The Protocol Book

for Intensive Care

Editor
Soumitra Kumar

6th Edition

JAYPEE

Contents

7 Tachycardias

CHAPTER

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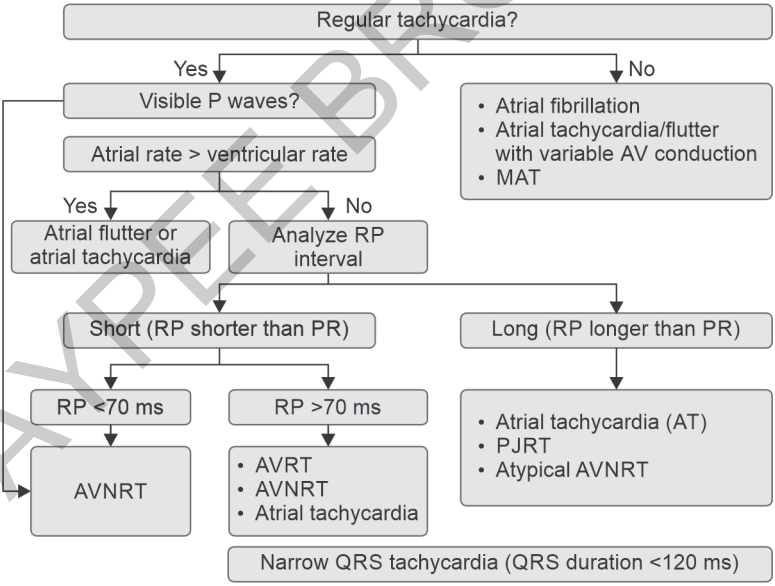
Tachycardias can be broadly classified based on the QRS complex duration into:

- Narrow QRS complex tachycardia when QRS is <120 ms in duration.
- Wide QRS complex tachycardia when QRS is ≥120 ms in duration.

Flowchart 1: Approach to the patient with narrow QRS tachycardia (QRS duration).



**In surface ECG, the onset of QRS wave to the onset of P wave, i.e., RP = 90 ms; as opposed to 70 ms in case of ventriculoatrial interval.*

(AT: atrial tachycardia; AV: atrioventricular; AVNRT: AV nodal reentry tachycardia; AVRT: atrioventricular reciprocating tachycardia; MAT: multifocal atrial tachycardia; PJRT: permanent junctional reciprocating tachycardia)

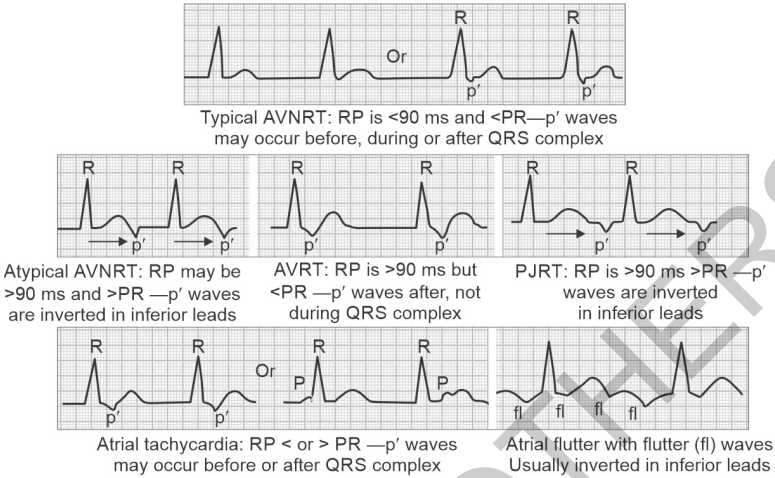


Fig. 1: P wave morphology and its relation with RP and PR Intervals in narrow QRS tachycardia. (AVNRT: AV nodal re-entrant tachycardia. AVRT: AV re-entry tachycardia. PJRT: permanent form of AV junctional reciprocating tachycardia)

P wave morphology and its relationship with RP and PR intervals in narrow QRS tachycardia.

