

Iatrogenic macroreentry arising after transseptal puncture: A case series



Robert Douglas Paisley, MD,^{*} Michael Charles Tan, MD,^{*} Mehdi Razavi, MD,[†] Mohammad Saeed, MD[‡]

From the ^{*}Department of Cardiac Electrophysiology, Texas Heart Institute, Houston, Texas, [†]Department of Cardiology, Texas Heart Institute, Houston, Texas, and [‡]Electrophysiology Clinical Research and Innovations, Texas Heart Institute, Houston, Texas.

Introduction

Macroreentry can arise when the anatomic substrate contains both an area of unidirectional block and an area of slow conduction that allow for the formation of a reciprocating electrical circuit. The conditions for macroreentry in the atria may develop in patients with congenital malformations, such as atrial septal defects,¹ iatrogenic etiologies related to surgical incision of the atria,^{2,3} or penetrating myocardial trauma.⁴ Idiopathic septal flutters involving both the left⁵ and right⁶ atria have also been described. In these patients, conduction slowing at the limbus of the fossa ovalis was suspected to be a substrate for the initiation of macroreentry. This zone of abnormal conduction is thought to be related to the bilaminar structure of the septum primum, in which a thin layer of muscle is enveloped between the endocardial surfaces of each atrium.

Transseptal puncture is a procedure commonly used for left atrial and ventricular access during ablation procedures. Routinely performed procedures requiring transseptal puncture for left atrial access include atrial fibrillation ablation, tachyarrhythmia ablation in the left atrium or left ventricle, left atrial appendage occlusion, percutaneous mechanical circulatory support, and transcatheter mitral valve repair. During transseptal puncture, sheaths are used to maintain access to the left atrium across the interatrial septum after puncture with a curved needle from the right atrium to the left atrium. These sheaths range in size from 8F (eg, for atrial fibrillation ablation) to 22F (eg, for transcatheter mitral valve repair). Scar formation at the site of transseptal access may lead to conduction block and may further predispose to macroreentry.

KEYWORDS Atypical atrial flutter; Transseptal access; Macroreentry; Septal flutter; Ablation
(Heart Rhythm Case Reports 2022;8:270–274)

Funding Support: The authors have no funding sources to report. Disclosures: The authors have no conflicts of interest to report. **Address reprint requests and correspondence:** Dr Mohammad Saeed, Electrophysiology Clinical Research and Innovations, Texas Heart Institute, 6770 Bertner Ave, Houston, TX 77030. E-mail address: mohammadsaeed@gmail.com.

KEY TEACHING POINTS

- Electrophysiologists should be cognizant of the potential for macroreentrant atrial flutter to originate from the interatrial septum after mechanical trauma such as repeated transseptal access.
- An increase in the global volume of invasive procedures requiring transseptal access, such as left atrial endocardial ablation, left atrial appendage occluder device placement, transcatheter mitral valve repair, and mechanical circulatory support with TandemHeart, as well as the burden of atrial myopathy may lead to an increased prevalence and recognition of septal flutter.
- Ablation for septal flutter can be effectively performed by anchoring between the right upper pulmonary vein and the septal point of block on the left atrium or between the inferior vena cava and superior vena cava on the right atrial aspect.

Here, we present a series of 3 cases of atypical atrial flutter involving the septum in patients with a history of multiple prior transseptal punctures related to previous ablation procedures.

Case report

Case 1

A 50-year-old man presented for the evaluation of recurrent episodes of palpitations, most recently occurring 1 week before evaluation. The patient reported a heart rate of up to 160 beats per minute and reported that his palpitations lasted 3 days before spontaneously terminating. His medical history was notable for previous hypertension, coronary artery disease

