Hemodynamic response to supraventricular tachycardia in a patient with hypertrophic cardiomyopathy

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Introduction

Syncope in patients with hypertrophic cardiomyopathy (HCM) is a complex and challenging entity to investigate. Ventricular arrhythmias are frequent in this population and may result in loss of consciousness. However, supraventricular tachycardias (SVT) with fast ventricular response have been reported to precipitate hemodynamic instability.[1] The hemodynamic response to SVT is dependent not only on heart rate, loading conditions, and ventricular function, but also on the timing of atrial and ventricular systole. Variation in blood pressure response has been documented in normal subjects with atrioventricular nodal reentry tachycardia (AVNRT) and atrioventricular (AV) reentrant tachycardia.[2] In this instance, acute changes in blood pressure are thought to be related to the extent of the ventriculoatrial (VA) interval, with simultaneous AV activation during AVNRT causing more hemodynamic compromise than sequential AV activation in AV reentrant tachycardia.[3–5]

We report a clinical case of a patient with HCM presenting with syncope preceded by palpitations during a narrow-complex tachycardia, as well as electrophysiological findings and hemodynamic response during arrhythmia evaluation.

Case report

A 36-year-old previously healthy man was evaluated in the Emergency Department for syncope in the setting of palpitations. He described an episode of rapid regular heart rate associated with neck pounding and lightheadedness, followed by loss of consciousness. His medical and family history were otherwise unremarkable.

KEY TEACHING POINTS

- Supraventricular tachycardia (SVT) can precipitate symptomatic hemodynamic instability in patients with hypertrophic cardiomyopathy.
- The hemodynamic response to SVT is related to heart rate, loading conditions, and ventricular function, but importantly also to the timing of atrial and ventricular systole.
- Acute changes in blood pressure are related to the extent of the ventriculoatrial interval, with simultaneous atrioventricular activation leading to more hemodynamic compromise than sequential activation.

At his initial evaluation, he was in sinus rhythm and asymptomatic. His physical examination included a 2/6 systolic ejection murmur loudest at the base of the heart, without physical signs of cardiomegaly. His electrocardiogram was abnormal, with Q waves in the inferior leads, as well as 2 mm ST-segment elevation in II, III, aVF and T-wave inversion in I, aVL, V_2 (Figure 1A). A transthoracic echocardiogram showed normal regional and global systolic function with an ejection fraction of 69% and asymmetric left ventricular hypertrophy (septal wall thickness of 2.5 cm) without resting or inducible left ventricular outflow gradient, but highly suggestive of HCM. Cardiac magnetic resonance imaging confirmed the diagnosis, showing hypertrophy of the basal-mid septum with early and late patchy gadolinium enhancement in the anteroseptum and the presence of systolic anterior motion of the mitral valve. While the patient was hospitalized for monitoring, recurrent episodes of narrow complex tachycardia at around 170–180 beats per minute were documented and associated with presyncope. All episodes were reproducibly terminated with carotid sinus pressure or vagal maneuvers (Figure 1B).

KEYWORDS Atrioventricular nodal reentrant tachycardia; Blood pressure; Hemodynamic response; Hypertrophic cardiomyopathy; Supraventricular tachycardia

