

## Case Report

# Persistent Narrow Complex Tachycardia: What is the Diagnosis?

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**ABSTRACT**

Supraventricular tachycardia (SVT) with long RP interval and short PR interval is a unique form of tachycardia. The differential diagnosis includes sinus tachycardia, focal atrial tachycardia, atrial flutter with two to one ventricular response and atrioventricular reciprocating

tachycardia with slow retrograde ventriculoatrial conduction. In this report, we present a case of long RP SVT and a review of the electrocardiographic features of each type of tachycardia.

KEYWORDS : atrial flutter, long RP tachycardia, supraventricular tachycardia.

**INTRODUCTION**

Narrow complex tachycardia (NCT) is a commonly presenting condition to the emergency room. The optimal short-term and long-term patient's management will be influenced by understanding the exact mechanism of the tachycardia. The routine use of AV node blocking agent or maneuvers such as intravenous adenosine is not always successful in terminating Supraventricular tachycardia (SVT), as the case with atrial flutter (AFL) or focal atrial tachycardia (AT) demonstrated. The long-term medical and catheter-based therapy will vary from one tachycardia to the other. SVT with long RP, where the P wave is seen before each QRS complex, is a more challenging diagnostic condition, since it has a wide range of differential diagnosis. A careful electrocardiographic assessment, during tachycardia, even before termination, is the most important step to understand the tachycardia mechanism and to apply the appropriate treatment plan.

**Case:**

A 45-year-old man was referred with a history of palpitation at rest accompanied by dyspnea. He had a previous history of myocardial infarction and mild congestive heart failure. The 12-lead electrocardiogram shown in Fig. 1 was recorded after two days of heart failure stabilization.

**DISCUSSION**

This ECG shows narrow complex regular tachycardia at a rate of 140 bpm with P wave preceding every QRS complex. The P to R

relationship is at ratio of 1:1 with abnormal P wave vector of negative P wave in the inferior leads (II, III and AvF). The normal sinus rhythm P wave is usually positive in the II, III and AvF leads and biphasic in V1 lead. These features categorize the tachycardia as supraventricular tachycardia with long RP interval. The differential diagnosis of long RP SVT is the following:

1. Sinus tachycardia, which was ruled out by the abnormal P wave morphology. This patient also has persistent tachycardia at a fixed heart rate in spite of good heart failure stabilization. Sinus tachycardia usually has warming-up and cooling-down characteristics, as a result of autonomic tone fluctuation.

2. Paroxysmal supraventricular tachycardia (PSVT) or Re-entrant AV tachycardia, with atypical slow return pathway from the ventricle to the atrium causing delay of atrial activation. Atrioventricular nodal re-entrant tachycardia hyper (AVNRT) with slow-slow physiology and AV reciprocating tachycardia (AVRT) using a retrograde slow accessory pathway, both can present with long RP tachycardia instead of the common variant of short RP interval. In the typical form of PSVT, RP usually is short and measures equal or less than 90 mseconds with longer PR interval<sup>[1]</sup>. As a result, P wave is falling in the T wave in the service ECG, which makes the P wave either invisible or seen as small squiggles within the T-wave when using unfiltered ECG<sup>[2]</sup>. While in the long RP PSVT, the P wave is falling later in diastole with abnormal morphology due to different sequence of activation of the atria in caudo-cranial direction, i.e., low to