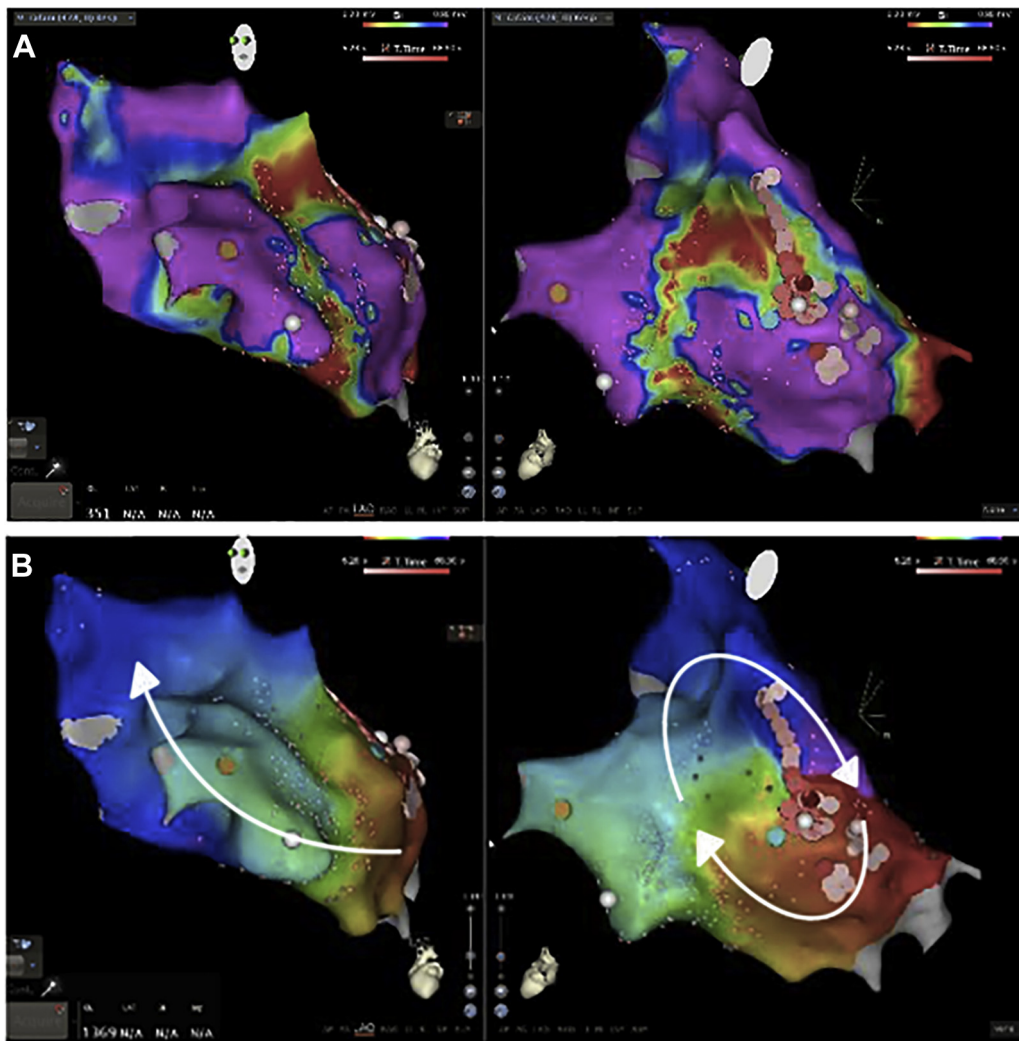


Figure 1 Mapping of the right atrium (case 1) revealing a non-isthmus-dependent macroreentrant circuit at the right atrial aspect of the septum. **A:** Voltage map of the right atrium (case 1) depicting ablation points drawn across a region of low voltage identified at the posterior right atrial wall. Ablation points connect the superior vena cava and inferior vena cava across the posterior right atrial wall. Regions colored red denote low voltage (<0.05 mV). Regions colored purple indicate normal voltage. **B:** Local activation time maps of the right atrium depicting ablation points drawn across the posterior right atrial wall connecting the superior vena cava and the inferior vena cava. Arrows depict the electrical activation sequence. In **A** and **B**, the ablation points are represented by circles in shades of gray, pink, and red. Red circles indicate a higher level of ablation time and energy, and gray-shaded circles indicate a lower level of ablation time and energy.