THERAPY OF SINUS TACHYCARDIAS

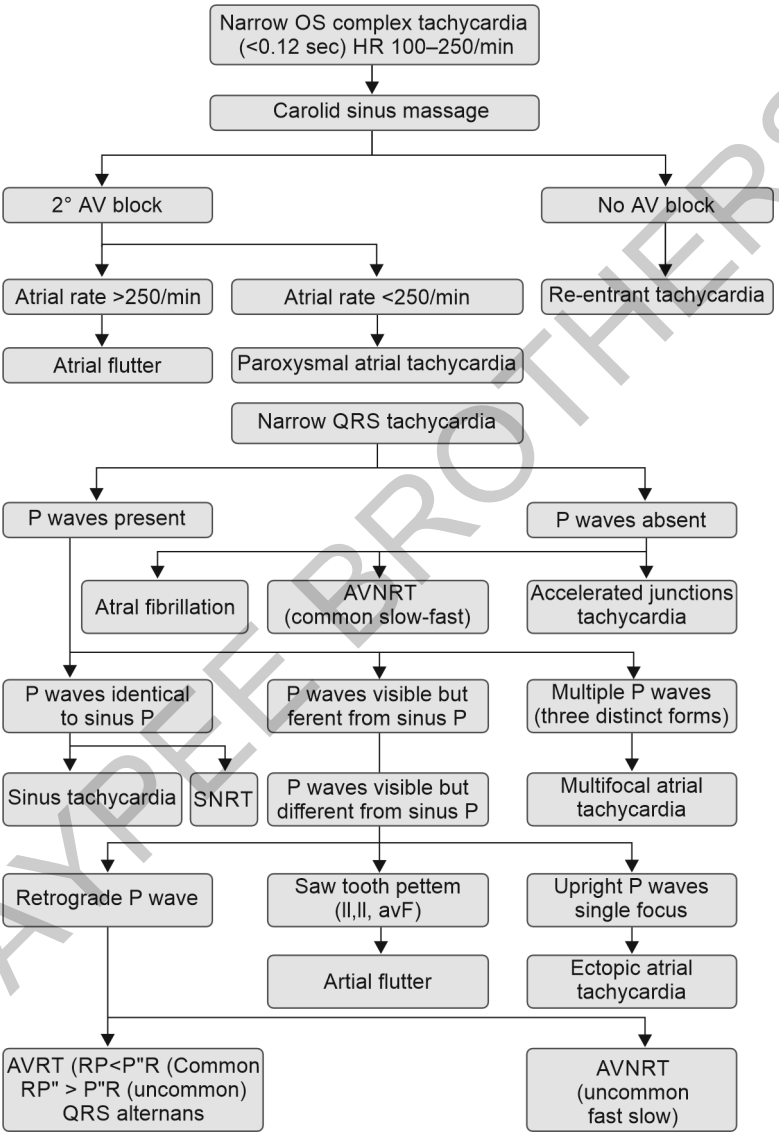
Focal Atrial Tachycardia

Focal atrial tachycardia (AT) is defined as an organized atrial rhythm ≥100 bpm arising from a discrete origin and disseminating over both atria in a centrifugal pattern. The ventricular rate varies, depending on AV nodal conduction. In asymptomatic young people (<50 years of age), the prevalence of focal AT has been reported to be as low as 0.34% with an increased prevalence of 0.46% in symptomatic arrhythmia patients. Most studies have not reported any gender preferences.

Symptoms may include palpitations, shortness of breath, chest pain, and rarely syncope or presyncope. The arrhythmia may be sustained or incessant. Dynamic, forms with recurrent interruption and reinitiations have also been described.

