can be explained by tissue anisotropy. Cardiac anisotropy is in part dependent on myocardial fiber orientation and is expressed as a ratio of longitudinal conduction velocity to transverse conduction velocity.¹⁰ In comparison to the SA and AV node, cardiac anisotropy is relatively increased in the interatrial septum, crista terminalis, and trabeculae.⁴

During biatrial dissociation, the LA was in AFL and RA was in sinus rhythm. However, surprisingly, the ventricular rhythm did not correspond to the right atrial sinus rhythm;

instead, the ventricular electrograms were irregular, consistent with conduction of LA to AV node. This is a noteworthy finding, as the 3 internodal pathways all are in the RA. The occurrence of this preferential conduction demonstrated that in our patient the AV node had a distinct input from the LA. Left atrial input to AV node has been previously described in histology, where transitional cells were found in the left atrial septum.^{4,5} Left atrial input has also been described in an electrophysiology study; where pacing at identical cycle lengths, left atrial stimulation demonstrated a shorter atrial–His interval compared to right atrial stimulation.¹¹ Preferential conduction is affected by cardiac anisotropy; the defining ratio has been found to be rate-dependent, with higher rates resulting in a lower transverse conduction velocity in relation to longitudinal conduction velocity.¹⁰ Thus with disrupted interatrial communication, preferential left atrial conduction occurred secondary to the higher-frequency flutter circuit engaging the transitional cells longitudinally compared to the RA.

This case clearly illustrates the anatomy and physiology of the cardiac conduction system, as well as provides a greater understanding of preferential conduction, tissue anisotropy, and conduction velocity explaining AD. One must appreciate the limitations of multiple ablation procedures in our patient population. For future cases of AD, the cardiac anisotropy and functional block should be considered and long-term consequences of this phenomenon should be studied in a systematic fashion.

Appendix Supplementary data

Supplementary data associated with this article can be found in the online version at <https://doi.org/10.1016/j.hrcr.2018.12.010>.

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