with prior percutaneous coronary intervention, type 2 diabetes mellitus, obstructive sleep apnea, paroxysmal atrial fibrillation, and atrial flutter. He had previously undergone pulmonary vein isolation (PVI) with radiofrequency (RF) ablation twice: once 4 years before the current presentation, and again 2 years after the initial ablation. One of the patient's previous ablation procedures was notable for the use of a double transseptal puncture approach. He also had a history of recurrent atypical atrial flutter and was undergoing medical treatment with sotalol and rivaroxaban. Despite antiarrhythmic drug therapy, he required direct current cardioversion on 2 previous occasions. The patient elected to pursue catheter ablation.

An electrophysiologic study was performed with a decapolar catheter in the coronary sinus and an intracardiac echocardiography catheter positioned in the right atrium. Atypical atrial flutter with a baseline tachycardia cycle length (TCL) of 360 ms and a conduction of 1:1 was found. Double transseptal puncture was performed, and the pulmonary veins

were interrogated and confirmed to be electrically silent with persistent entrance block from a previous RF ablation. Activation mapping of the entire left atrium failed to encompass the complete TCL. Right atrial activation mapping revealed a non-isthmus-dependent macroreentrant circuit at the right atrial aspect of the septum. By using RF ablation, a line was created from the superior vena cava along the posterior right atrial septum and then to the inferior vena cava, and the tachycardia was terminated during ablation (Figure 1A and 1B). Subsequent attempts to reinduce the arrhythmia were unsuccessful, and the procedure was terminated. Follow-up at 2 months after ablation showed no further evidence of recurrent arrhythmia.

Case 2

A 66-year-old man presented for the evaluation of recurrent symptomatic atrial fibrillation, despite adequate rate control and antiarrhythmic drug therapy with flecainide.