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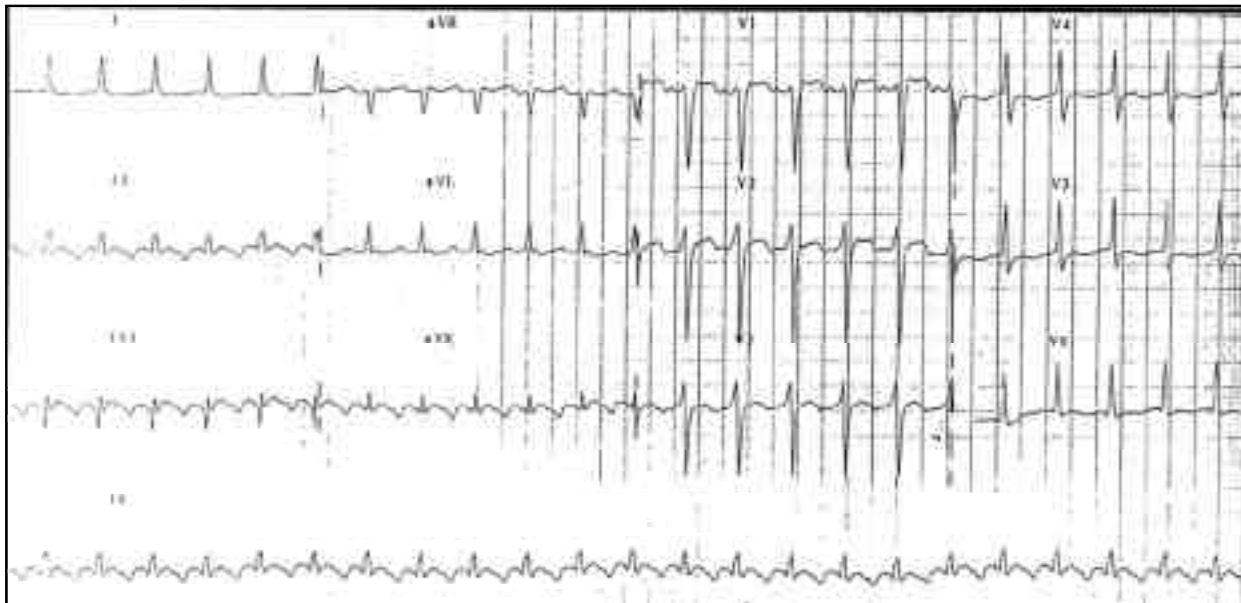


Fig. 1: Regular narrow complex tachycardia during initial presentation, with a heart rate of 140 bpm.

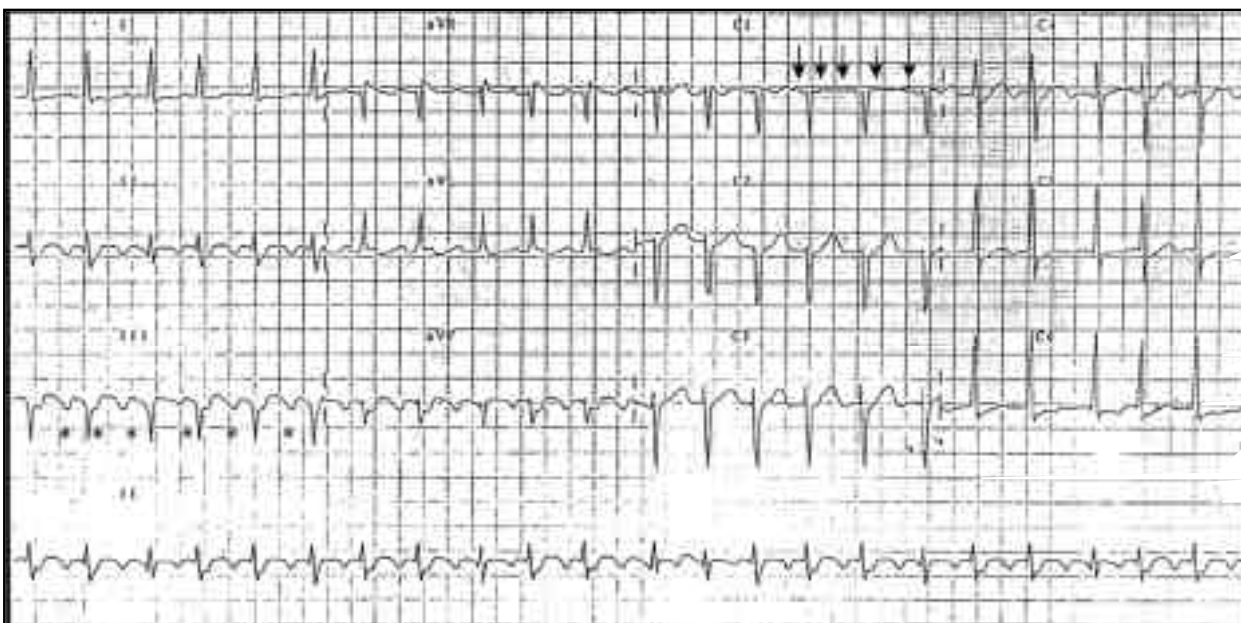


Fig. 2: Tachycardia after 2 mg of IV propranolol was given. Heart became 130 bpm with longer PR intervals and presence of variable conduction block. V1-V3 shows 2:1 pattern (arrows) and V4-V6 shows regularly irregular rhythm. Notice the typical morphology of flutter wave in lead II, III and AvF (asterisks).

high atrium.

