Beta Blocker Overdose.. *Atrial Standstill Then Double Wenckebach!*

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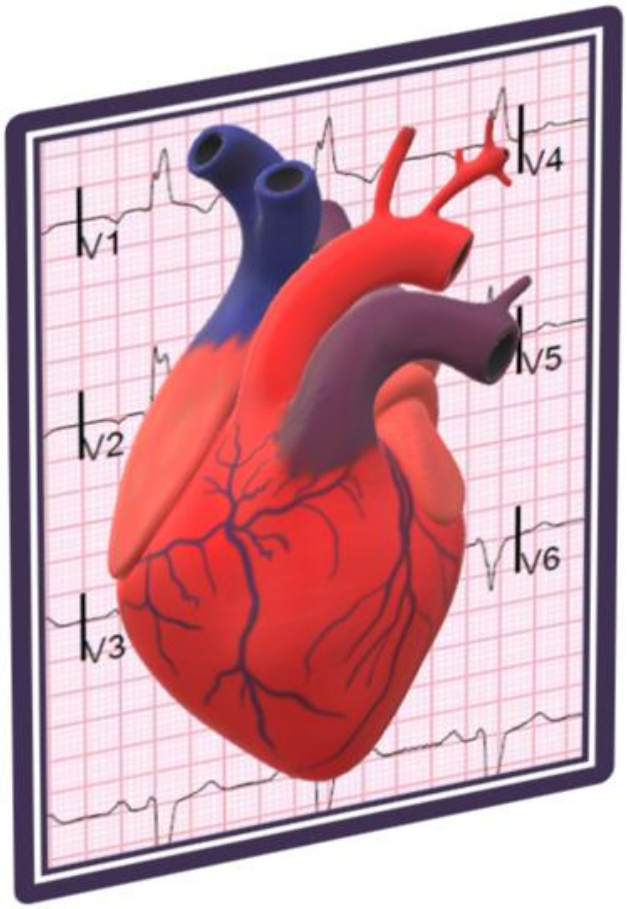

Introduction

Beta adrenergic receptor blocking agents (beta blockers) are a heterogeneous group of medications with variable physiologic and pharmacologic characteristics making them suitable for use as antihypertensive [1], anti-ischemic [2], antiarrhythmic [3] and heart failure [4] therapies. Certain beta blockers also have more specific noncardiac indications such as in the treatment of hyperthyroidism [5], prevention of migraine headaches [6] and prevention of variceal bleeding in cirrhotic patients [7]. Their ubiquitous use dictates that health care providers should be familiar with the recognition and treatment of beta blocker overdose and toxicity [8].

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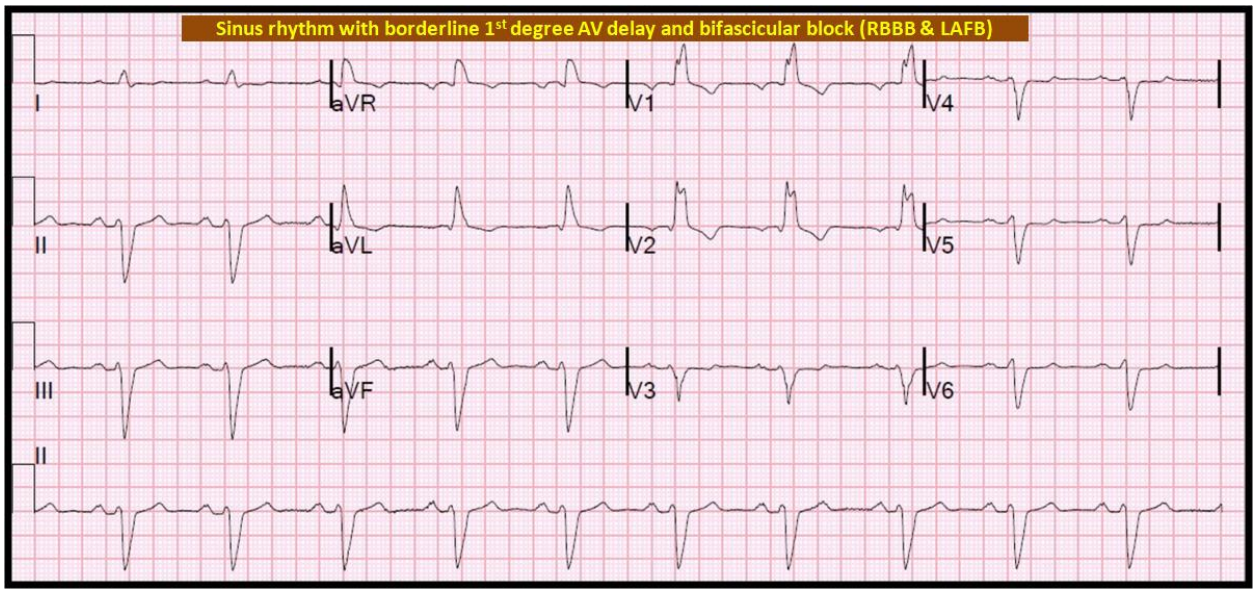