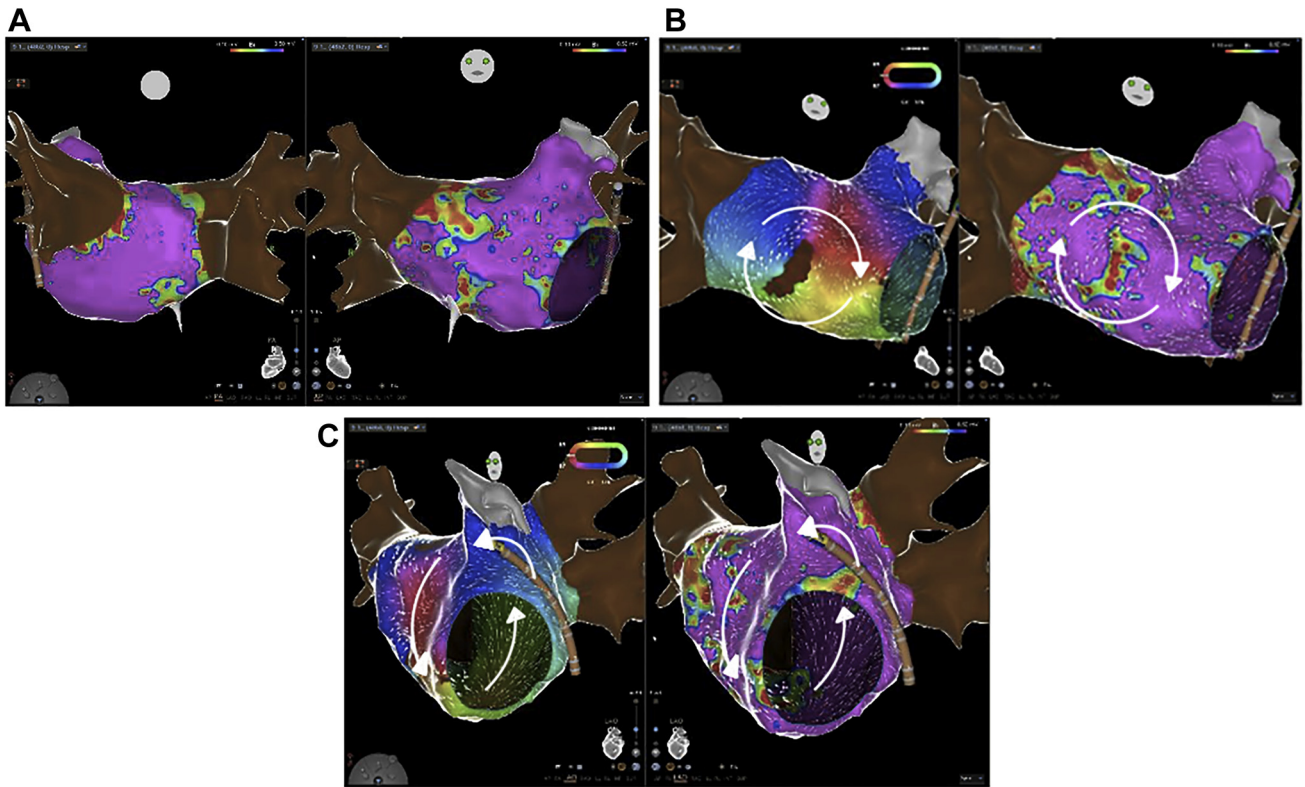


Figure 2 Mapping of the left atrium (case 3) revealing a macroreentrant circuit encircling the transeptal puncture site. **A:** Preablation voltage map demonstrating isolation of the pulmonary veins with regions of low voltage distributed over the superior aspect of the interatrial septum by the right upper pulmonary vein. An area of low voltage is also visible in the mid portion of the interatrial septum. Premature atrial contractions were identified in this region before ablation. Regions colored red denote low voltage (<0.05 mV). Regions colored purple indicate normal voltage. A decapolar catheter is positioned in the coronary sinus. **B:** Right anterior oblique caudal view of the coherent activation mapping of the macroreentrant circuit encircling the region of low voltage in the mid portion of the interatrial septum. Arrows depict the electrical activation sequence. A decapolar catheter is positioned in the coronary sinus. **C:** Left anterior oblique caudal view of the coherent activation mapping of the macroreentrant circuit encircling the region of low voltage in the mid portion of the interatrial septum. Arrows depict the electrical activation sequence. A decapolar catheter is positioned in the coronary sinus.

The patient's medical history was notable for hypertension, as well as paroxysmal atrial fibrillation diagnosed at the time of a cerebrovascular accident 8 years prior. A transesophageal echocardiogram at that time revealed a patent foramen ovale with right-to-left shunting that did not produce symptoms or evidence of hypoxia. The patient's clinical history was notable for recurrent atrial fibrillation requiring direct current cardioversion, despite increasing doses of flecainide. The patient was counseled on pharmacologic and catheter-based strategies for rhythm control, and the ultimate decision was made to pursue ablation.

The patient underwent a successful PVI with the use of a double transeptal puncture. At the time of PVI, the patient went into atypical flutter. Mapping during tachycardia revealed a left-sided macroreentrant circuit along the septum, with an initial TCL of 380 ms. Ablation resulted in the lengthening of the TCL to 500 ms, but it did not terminate the arrhythmia. The patient was electrically cardioverted, and rhythm control was attempted with sotalol. At the time of follow-up, the patient was found to have recurrence of the symptomatic atypical atrial flutter. Sotalol was discontinued, and a trial of amiodarone was initiated but was ultimately unsuccessful. After further

discussion with the patient, the decision was made to reattempt catheter ablation of the atypical atrial flutter.

The patient presented to the electrophysiology lab in atypical atrial flutter. Double transeptal puncture was performed, with mapping of the pulmonary veins demonstrating moderate electrical activity emanating from the left pulmonary veins and electrically silent right veins. RF ablation was performed to reisolate the left pulmonary veins. Low-voltage, fractionated potentials were identified near the right pulmonary veins. The right lower pulmonary vein was reisolated with RF ablation, and linear ablation was performed to connect the right lower pulmonary vein to the septum and then the mitral annulus through the sites of low-voltage, fractionated potentials. The flutter remained inducible, and further mapping in the right atrium revealed a critical isthmus along the septum. An ablation line was created from the coronary sinus ostium to the superior vena cava to avoid the His bundle. During ablation, TCL lengthened and tachycardia terminated. Subsequent attempts to reinduce the arrhythmia were unsuccessful. During a 4-year follow-up period, no recurrence of the atypical atrial flutter was detected, although the patient did experience recurrence of his paroxysmal atrial fibrillation.