3. Atrial flutter and/or atrial tachycardia with 2:1 conduction from the atria to the ventricles over the AV node<sup>13-41</sup>. The second flutter wave cannot be seen because it is hidden in the QRS complex. The flutter cycle length is usually 200-240 mseconds, equivalent to a rate of 280-300 bpm in the atria. With 2:1 conduction, the ventricular rate will be around 140 -150 bpm.

To verify the arrhythmia further, the patient was given a small dose of intravenous Propranolol. The rate of the tachycardia was slowed down only to 130 bpm, without causing tachycardia termination

(Fig. 2). Slowing of the tachycardia helps in two ways to confirm the presence of atrial flutter with 2:1 AV block as the definitive diagnosis of this case. First, the appearance of the second P wave at the slower rhythm ECG clearly demonstrates the presence of 2:1 atrioventricular activation (Fig. 2 arrows). Second, the prolongation of the RR interval due to AV node block during sustained tachycardia, which proves that the tachycardia is AV node independent and the AV node is not part of the tachycardia mechanism. Therefore, it is not likely for the tachycardia to be AV node dependent tachycardia such as AVNRT or AVRT (differential

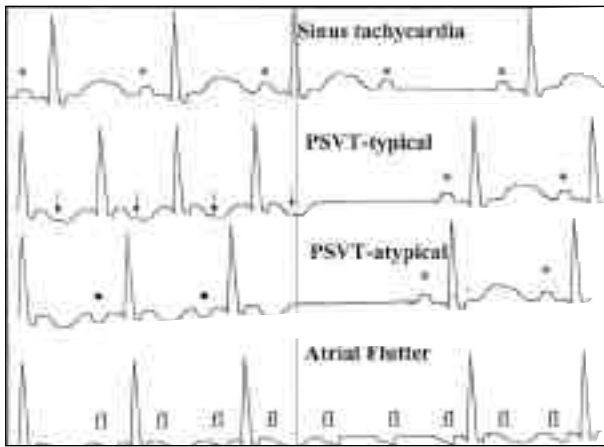


Fig. 3: Schematic diagram for the various forms of tachycardia, from top to bottom: sinus tachycardia, typical PSVT, atypical PSVT and atrial flutter with 2:1 conduction. Following the administration of intravenous adenosine (vertical dotted line), different effects are seen in different tachycardia. See text for details. \* For sinus Pwave, • for Pwave inscribed in T wave, • for Pwave of late RPPSVT and fl for flutter wave.

diagnosis number 2) were the tachycardia either terminated or sustained, but not slowed down when treated with AV blocking agent. Besides, the atrial signal clearly has the classical saw-tooth appearance with the descending slope being slower than the ascending slope, which supports the presence of the typical atrial flutter wave (Fig. 2).

The presence of high autonomic tones make the AV node less sensitive to AV blocking agent in particular in patients with decompensated heart function. The use of higher dose of beta-blocker should be made with caution since it might carry some paradoxical effect on the sympathetic tone through its hypotensive effect. When necessary, a very short acting drug can be used such as intravenous esmolol or adenosine with rapid ECG recording after the slowing phase of tachycardia. Effect of AV node blockade in different forms of tachycardia is illustrated in Fig. 3.

During an electrophysiology study of this

patient, an atrial flutter circuit was identified inside the right atrium, with counterclockwise movement down the free wall and up the septum, passing through the narrow cavo-triupid isthmus. Radiofrequency ablation with a linear lesion was performed at this area, which caused termination and cure of the atrial flutter. The patient was treated with amiodarone and small dose beta-blocker for many days before the ablation procedure.

In conclusion, long RP supraventricular tachycardia can be missed as an inappropriate sinus tachycardia. When left untreated, it can cause further deterioration of left ventricular function<sup>[5]</sup>. Atrial flutter with fixed 2:1 ratio conduction to the ventricle might be overlooked because of simultaneous occurrence of the QRS complex and the second flutter wave. Rare forms of re-entrant tachycardia can be manifested as long RP tachycardia due to late retrograde atrial activation. The use of short acting AV blocking agent and repeat electrocardiograms helps to make the precise diagnosis at bedside level.

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