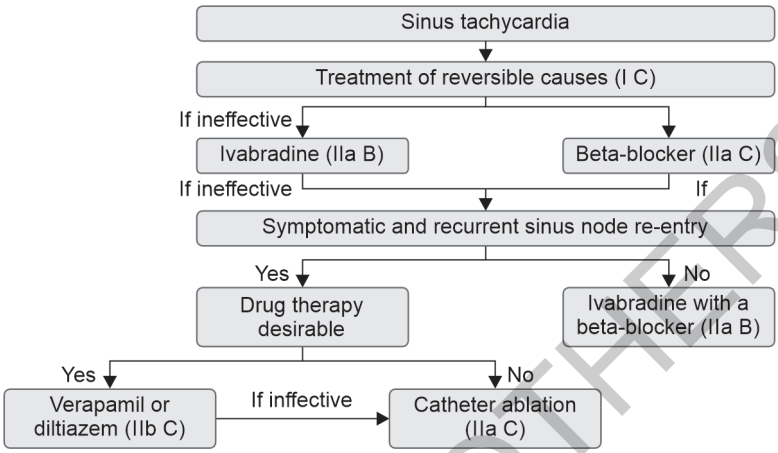
If patients with pulmonary vein-related (PV) AT, the focus is located at the ostium of the vein (or within 1 cm of the designated ostium) rather than further distally (2–4 cm).

Flowchart 2: Approach to the patient with narrow QRS complex tachycardia.

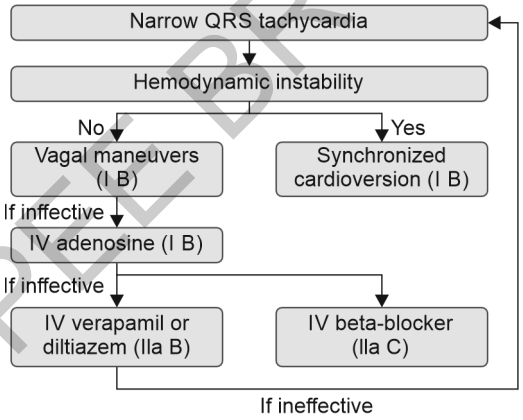


(AVNRT: AV nodal re-entry tachycardia; AVRT: atrioventricular reciprocating tachycardia; SNRT: sinus node recovery time; avF: ateriovenous fistula)

Flowchart 3: Therapy of sinus tachycardia.



Flowchart 4: Acute therapy of narrow QRS tachycardia in the absence of an established diagnosis.

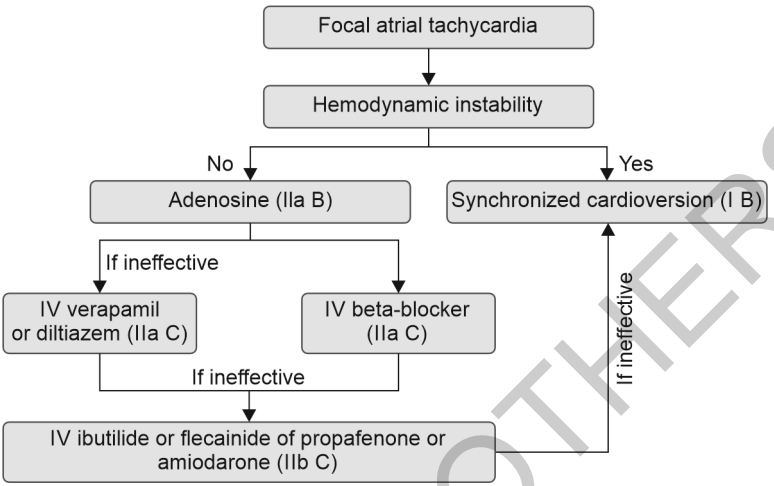


(IV: intravenous)