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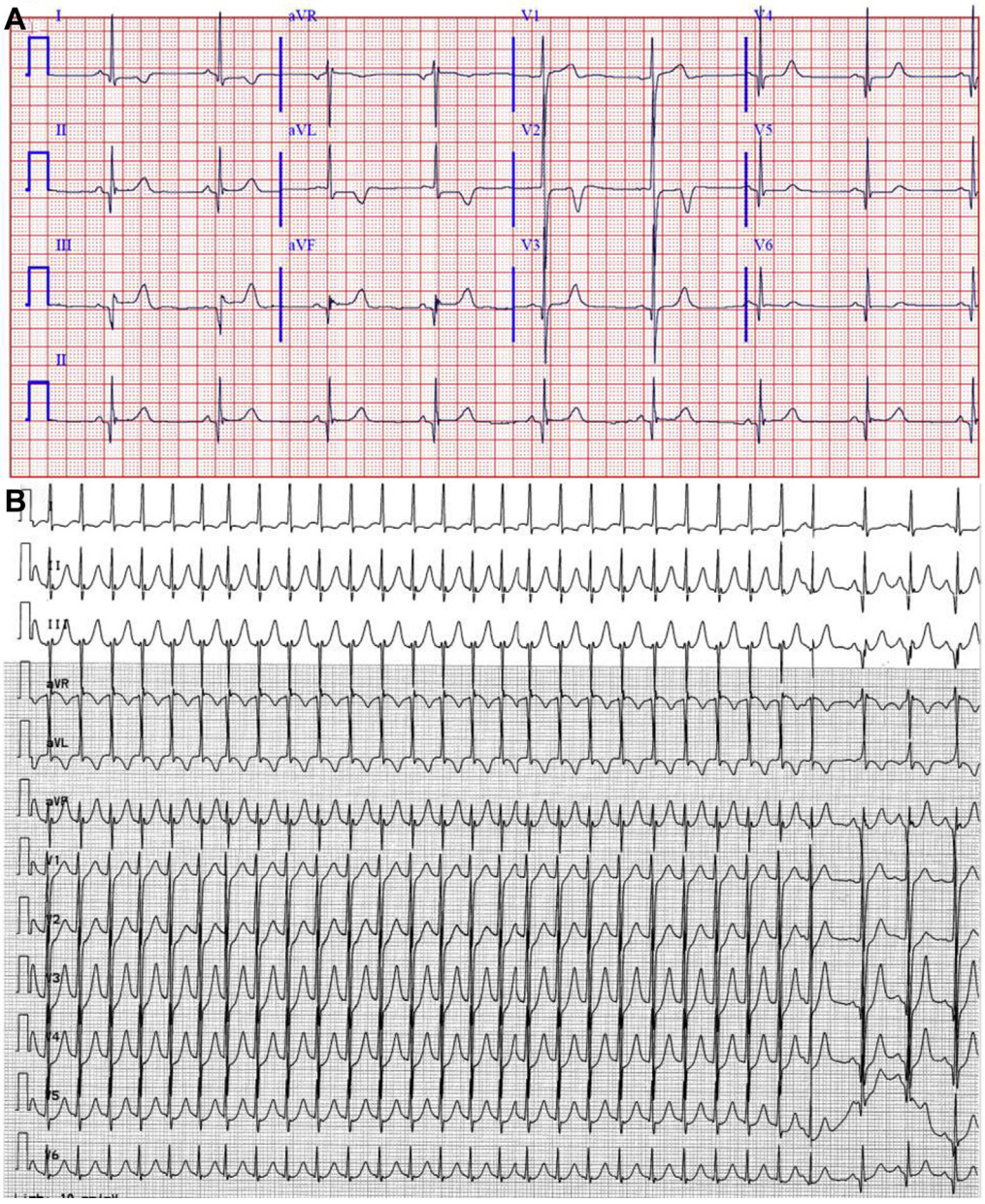

Figure 1 **A:** Baseline electrocardiogram showing sinus bradycardia at 56 beats per minute; Q waves in the inferior leads; 2 mm ST-segment elevation in II, III, aVF; and T-wave inversion in I, aVL, V_2. **B:** Narrow complex tachycardia with cycle length of 170 beats per minute and its termination.

Given these findings, an electrophysiology study was performed. An arterial line was placed for hemodynamic monitoring. Baseline A-A, AH, and HV intervals were 1040, 101, and 48 ms, respectively. Programmed stimulation from the proximal coronary sinus demonstrated dual AV nodal physiology. Two different types of SVT were reproducibly induced; pacing maneuvers were performed to evaluate arrhythmia mechanism, including His-refractory premature ventricular stimuli, ventricular overdrive pacing during SVT, and VAHV response to entrainment, all being consistent with #1 typical slow-fast AVNRT (Figure 2A) and #2 atypical fast-slow AVNRT (Figure 3A). Cycle length of SVT #1 was 380 ms with a VA interval of 18 ms, while the cycle length of SVT #2 was 320 ms with a VA interval of 170 ms. SVT #1 was not tolerated hemodynamically, causing lightheadedness and near syncope requiring termination by overdrive pacing. SVT #2 was better tolerated hemodynamically and was not symptomatic. Arterial tracings confirmed a different blood pressure response at initiation of each type of SVT. In typical slow-fast AVNRT, a significant systolic blood pressure drop from 130 mm Hg to 40 mm Hg was present and reproduced the patient's symptoms (Figure 2B). Conversely, atypical fast-slow AVNRT led to a milder drop in blood pressure (systolic of 100 mm Hg to 75 mm Hg), followed by gradual recovering to 85 mm Hg (Figure 3B). Radiofrequency ablation with a 4-mm-tip nonirrigated catheter was performed during sinus rhythm at the site of slow pathway potential recording. A single 60-second radiofrequency application at 40 watts and 60°C was delivered and resulted in an accelerated junctional rhythm without evidence of AV block. Postablation, none of the SVTs

