Fibrillation in the Superior Vena Cava Mimicking Atrial Tachycardia

Daniel Steven, MD; Kurt C. Roberts-Thomson, BSSC; Jens Seiler, MD; Gregory F. Michaud, MD; Roy M. John, MD, PhD; William G. Stevenson, MD

A 49-year-old male was referred for catheter ablation for paroxysmal atrial fibrillation (PAF). PAF had recurred after each of 2 prior left atrial ablation procedures. He had a structurally normal heart with a left atrial diameter of 43 mm. The patient presented to the electrophysiology laboratory in AF with an average AF cycle length (CL) of 150 ms and reconnected pulmonary veins. After electric disconnection of all 4 veins using an antral circumferential approach, AF organized to an atrial tachycardia (AT), which was successfully converted into right atrial common type atrial flutter at a CL of approximately 430 ms and a P-wave morphology (inferior axis, positive/negative P wave in V1), suggesting an origin in the vicinity of the sinus node. Mapping in this region revealed a focal tachycardia located in the superior vena cava (SVC) at a CL of approximately 215 ms and predominantly 2:1 veno-atrial conduction. Radiofrequency (RF) ablation at the SVC-RA junction terminated this focal tachycardia and restored sinus rhythm in the right atrium. Subsequent mapping of the SVC during the course of the procedure using a circumferential mapping catheter, however, revealed a rapid tachycardia (CL 150 ms) with the right atrium (RA) remaining in sinus rhythm, indicating exit block from the SVC to the RA (Figures 1 and 2). RF ablation of the SVC focus first slowed the SVC tachycardia and subsequently terminated it. Surprisingly, despite the presence of complete exit block from SVC to RA during SVC tachycardia, immediately after termination conduction from RA to SVC was observed, as indicated by SVC potentials preceded by an atrial farfield signal recorded in the circumferential mapping catheter positioned in the SVC (Figure 3). Further mapping and ablation of the “entrance-connection” of the SVC resulted in complete isolation of the SVC, proving unidirectional conduction properties of the SVC (Figure 4). After ablation AF was not inducible despite isoproterenol infusion and rapid burst pacing.

Discussion

The SVC has been reported to play a role in arrhythmia initiation and maintenance in ∼6% to 12% of patients with PAF.1,2 Unidirectional conduction properties of myocardial sleeves in the SVC but not of RA connections to the SVC have previously been shown using a multipolar basket catheter.3 In the left atrium, potential unidirectional conduction from PV to atrium has been shown, based on PV pacing with conduction to the LA despite absent PV potentials during sinus rhythm, although far-field capture of the left atrium could not be completely excluded.4 In the present case it is possible that unidirectional block from SVC to RA was frequency-dependent. However, even slowing of the SVC tachycardia to a cycle length of 280 ms did not result in any SVC to RA conduction (Figure 3). It is also possible that block was a manifestation of concealed conduction of impulses into the SVC–RA junction.

It is interesting that 2 tachycardias with very different rates were observed within the SVC. The mechanism is uncertain, but multiple potential reentry paths or 2 simultaneously circulating wavefronts along 1 reentry path could theoretically cause these findings.

This case also demonstrates that a SVC tachycardia can be a source for apparent irregular atrial tachycardia that can potentially mimic sinus tachycardia.

Sources of Funding

Dr Steven is the recipient of a research grant from Biosense-Webster. Dr Seiler is the recipient of a research grant from St Jude Medical (Switzerland). Mr Roberts-Thomson is the recipient of an Overseas-Based Clinical Research Fellowship from the National Health and Medical Research Council (NHMRC) of Australia (NHMRC grant 489419).

Disclosures

Dr Michaud is a consultant for St Jude Medical and Biosense-Webster and has received speakers honoraria from St Jude Medical, Medtronic, and Boston Scientific. Dr Stevenson has received speakers honoraria from St Jude Medical, Biosense-Webster, Boston Scientific, and Medtronic.

From the Cardiovascular Division, Department of Medicine, Brigham and Women’s Hospital, Harvard Medical School, Boston, Mass.

Guest Editor for this article was Michael E. Cain, MD.

Correspondence to Daniel Steven, MD, Cardiovascular Division, Brigham and Women’s Hospital, Harvard Medical School, 75 Francis Street, Boston, MA 02115. E-mail dsteven@partners.org

(Circ Arrhythmia Electrophysiol. 2009;2:e4-e7.)
© 2009 American Heart Association, Inc.

Circ Arrhythmia Electrophysiol is available at http://circep.ahajournals.org

DOI: 10.1161/CIRCEP.108.833400

e4
References


Key Words: arrhythmia, clinical electrophysiology, drugs ■ ablation/ICD/surgery ■ electrophysiology
Figure 2. Fluoroscopic image demonstrating a left anterior oblique view with coronary sinus catheter, mapping catheter, and circumferential catheter positioned in the SVC.
Figure 3. Tracing demonstrating termination of the focal SVC tachycardia with increased CL without affecting the atrial CL (860 ms), indicating exit block, even after the SVC tachycardia had slowed to a cycle length of 280 ms (surface EKG leads I, II, III, V1, and V5). Immediately after termination of the SVC tachycardia, conduction from the atrium to SVC is present, indicating unidirectional conduction from the RA to the SVC. LS indicates circumferential mapping catheter. *SVC activation preceded by the RA far-field potential.

Figure 4. The tracing demonstrates conduction from the RA to the SVC during RF ablation of the connection. Conduction into the SVC delays (asterisk) then blocks. L1–L20 were recorded from the circular catheter in the SVC, indicating complete SVC isolation.
Fibrillation in the Superior Vena Cava Mimicking Atrial Tachycardia
Daniel Steven, Kurt C. Roberts-Thomson, Jens Seiler, Gregory F. Michaud, Roy M. John and William G. Stevenson

Circ Arrhythm Electrophysiol. 2009;2:e4-e7
doi: 10.1161/CIRCEP.108.833400

Circulation: Arrhythmia and Electrophysiology is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2009 American Heart Association, Inc. All rights reserved.
Print ISSN: 1941-3149. Online ISSN: 1941-3084

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circep.ahajournals.org/content/2/2/e4

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation: Arrhythmia and Electrophysiology can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation: Arrhythmia and Electrophysiology is online at:
http://circep.ahajournals.org//subscriptions/