Diagnosis

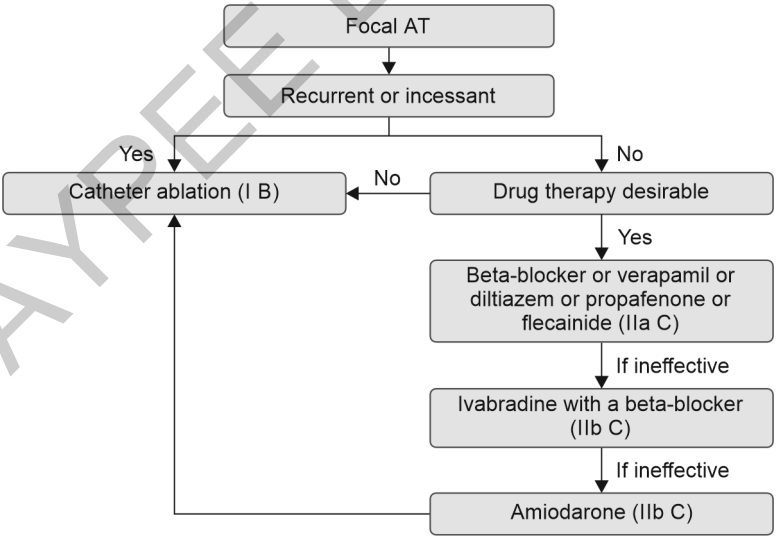
P wave identification from a 12-lead ECG recording during tachycardia is critical. Depending on the AV conduction and AT rate, the P waves may be hidden in the QRS or T waves. The P waves are monographic with stable CL, which helps to rule our organized AF. Adenosine injection can help by slowing the ventricular rate

Flowchart 5: Acute therapy of focal atrial tachycardia.



(AT: atrial tachycardia; IV: intravenous)

Flowchart 6: Chronic therapy of focal atrial tachycardia.



(AT: atrial tachycardia)

TABLE 1: ESC 2019 recommendations for the therapy of multifocal atrial tachycardia.

Recommendations	***Class***[a]	***Level***[b]
Acute therapy		
Treatment of an underlying condition is recommended as a first step, If feasible	I	C
IV beta-blockers or IV nondihydropyridine calcium channel blockers (verapamil or diltiazem) should be considered	IIa	B
Chronic therapy		
Oral verapamil or diltiazem should be considered for patients with recurrent symptomatic multifocal AT in the absence of HFrEF	IIa	B
A selective beta-blocker should be considered for patients with recurrent symptomatic multifocal AT	IIa	B
AV nodal ablation followed by pacing (preferable biventricular or His-bundle pacing) should be considered for patients with LV dysfunction due to recurrent multifocal AT refractory to drug therapy	IIa	C

IV = Verapamil and diltiazem are contraindicated in the presence of hypotension or HFrEF
IV = Beta-blockers are contraindicated in the presence of decompensated heart failure
Note:
[a]Class of recommendation
[b]Level of evidence
(AT: atrial tachycardia; HF: heart failure; HFrEF: heart failure with reduced ejection fraction; IV: intravenous; LV: left ventricular)

or less frequently, by terminating focal AT. A discrete P wave with an intervening isoelectric interval suggests a focal AT. However, distinguishing focal from macroreentrant arrhythmias by surface ECG is not always possible. The presence of an isoelectric line does not always rule out a macroreentrant mechanism, particularly in the presence of scar atrial tissue (from structural heart disease or previous extensive ablation/surgery procedures). In a normal heart and in the absence of pervious ablation, the usual ECG localization rules apply.

MACROREENTRANT ATRIAL TACHYCARDIAS

Atrial flutter and focal AT are traditionally defined according to the ECG appearance of continuous regular electrical activity, most commonly a saw-tooth pattern, versus discrete P waves with an isoelectric line in between ECGs with flutter-like appearances are mostly due to macro-co-entrant atrial circuits but microreentry is also possible.

However, MRATs with a significant part of the activation of the circuit in protected areas may display a focal AT pattern, with discrete P waves.

Typical Atrial Flutter: Counter-clockwise and Clockwise

Typical common atrial flutter is the most frequent cavotricuspid isthmus (CTI)-dependent flutter, i.e., a macroreentry circuit around the tricuspid annulus using the CTI as a critical passage at the inferior boundary. Activation goes downward in the RA-free wall, through the CTI, and ascends in the right septum. Activation of the LA is passive. The upper part of the circuit may be anterior or posterior to the superior vena cava. This activation is also known as counter-clockwise (or anticlockwise) when seen from the apex. When the circuit is activated in the opposite direction, i.e., clockwise, it results in different ECG pattern, then called typical reverse flutter.