Figure 1. Sinus rhythm 1st degree AV delay and bifascicular block

The above ECG reveals sinus rhythm at 63 beats per minute (BPM). There is borderline first degree AV delay with a PR interval of 210 milliseconds (ms). There is right bundle branch block (RBBB). There is left axis deviation of about – 80 degrees indicative of left anterior fascicular block. This baseline ECG is indicative of extensive disease in the conduction system often referred to as trifascicular block [9], though this may be a misnomer given that the AV node is not considered a fascicle.

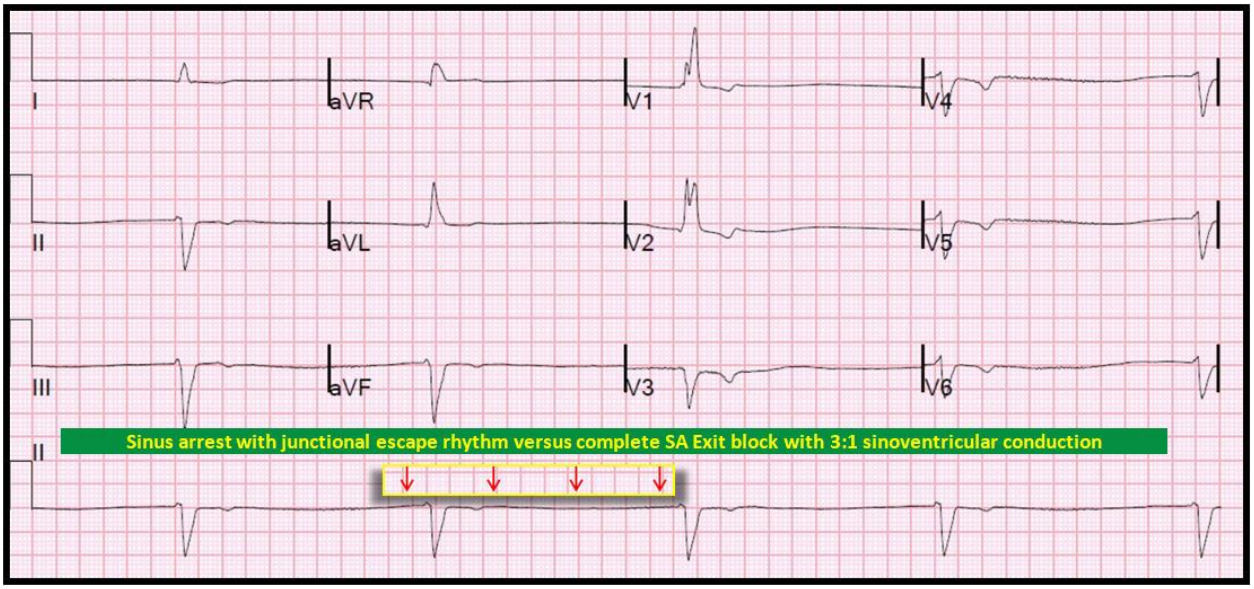

Figure 2. Atrial standstill with wide complex escape rhythm on high dose beta blocker.