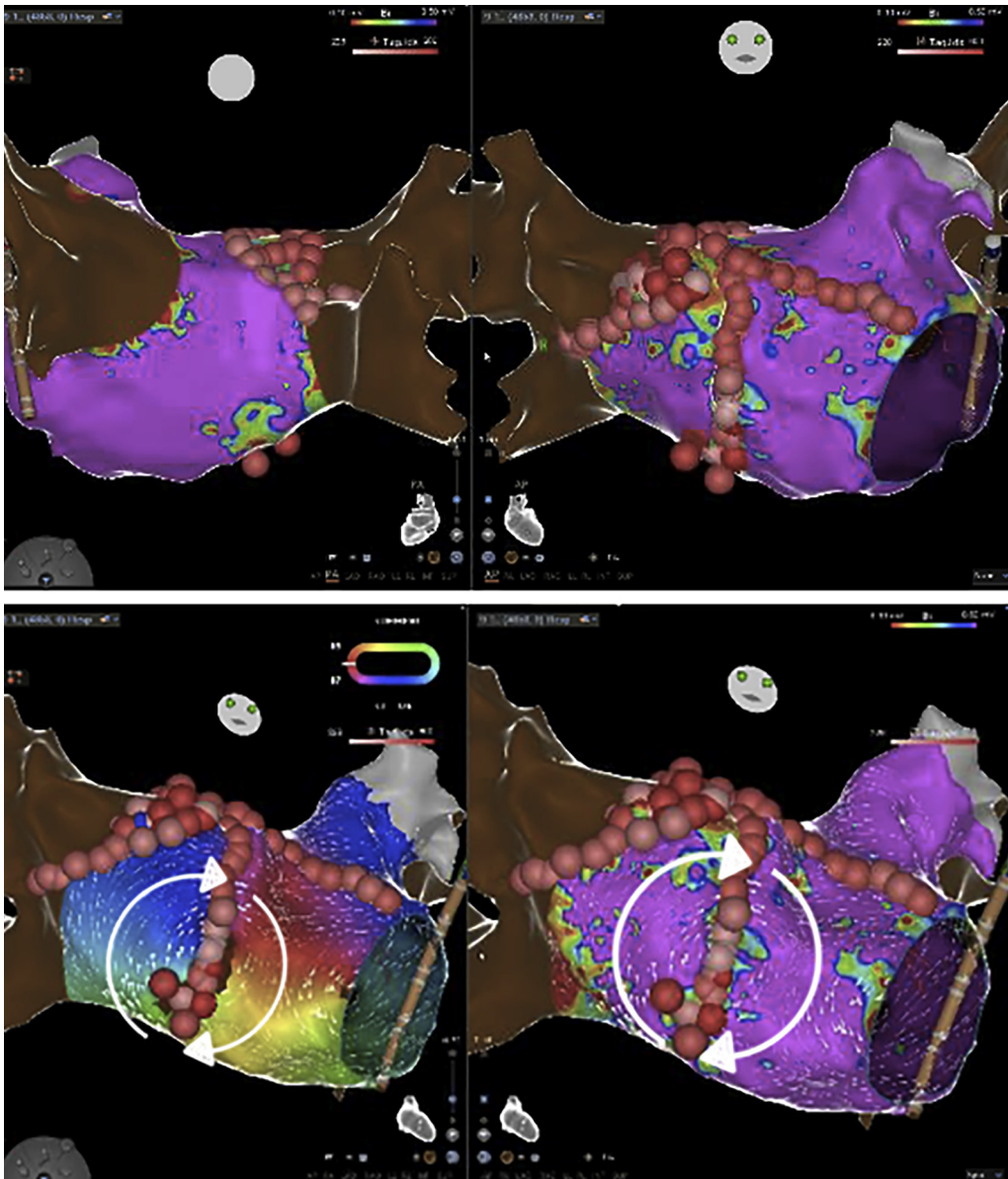


Figure 3 Ablation points extending from the region of low voltage at the right upper pulmonary vein (case 3). Lines are drawn to the mitral annulus and down across the region of low voltage in the interatrial septum, interrupting the macroreentrant circuit identified by Coherent mapping in CARTO. Top: a voltage map; bottom: the depiction of local activation time (left) and voltage (right). The ablation points are represented by circles in shades of gray, pink, and red. Red circles indicate a higher level of ablation time and energy, and gray-shaded circles indicate a lower level of ablation time and energy. Arrows depict the electrical activation sequence.

Case 3

A 42-year-old man presented for the evaluation of recurrent palpitations. Ambulatory cardiac monitor interrogation revealed an 18% burden of atrial fibrillation. The patient's medical history was notable for atrial fibrillation and multiple prior ablation procedures. A cryoablation PVI was performed 4 years earlier. One year later, redo cryoablation PVI and RF ablation along the cavotricuspid isthmus (CTI) line were performed. Two repeat ablation procedures were performed at the CTI for reinterrogation and reinforcement of the original ablation line. The patient was counseled on management options and decided to pursue repeat ablation for the atrial flutter.

In the electrophysiology lab, a single transseptal puncture was performed, and reconnection of the left upper and left lower pulmonary veins was found. Wide-area circumferential RF ablation was performed, and entrance and exit block was achieved in both left veins. The prior CTI line was confirmed to have bidirectional block.

At follow-up, the patient reported recurring symptoms of palpitations and heart rates of up to 130–140 beats per minute, which continued for several hours at a time. Repeated ambulatory cardiac monitoring showed atrial flutter with episodes of 1:1 conduction at a heart rate of 210 beats per minute. The decision was made to pursue repeat catheter ablation.