Diagnosis

In counter-clockwise flutter, the circuit results in regular atrial activation from 250 to 330 bpm with negative saw-tooth waves in inferior leads and positive waves in V1. In clockwise flutter, ECG flutter-waves in inferior leads look positive and broad, and are frequently bimodal negative in V1. Typical atrial flutter has a strong reproducible anatomical dependence, resulting in the morphological reproducibility of the ECG. However, this well-recognized ECG pattern may be significantly altered when atrial activation has been modified, as it is in cardiac surgery involving atrial tissue, after extensive radiofrequency ablation, or in advanced atrial disease. Antiarrhythmic drugs may also modify the typical ECG pattern.

TABLE 2: Recommendations for the therapy of macro-re-entrant atrial arrhythmias (MRATs)/atrial flutter.

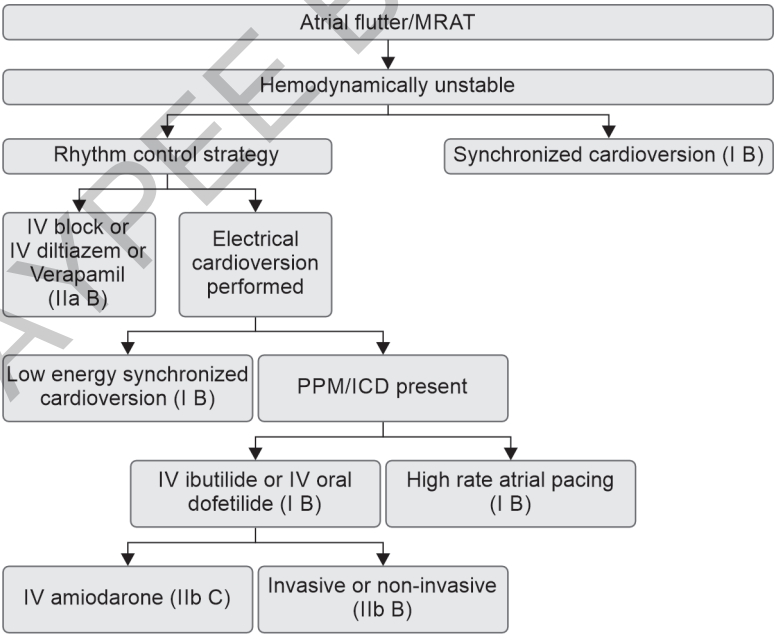
Recommendations	***Class***	***Level***
Anticoagulation, as in AF, is recommended for patients with atrial flutter and concomitant AV	I	B
Patients with atrial flutter without AF should be considered for anticoagulation, but the threshold for initiation has not been established	IIa	C
Acute therapy		
Hemodynamically unstable patients: Synchronized DC cardioversion is recommended for hemodynamically unstable patients hemodynamically stable patients	I	B
IV ibutilide or IV or oral (in-hospital) dofetilide is recommended for conversion to sinus rhythm	I	B
Low-energy (≤100 biphasic) electrical cardioversion is recommended for conversion to sinus rhythm	I	B
High-rate atrial pacing is recommended for termination of atrial flutter in the presence of an implanted pacemaker or defibrillator	I	B
IV beta-blockers or nondihydropyridine calcium channel blockers (verapamil or diltiazem) (IV) should be considered for control of rapid ventricular rate	IIa	B
Invasive and noninvasive high-rate atrial pacing may be considered for termination of atrial flutter	IIb	B
IV amiodarone may be tried if the above are not available or desirable	IIb	C
Propafenone and flecainide are not recommended for conversion to sinus rhythm	III	B
Chronic therapy		
Catheter ablation should be considered after the first episode of symptomatic typical atrial flutter	IIa	B
Catheter ablation is recommended for symptomatic, recurrent episodes of CTI-dependent flutter	I	A
Catheter ablation is experienced centers is recommended for symptomatic, recurrent episodes of non-CTI-dependent flutter	I	B

Contd...