The above ECG is the effect of high dose beta blocker on the baseline ECG in Figure 1. There are no P waves (atrial standstill) which may be due to sinus arrest versus complete (third degree) sinoatrial (SA) exit block [10]. The underlying ventricular rhythm is most likely junctional versus sinus capture as is resembles the QRS morphology in Figure 1. The insert represents the simulated sinus rate from Figure 3 to illustrate the possibility of sinus rhythm with complete SA exit block and 3:1 sinoventricular capture [11].

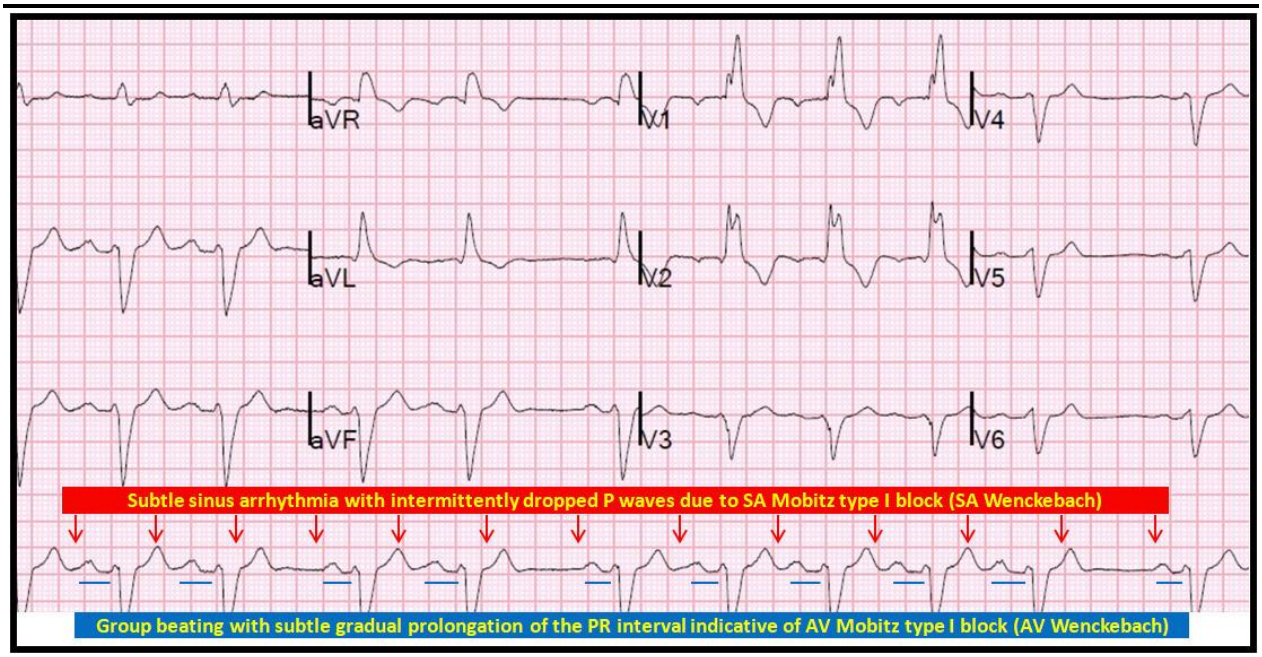

Figure 3. Recovery phase after withdrawal of beta blocker.

The above ECG demonstrates sinus rhythm with borderline first degree AV block, gradual prolongation of the PR interval and group beating suggestive of Mobitz type I AV block (AV Wenckebach). However, there are no blocked P waves when expected. This is often caused by a similar phenomenon at the level of the SA node, with gradual prolongation of the SA conduction until one does not conduct resulting in a pause on the surface ECG; this is second degree type I SA exit block (SA Wenckebach). The simulated sinus rhythm has minor irregularity due to presumed sinus arrhythmia. The R – R intervals in Figure 2 are equivalent to three simulated sinus intervals from Figure 3, therefore suggesting complete SA exit block with 3:1 sinoventricular conduction as the mechanism of the atrial standstill.

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