In the electrophysiology lab, isoproterenol infusion and adenosine administration revealed bigeminal premature atrial contractions emanating from the right upper pulmonary vein, which was reisolated with RF ablation. Atrial extrastimulus resulted in the induction of atypical atrial flutter with a cycle length of 220 ms. Mapping of the left atrium revealed a macroreentrant circuit encircling the transseptal puncture site and mitral annulus (Figure 2A–2C). An initial ablation line connecting the right upper pulmonary vein to the anterior aspect of the mitral annulus failed to terminate the tachycardia. A second line connecting the right upper pulmonary vein to the transseptal puncture site successfully terminated tachycardia (Figure 3). Subsequently, tachycardia was noninducible, despite isoproterenol infusion and atrial pacing maneuvers. At the time of follow-up, no further palpitations or arrhythmia was detected.

Discussion

In this case series, we describe macroreentry arising from the interatrial septum in patients who underwent multiple prior transseptal punctures. In each of these cases, electroanatomic mapping was used to identify the macroreentrant circuit and guide ablation to anchor points within the atrium. During these procedures, entrainment was intentionally not performed to avoid terminating tachycardia or inducing macroreentry in alternate circuits through diseased and scarred atrial tissue.⁷ Anchor points included the superior vena cava, inferior vena cava, coronary sinus ostium, right lower pulmonary vein, mitral annulus, and the site of transseptal puncture in the fossa ovalis. Previous descriptions^{1,5,6} of idiopathic septal flutter ablation have included the use of identical anchor points for ablation lines to terminate macroreentry. In each case, anchor points for ablation were selected to provide the shortest distance across the macroreentrant circuit between 2 nonconducting structures and to avoid the conduction system.

Atrial fibrillation is the most common sustained arrhythmia. As of 2017, an estimated 75,000 ablation procedures for atrial fibrillation were performed annually. Approximately 28% of these patients are expected to require repeat atrial fibrillation ablation within 3 years of the first ablation procedure.⁸ An increasing body of evidence has transformed standard-of-care therapy for the management of atrial fibrillation, particularly in patients who have symptomatic and hemodynamically significant atrial fibrillation, with an emphasis on the earlier consideration of catheter-based strategies. Despite improved techniques and ablation technologies, repeat ablation procedures are expected to be necessary for patients with recurrent tachyarrhythmia.

