# Supraventricular Reentrant Tachycardias

19

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## 19.1 Case Report

A 61-year-old man presented to the emergency room for an episode of malaise characterized by palpitations, sweating, dizziness, and chest pain that lasted for more than an hour.

### Medical History and Cardiovascular Risk Factors

- Type 2 diabetes mellitus
- Arterial hypertension
- Dyslipidemia
- Overweight
- Post-traumatic subdural hematoma which required neurosurgical drainage about 1 year ago

### Allergies

No allergy is referred by the patient.

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### Social History

The patient does regular physical activity, smoked about 30 cigarettes/day until 7 years ago, and never used illicit drugs.

### Medications

Pantoprazole at 8:00 AM, telmisartan/hydrochlorothiazide 80/12.5 mg at 8:00 AM, aspirin 100 mg at 12:00 AM, metformin 500 mg at 12:00 AM and at 8:00 PM, and atorvastatin 20 mg at 10:00 PM

### Vital Signs

- Temperature: 35.4 °C
- Heart rate: 200 beats per minute
- Blood pressure: 115/80 mmHg
- Respiratory rate: 18/min
- Oxygen saturation while breathing in ambient air: 98 %

### Physical Examination

- General: fatigued, sweaty, alert, awake, and oriented
- Head, eye, ear, nose, and throat: normocephalic, atraumatic, mucous membranes moist,



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extraocular muscles intact, pupils equally round and reactive to light and accommodation bilaterally, bilateral tympanic membrane intact, bilateral sclera anicteric, and no conjunctival injection
- Neck: supple, no jugular venous distention, no lymphadenopathy, no carotid bruit
- Cardiovascular: regular rhythm, tachycardia rate, S1 and S2 normal, and no murmurs
- Lungs: no rales, rhonchi, or wheezes, no egophony, no alterations in tactile fremitus, and normal percussion
- Abdomen: overweight, no pulsatile masses, normal bowel sounds in all four quadrants, no high-pitch or tinkling sounds, resonant to percussion, soft, non-distended/non-tender, no rebound or guarding, no costovertebral angle tenderness, and no hepatosplenomegaly
- Extremities: no cyanosis or clubbing and no edema
- Neurologic: cranial nerves I through XII intact and no focal deficit
- Psychiatric: normal affect, no hallucinations, and normal speech
- Skin: intact, sweaty, and no rushes, and no lesion

## Routine Laboratory Tests

- Complete blood count: normal (hemoglobin 13 g/dl)
- Inflammatory markers (ESR, CRP): normal
- Hepatic function (GOT, GPT, γ-GT, ALP, total bilirubin, direct and indirect): normal
- Renal function (creatinine, BUN): normal
- Electrolytes ($Na^+$, $K^+$, $Ca^{++}$, $Mg^{++}$, $Cl^-$): normal
- Thyroid function (TSH, fT3, fT4): normal
- Fasting blood glucose: 93 mg/dl
- HbA1C: 7.0 % (53 mmol/mol)
- Hs-TnT: 120 pg/ml (highest value)

## Routine 12-Lead ECG at Rest (Fig. 19.1)

The ECG showed a narrow QRS complex tachycardia. Heart rate was 200 beats per minute (RR 300 ms). The QRS axis is about + 20°. P waves were not clearly visible. ST segment depression was present in I, II, aVF, and V3–V6 leads. ST segment elevation was present in aVR and V1 leads.

## What Are the Possible Types of Supraventricular Arrhythmias?

- Sinus tachycardia
- Reentrant supraventricular tachycardias
  - Atrioventricular reciprocating tachycardia
  - Atrioventricular nodal reciprocating tachycardia
- Focal atrial tachycardia
- Atrial flutter
- Atrial fibrillation

The presence of a regular rhythm excludes the hypothesis of atrial fibrillation. There isn't a clearly visible P wave, probably because it is inside the terminal part of the ventricular complexes. The atrioventricular ratio is 1:1. Heart rate (200 bpm) is too low for a 1:1 atrial flutter, so this hypothesis is unlikely. Even if the morphology of the P wave is not assessable, we can exclude the hypothesis of a sinus tachycardia because there were no physiological causes for having high heart rate at rest. RP interval was shorter than PR and longer than 70 ms. The most likely diagnoses left are therefore atrioventricular reciprocating tachycardia, atrioventricular nodal reciprocating tachycardia, and atrial tachycardia.

Vagal maneuver response may allow to distinguish between the different forms of supraventricular tachycardias. Atrial tachycardia generally is conducted with a transitory high-grade atrioventricular block. Reentrant supraventricular tachycardias end suddenly. In this case, the sinus carotid massage caused a sudden tachycardia interruption (Fig. 19.2).