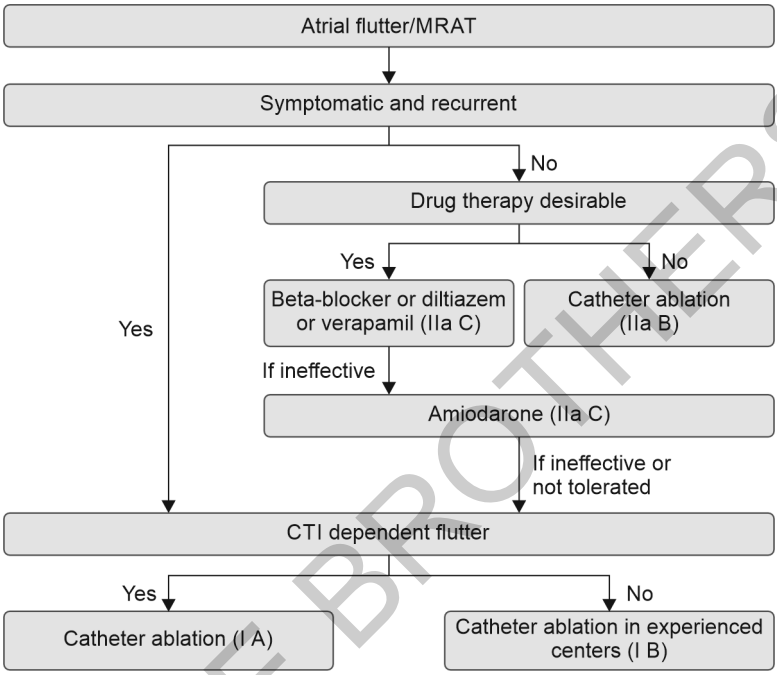
Contd...

Recommendations	*Class*	*Level*
Catheter ablation is recommended in patients with persistent atrial flutter or in the presence of depressed LV systolic function due to TCM	I	B
Beta-blockers or non-dihydropyridine calcium channel blockers (verapamil or diltiazem, in the absence of HFrEF should be considered if ablation is not desirable of feasible	IIa	C
Amiodarone may be considered to maintain sinus rhythm if the above measures fail	IIb	C
AV nodal ablation with subsequent pacing (ablate and pace), either biventricular or His-bundle pacing, should be considered if all the above fail and the patient has symptomatic persistent macroreentrant atrial arrhythmias with fast ventricular rates	IIa	C

Flowchart 7: Acute therapy of stable atrial flutter or macroreentrant atrial tachycardia (ESC 2019 guidelines).



Flowchart 8: Chronic therapy of atrial flutter/macroreentrant atrial tachycardia (ESC 2019 guidelines).



(CTI: cavotricuspid isthmus; MRAT: macroreentrant atrial tachycardia)

In these situations, an atypical ECG does not rule out a circuit of typical flutter using the CTI.

Typical flutter is related to AF in clinical practice, with both being associated with similar clinical settings and coexisting in the same patients. AF may trigger atrial flutter, and after typical flutter ablation AF is frequent. Typical flutter may also frequently occur in patients treated for AF with class IC drugs or amiodarone. In this case, flutter rate may be reduced to <200 bpm facilitating 1:1 AV conduction. The action of antiarrhythmic drugs on ventricular activation may result in wide QRS tachycardia.

Beyond symptoms associated with high-rate and loss of atrial kick, reversible systolic dysfunction, and subsequent DCM are not unusual.

OTHER CAVOTRICUSPID ISTHMUS-DEPENDENT MACROREENTRANT ATRIAL TACHYCARDIA

An atypical ECG pattern may not exclude CTI-dependent MRAT. Lower-loop reentry refers to a circuit rotating around the inferior vena cava instead of around the tricuspid annulus. It may be clockwise or counter-clockwise. when rotating counter-clockwise, it might be considered a variant of typical counter-clockwise flutter with a caudal shift of the cranial turning point posterior to the entry of the superior vena cava, resulting in a similar ECG appearance. Figure-of-eight double-loop reentry may also occur around the inferior vena cava and tricuspid annulus, and mimic typical clockwise atrial flutter. Other circuits using part of the CTI or even restricted inside it are in essence CTI-dependent with a similar ECG appearance to typical common flutter.

