Adenosine Responsive Narrow Complex Tachycardia: What is the Mechanism?

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ABSTRACT. This case is that of a 72-year-old man with a symptomatic narrow complex long RP tachycardia that was responsive to adenosine. Invasive recordings confirmed a likely atrial tachycardia, then mapping showed earliest atrial sites near the His bundle. However, many sites were equally early. Further mapping of the non-coronary aortic cusp revealed an even earlier site where ablation was immediately successful. This article discusses the responsiveness of aortic cusp tachycardias to adenosine, their intra-atrial mapping signature, and the anatomic factors that explain these features.

KEYWORDS. adenosine sensitive, supraventricular tachycardia, catheter ablation.

Case presentation

A 72-year-old man presented with frequent and very troubling palpitations that caused lightheadedness and necessitated visits to the emergency room. His palpitations were non-exertional and terminated with vagal maneuvers and, in the emergency room, with IV adenosine. His symptoms continued despite metoprolol 100 mg/day. His past medical history was otherwise unremarkable. Physical examination was within normal limits, and baseline biochemistry and blood counts were unremarkable, as were echocardiography and adenosine stress testing. His baseline electrocardiogram (ECG) showed sinus bradycardia and left anterior fascicular block without pre-excitation, but an ECG captured during symptoms revealed a long RP narrow complex tachycardia with cycle length of 360 ms and PR interval of 104 ms (Figure 1a). What is the differential diagnosis?

At electrophysiology study, baseline intervals in sinus rhythm were AH 90 ms and HV 42 ms. Ventricular pacing caused concentric retrograde atrial activation that decremented with extra stimuli without a sudden jump in VA time or atrial activation sequence. Atrial extra stimulus pacing provided no evidence for dual AV nodal physiology. Notably, however, atrial burst pacing at 400 ms reliably induced clinical tachycardia (CL 360 ms). Atrial activation was earlier in the His catheter than in the coronary sinus or high right atrium. VA linking was not present and A-A cycle length changes preceded V-V cycle length change. Ventricular overdrive pacing resulted in a VAAV response (Figure 1b), consistent with an atrial tachycardia (AT). The tachycardia could be reliably terminated with burst pacing but could not be entrained. What are likely locations for the tachycardia?

The wide area of early activated tissue near the His bundle position suggested passive breakthrough, such that mapping of the aortic root may be revealing. Mapping of the non-coronary cusp via femoral arterial access uncovered a site activating 45 ms before the P wave. Detailed activation mapping with EnSite NavX (St. Jude Medical, St. Paul, MN) confirmed earliest right atrial activation near the compact AV node, which preceded the P wave in ECG lead II by 20 ms (Figure 2). However, activation was similarly early for a wide contiguous area of atrium. This activation was earlier than any other right atrial or coronary sinus site. Based on these observations what would be the next procedural step?

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wave in ECG lead II (Figure 3). Radiofrequency ablation (55 W, 50°C) at this site under careful impedance and temperature monitoring terminated the tachycardia within 4 s (Figure 3b). After 1 min of radiofrequency energy delivery, the tachycardia was no longer inducible with isoproterenol or burst pacing. The patient remains asymptomatic 4 months after the procedure.

**Commentary**

AT accounts for approximately 5% of supraventricular tachycardia cases presenting for ablation,\(^1\) of which 25% are terminated by vagal maneuvers and 50–80% by adenosine.\(^2\) Termination with adenosine can help to distinguish macro or micro-reentrant atrial tachycardia from focal atrial
NCC Tachycardia

tachycardia due to abnormal automaticity or triggered activity as in our patient. Reliable initiation and termination by burst pacing without entrainment suggest triggered activity as the basis for clinical tachycardia. Focal ATs commonly arise from pulmonary veins, crista terminalis, and atrial appendages. Uncommonly, they arise from the epicardial aspect of the inter-atrial septum, in which case the ECG P wave is typically negative in V1 and negative-positive or negative in the inferior leads, and earliest apparent intra-atrial activation appears in the para-Hisian region mimicking a true para-Hisian focal tachycardia. Surface EKG P wave morphology can be influenced by lead placement, orientation of the heart in the chest cavity, chamber dimensions, inter-atrial conduction delay and endocardial break-through point of an epicardial focal source in the inter-atrial septum. The non coronary cusp provides easy access to the epicardial aspect of inter-atrial septum. These epicardial atrial tachycardias that are ablated from the non coronary cusp (NCC) have distinct endocardial activation pattern. This activation sequence can be understood by considering the anatomy of the common left ventricular (LV) ostium.

The aorta joins the LV ostium at an angle with its three semi-lunar cusps. The non-coronary cusp (NCC) is the most inferior and the left coronary cusp (LCC) is the most superior in position. The right coronary cusp (RCC) is the most anterior semi-lunar valve. The RCC and LCC are attached to the LV ostial myocardium. The base of the RCC is closely opposed to the LV ostial myocardium with muscle fibers running parallel to the cusp, while, conversely, the LCC is surrounded by fibrous tissue with sparse muscle fibers near the LV ostium. The NCC abuts the inter-atrial septum above the pyramidal space. At its nadir the NCC is in contact with the membranous interventricular septum near the penetrating bundle of His.

The NCC does not contact the LV ostium, unlike the LCC and RCC, which are common sources for idiopathic VT/PVC. Rather, abnormal automaticity or triggered activity from musculature in the epicardial inter-atrial septum accessible from the NCC presents as focal atrial tachycardia with a distinct activation pattern showing simultaneous depolarization in the para-Hisian right atrium and anteroseptal left atrium. There is diffuse endocardial activation the first 20 milliseconds, as seen in our patient. For these reasons and the real possibility of AV nodal damage if ablating in the para-Hisian region, the NCC coronary cusp and the anterior left atrial septum should be mapped if earliest atrial activation during tachycardia lies in the para-Hisian region. We believe that it may be preferable to map the aortic cups prior to LA mapping as studies have shown that LA endocardial activation time is not much earlier than the para-Hisian region. The coronary cusps are easily identified on fluoroscopy. The NCC is the most inferior and posterior cusp on fluoroscopy with a close relationship to the His bundle recording catheter, and additional imaging may be required to confirm a safe distance of the ablation catheter from the coronary arteries. NCC tachycardias are highly amenable to ablation, with success rates of 100% in the

Figure 2: Electroanatomic map in tachycardia created with NavX system shows diffuse early activation (relative to the P wave in electrocardiogram lead II) in the para-Hisian region. Time scale dynamic range is set at −20 ms to +20 ms.
literature on fewer than three RF applications. The maximum time to termination has been reported to be under 8 s, with few complications.7

References

Figure 3: (a) Ablation catheter position at successful site in left anterior oblique and right anterior oblique view. His bundle catheter had moved inferior to the optimum position at this moment. (b) Ablation terminates tachycardia within 4 s.