Previous studies have shown an increased propensity for macroreentrant atrial flutter to arise from incisional atrial scar tissue with a narrow isthmus between the incision and

nonconductive tissue, such as the cavoatrial junction,⁹ or between 2 surgical incisions.¹⁰ Repeat transseptal puncture may create such a narrow isthmus, with clusters of lesions created by transseptal access. An increased number of transseptal punctures and the larger size of transseptal access sheaths may predispose patients to increased scar burden and therefore a propensity to develop a substrate supporting macroreentry. To date, percutaneous transseptal access has not been investigated as a potential cause of iatrogenic macroreentrant tachycardia. We believe this merits further consideration, particularly in patients with enlarged, scarred atria serving as an arrhythmogenic substrate and in patients who may undergo a host of procedures requiring transseptal access, such as left atrial ablation, left atrial appendage closure, percutaneous left ventricular mechanical circulatory support, and transcatheter mitral valve intervention. Additionally, patients with septal flutters who do not undergo careful study of right atrial macroreentrant circuits may be assumed to have CTI-dependent flutter on the basis of coronary sinus catheter activation patterns. Inappropriate ablations directed at the CTI may result from failure to recognize a septal flutter related to prior transseptal access. As PVI becomes more commonly used for the management of atrial fibrillation, iatrogenic arrhythmia arising from transseptal access is an increasingly important entity to consider when formulating treatment plans for individuals with recurrent atrial fibrillation.

References

1. Mikhaylov E, Gureev S, Szili-Torok T, Lebedev D. Atypical atrial flutter in a patient with atrial septal defect without previous surgery: the role of septal defect as a part of the arrhythmia substrate. *Europace* 2009;11:1705–1708.
2. Adachi T, Yoshida K, Takeyasu N, et al. Left septal atrial tachycardia after open-heart surgery: relevance to surgical approach, anatomical and electrophysiological characteristics associated with catheter ablation, and procedural outcomes. *Circ Arrhythm Electrophysiol* 2015;8:59–67.
3. Wasmer K, Kobe J, Dechering DG, et al. Isthmus-dependent right atrial flutter as the leading cause of atrial tachycardias after surgical atrial septal defect repair. *Int J Cardiol* 2013;168:2447–2452.
4. Steven D, Rostock T, Lutomsy B, Willems S. Three-dimensional mapping of atypical right atrial flutter late after chest stabbing. *Pacing Clin Electrophysiol* 2008;31:382–385.
5. Marrouche NF, Natale A, Wazni OM, et al. Left septal atrial flutter: electrophysiology, anatomy, and results of ablation. *Circulation* 2004;109:2440–2447.
6. Wiczcerek M, Hoeltgen R. Right atrial tachycardias related to regions of low-voltage myocardium in patients without prior cardiac surgery: catheter ablation and follow-up results. *Europace* 2013;15:1642–1650.
7. Vollmann D, Stevenson WG, Luthje L, et al. Misleading long post-pacing interval after entrainment of typical atrial flutter from the cavotricuspid isthmus. *J Am Coll Cardiol* 2012;59:819–824.
8. Hosseini SM, Rozen G, Saleh A, et al. Catheter ablation for cardiac arrhythmias: utilization and in-hospital complications, 2000 to 2013. *JACC Clin Electrophysiol* 2017;3:1240–1248.
9. Yang G, Du X, Ni B, et al. Prevention of postsurgical atrial tachycardia with a modified right atrial free wall incision. *Heart Rhythm* 2015;12:1611–1618.
10. Ishii Y, Nitta T, Sakamoto S, Tanaka S, Asano G. Incisional atrial reentrant tachycardia: experimental study on the conduction property through the isthmus. *J Thorac Cardiovasc Surg* 2003;126:254–262.