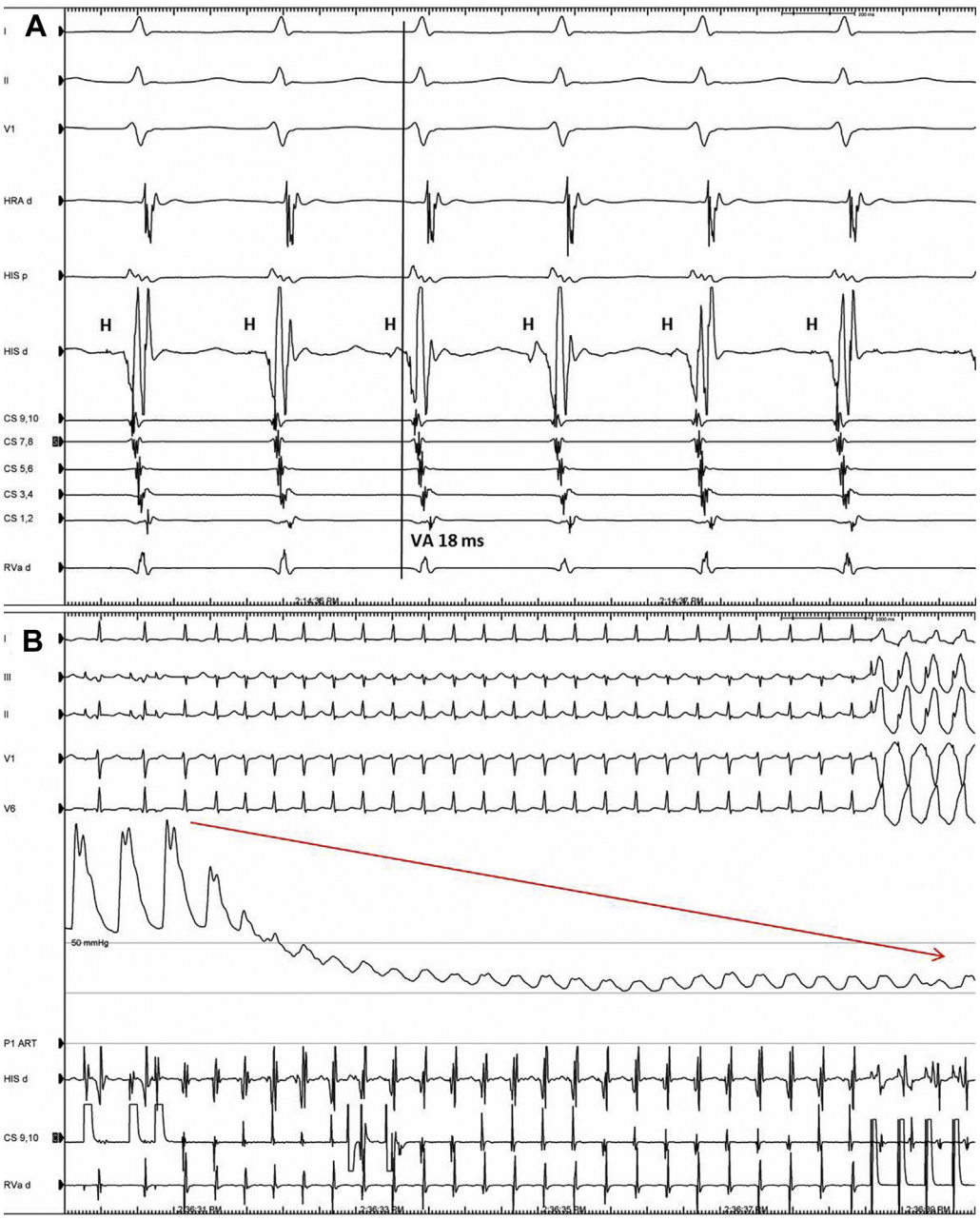

Figure 2 **A:** Typical slow-fast atrioventricular nodal reentry tachycardia (AVNRT), tachycardia cycle length of 380 ms, ventriculoatrial (VA) interval of 18 ms. The vertical line shows the timing of the first ventricular electrogram. **B:** Supraventricular tachycardia initiation with evidence of significant systolic blood pressure drop from 130 mm Hg to 40 mm Hg during typical slow-fast AVNRT. On the right side of the panel, ventricular overdrive pacing for tachycardia termination is shown. H = His.

