All That Glitters is not Gold… All That Faints is not Slow

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Abstract
An 82 year old man presented with multiple syncopal episodes. Since his ECG showed LBBB (left bundle branch block) with first degree AV block, he was advised permanent pacemaker implantation. However, a wide QRS tachycardia on the Holter raised the possibility of tachycardia-mediated syncope. EP (electrophysiological) studies revealed easily and repeatedly inducible short lasting slow-fast AVNRT (atrioventricular nodal reentry tachycardia) with severe hypotension. After RF ablation of the slow pathway, he is asymptomatic at six month follow-up. ©

INTRODUCTION
Finding the cause of syncope is one of the diagnostic challenges. Sometimes despite all the tests the cause remains undetermined. A careful history and sharp clinical acumen helps unravel the cause in many of these cases.

CASE REPORT

A 82 year old gentleman presented with history of numerous “black outs” since 3 years. Of late these episodes occurred daily, unrelated to posture or fasting state. These lasted for a few seconds and were not associated with any other neurological symptoms. He had associated diabetes mellitus and hypertension. Carotid Doppler studies, EEG, MR angiogram of the brain and echocardiogram of the heart were normal.

His baseline ECG showed sinus bradycardia with first degree AV block (P-R interval 220 ms). At sinus rates above 70/ minute, LBBB was also seen. He was referred to us for pacemaker implantation.

Holter monitoring for 24 hrs revealed that the patient was in sinus rhythm with sinus bradycardia for a majority of the time. His average heart rate was 53/min. No pause of 2 seconds or more was noted. There was LBBB whenever his heart rate was faster than 70/min. An ill-sustained broad QRS tachycardia @ cycle length of 326 ms once and 252 ms at another time was noted (Fig. 1). The patient was asymptomatic at both these times.

The patient was subjected to EP study. During the study the basal heart rate was 84/min with LBBB. His basal PR was 203 ms, HV was 62 ms and AH was 108 ms. The AV conduction was robust and AV Wenckebach was noted at 280 ms. The VA conduction was concentric.

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and decremental. The retrograde AV nodal ERP was 290 ms. Ventricular stimulation protocols did not induce any ventricular tachycardia (VT). Atrial premature stimuli repeatedly induced slow fast AVNRT cycle length of 294 ms with LBBB. The tachycardia was associated with a steep drop in systolic blood pressure to 40 mmHg (Fig. 2). This AVNRT was always self-terminated within 10 seconds. When it lasted beyond 5 seconds, the patient’s clinical symptoms were reproduced. Successful RF ablation of the slow pathway was performed. At 6 months follow up he remains asymptomatic.

**DISCUSSION**

The LBBB with 1st degree AV block was presumed to be a marker for bradycardia as the cause of syncope. However, despite multiple syncopal attacks over a period of three years but not a single ECG at any point showed high grade AV block. This should alert one to an alternative etiology of syncope.

His 24 hours Holter monitoring showed few episodes of ill-sustained broad QRS tachycardia. One gets tempted to label these as VT, but when his entire tracings were analyzed this seemed unlikely. Whenever the patient’s heart rate accelerated to more than 70/min he developed LBBB. During the ill-sustained tachycardia episodes the morphology of the QRS complexes was exactly like this LBBB. Importantly, the tachycardia started after a premature atrial complex (*), which conducted with a long PR interval. Supraventricular tachycardia was therefore the most likely diagnosis.

This was confirmed during the EP study when AVNRT was consistently induced. AVNRT is known to cause syncope by various mechanisms: the rapid ventricular rate, reduced cardiac output, reflex vasodilatation and asystole when the tachycardia terminates [as a result of tachycardia-induced depression of sinus node automaticity]. Some authors have noted no relationship between tachycardia rate and the blood pressure.\(^1,^3\) The reproducibility of the patient’s symptoms with the induction of the arrhythmia in the electrophysiology laboratory confirmed AVNRT to be the cause of syncope. Interestingly the AVNRT was always transient and hence could never be recorded prior to the Holter study.

Thus syncope should not be presumed to be due to the first abnormality noted that can cause it. Rather, a careful correlation with the patient’s symptomatology should be attempted. Else, one’s fate may be similar to the Prince of Morocco who read to his dismay: “All that glitters is not gold. . . . . . . . . . \(^4\)”

**REFERENCES**