Noncavotricuspid Isthmus-dependent Macroreentrant Atrial Tachycardia

The terms non-CTI-dependent MRAT and atypical flutter are used interchangeably, and describe flutter waves in the ECG not suggestive of typical circuits. The pitfall with this use comes from the atypical ECG that may happen when typical circuits develop in diseased atria, most frequently after surgery or extensive ablation, or under the effects of antiarrhythmic drugs. Conversely, upper-loop reentry may mimic a typical flutter ECG pattern without being CTI-dependent. True atypical flutter is actually a posthoc diagnosis when the circuit has been outlined and dependence on CTI has been ruled out.

ATRIOVENTRICULAR JUNCTIONAL ARRHYTHMIAS

Atrioventricular Nodal Reentrant Tachycardia

Atrioventricular nodal reentrant tachycardia (AVNRT) denotes reentry in the area of the atrioventricular node (AVN), but the exact circuit remains elusive. The AVN is a three-dimensional structures with greater variability in the space constraint of tissue, and poor gap junction connectivity due to differential expression of connexin isoforms., conditions that provide an explanation of or dual conduction and nodal reentrant arrhythmogenesis. There has also

been considerable histological and electrophysiological evidence that the right and left inferior extensions of the human AVN, and the atrionodal inputs that they facilities may provide the anatomical substrate for the slow pathway. Thus, comprehensive models of the tachycardia circuit for all forms of AVNRT based on the concept of atrionodal inputs have been proposed.

Onset of AVNRT seems to occur bimodally over time. In many patients, attacks indeed manifest early in life. Whereas in a

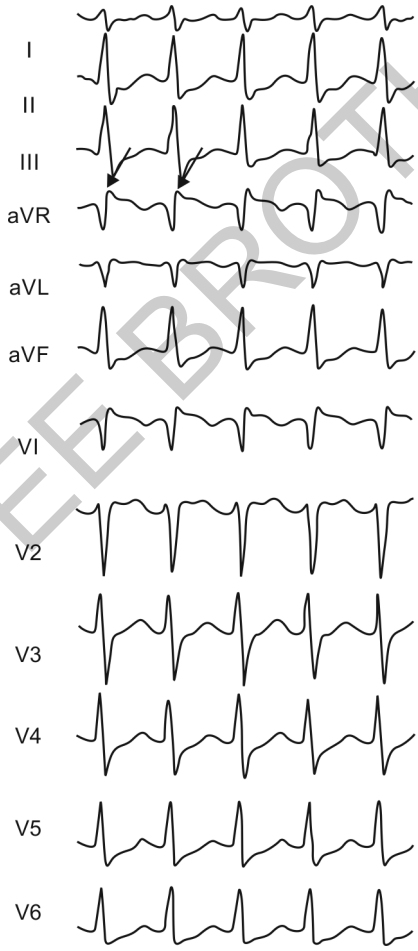


Fig. 2: Typical atrioventricular nodal reentrant tachycardia.

The Protocol Book
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The sixth edition (2025) of *The Protocol Book for Intensive Care* is a comprehensive compendium of contemporary guidelines, statements, and consensus opinions on various cardiac and allied medical topics. All the chapters have been appropriately modified or upgraded to present the latest scientific evidence-based information on these subjects.

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Apart from the first five editions of this title *(The Protocol Book for Intensive Care)*, he has also edited *Current Trends in Cardiology* (1st edition, 2000) and (2nd edition, 2005) and *Update in Cardiology 2006* (for CSI, West Bengal Branch). He has been Joint Editor of a treatise on *Acute Coronary Syndrome* (2013) with Dr Christopher P Cannon (USA) and a treatise on *Echocardiography in Clinical Practice*. He has been an Associate Editor of a *Monograph on Nonspecific Aorto-arteritis*. He has edited two editions of a treatise-titled *Clinico-Echo Ensemble* (2017 and 2019). He has played a key role as an Associate Editor of a prestigious publication by CSI, captioned *Cardiology in Indian Perspective: A Comprehensive Text* (2017). Dr Soumitra Kumar has established himself as a distinguished Academician, keen Clinician, and erudite Orator and a Teacher.

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