were inducible anymore at baseline and during/after isoproterenol infusion. No dual AV nodal physiology or AV nodal echo was observed. The patient's HCM risk–SCD score was calculated: his risk of sudden cardiac death was 2.3% at 5 years and an implantable cardiac defibrillator was not indicated. The patient was discharged on 240 mg of verapamil extended release.

Discussion

SVT, regardless of their mechanism or cause, may result in disabling symptoms such as palpitations, chest and neck pounding, shortness of breath, lightheadedness, dizziness, or syncope.[6] The latter is felt to be mainly due to decreased diastolic filling with subsequent reduction in stroke volume and cardiac output.[7,8] The rate of the tachycardia and the presence of underlying cardiac dysfunction play an important role in the severity of signs and symptoms as well. However, AV synchrony is also a major determinant of hemodynamic stability. Several studies have shown that the timing of atrial systole can be responsible for acute increases in atrial pressure if it occurs against a closed AV valve, leading to an increase in pulmonary

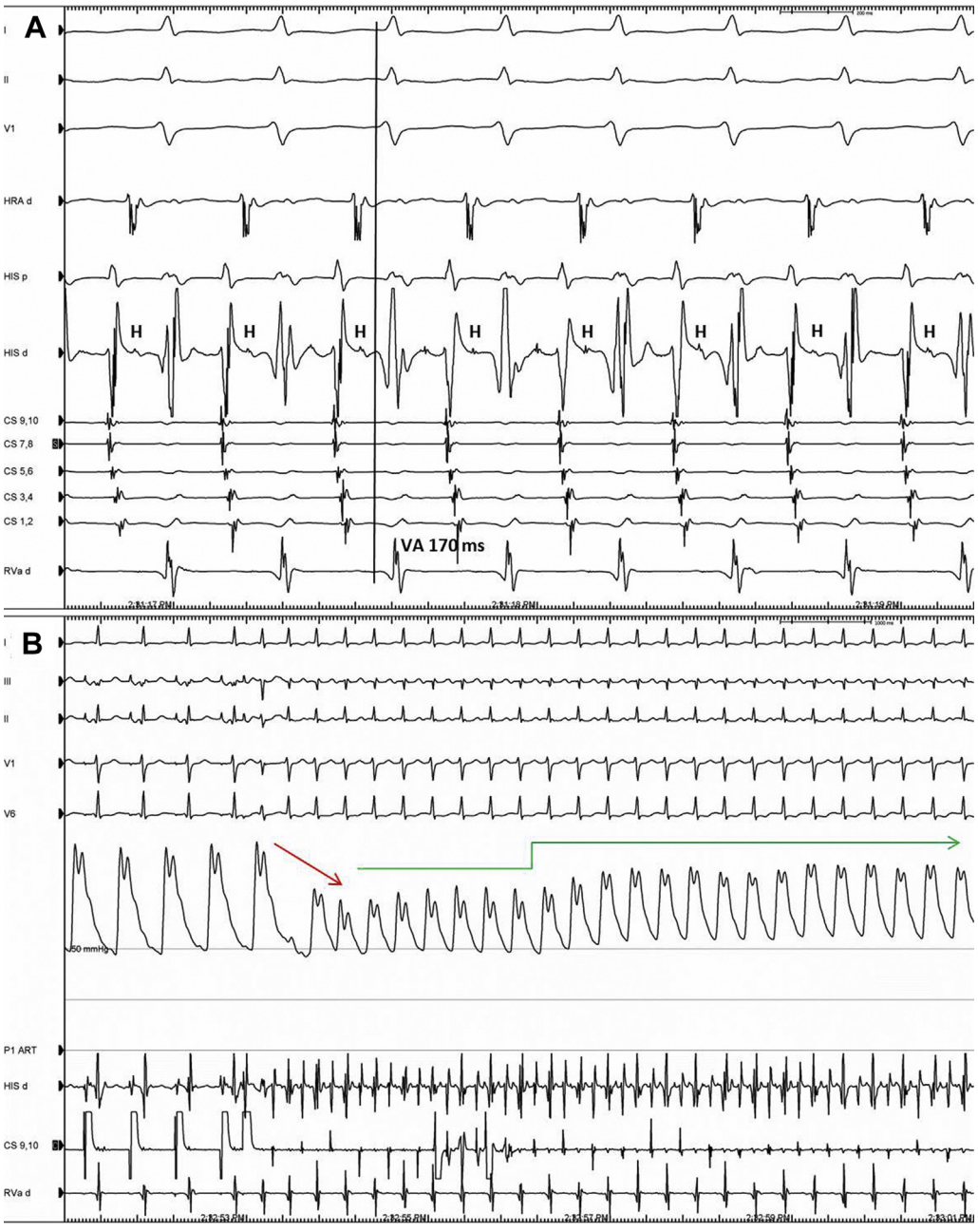

Figure 3 **A:** Atypical fast-slow atrioventricular nodal reentry tachycardia, tachycardia cycle length of 320 ms, ventriculoatrial (VA) interval of 170 ms. The vertical line shows the timing of the first ventricular electrogram. **B:** Supraventricular tachycardia initiation with evidence of milder drop in blood pressure (systolic of 100 mm Hg to 75 mm Hg), followed by gradual recovering to 85 mm Hg. H = His.

pressure and a decrease in cardiac index and blood pressure.[2,6] These changes may be more pronounced in the setting of HCM.

Our patient had AVNRT, induced in its typical and atypical forms. There was a 60 ms difference in tachycardia cycle length between the two, with the atypical fast-slow AVNRT being slightly faster. Nonetheless, the typical slow-fast AVNRT resulted in hemodynamic compromise requiring pace termination with overdrive pacing, whereas the fast-slow AVNRT was better tolerated. A radial arterial line allowed us to document this different blood pressure response during tachycardia. At induction of typical slow-fast AVNRT with near-simultaneous AV activation (VA time 18 ms), the systolic blood pressure dropped from a baseline of 130 mm Hg to 70 mm Hg in less than 2 seconds and continued to decrease down to 40 mm Hg in about 3 seconds. This reproduced the patient's clinical symptoms of dizziness and presyncope even while supine. However, when the atypical fast-slow AVNRT with a VA interval of 170 ms was induced, the blood pressure response was less dramatic (-25 mm Hg from baseline) and gradually improved 3 seconds after tachycardia initiation. This is partially in line with a previous finding by Razavi and colleagues.[9] In a series of 17 patients with AVNRT, blood pressure behavior was examined during sinus rhythm, during AVNRT, and during pacing with an AV delay of 150 ms and 0 ms. Razavi and colleagues observed that blood pressure

decreased immediately after AVNRT initiation, followed by gradual recovery during the first 30 seconds despite persistence of the tachycardia. The same finding was confirmed when patients were paced with an AV delay of 0 ms. When the AV delay was 150 ms, the difference in blood pressure between tachycardia induction and 30 seconds after was not statistically significant. In our case, during typical slow-fast AVNRT, blood pressure decreased immediately but did not recover in the first 30 seconds, explaining the hemodynamic instability and presyncopal symptoms. This finding can likely be ascribed to the cardiac substrate. Patients with HCM have marked diastolic dysfunction, likely exacerbated by intracellular calcium $++$ overload.[10] Diastolic dysfunction, in turn, can impair systolic force generation and function. This phenomenon may explain why patients with HCM poorly tolerate tachycardia, particularly with simultaneous atrial and ventricular activation, and emphasizes the importance of a thorough evaluation of syncope to exclude those with potentially reversible conditions, such as SVT. Since late diastolic function relies on atrial contraction, the loss of effectiveness of atrial systole against a closed AV valve in typical AVNRT has a greater impact than heart rate alone on cardiac output, especially in the presence of structural heart disease and HCM.

Conclusion

This case documents the unique hemodynamic response to SVT depending on its mechanism. Simultaneous AV activation may result in reflex hypotension and cause lightheadedness or syncope. These symptoms may be exacerbated in patients with structural heart disease and diastolic dysfunction, particularly those with HCM and outflow obstruction.

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