Therefore, the diagnosis was reentrant supraventricular tachycardia. The high heart rate (about 200 bpm) was more suggestive for an

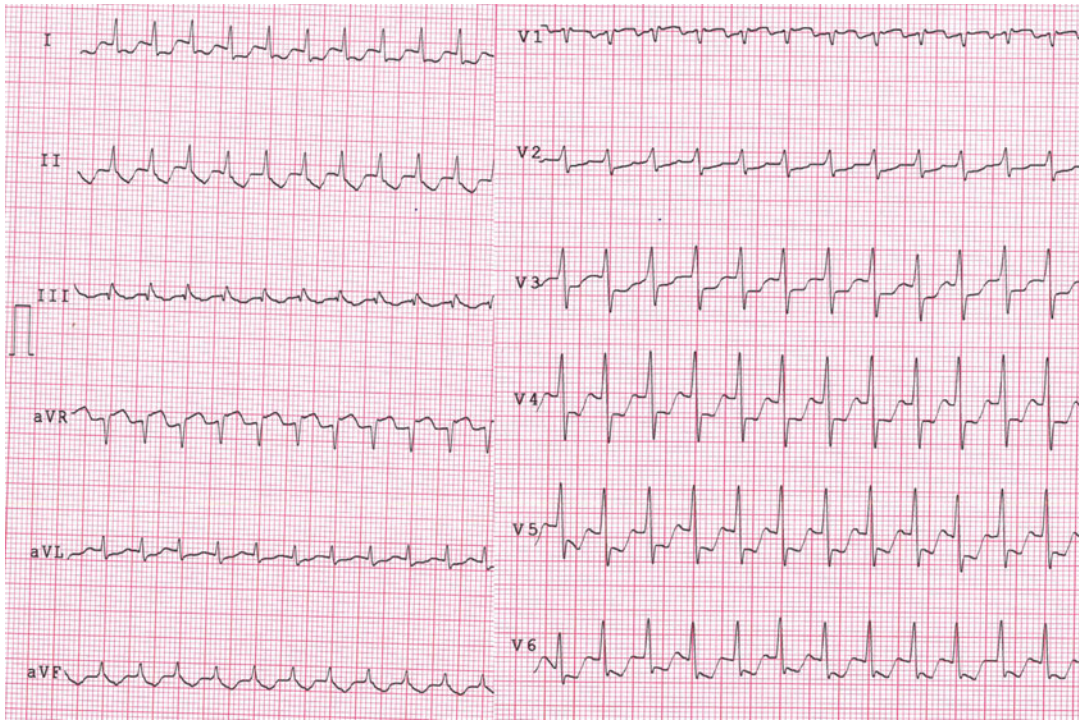


**Fig. 19.1** ECG shows a narrow QRS tachycardia

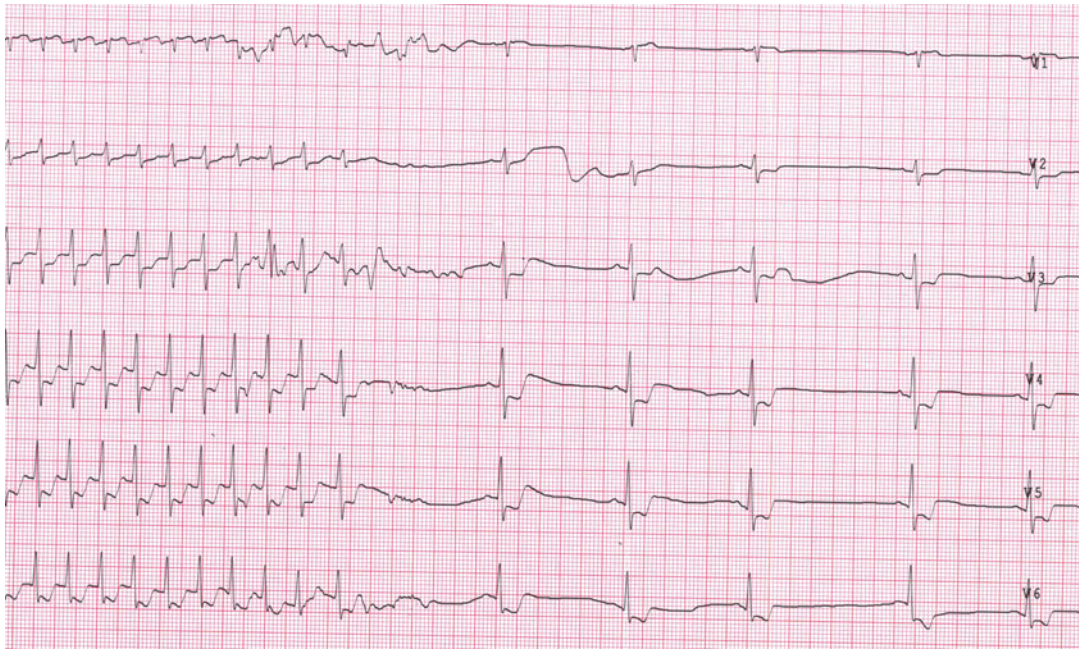


**Fig. 19.2** Tachycardia termination during sinus carotid massage

atrioventricular reciprocating tachycardia, but this is not a definite clue.

The patient was admitted to the cardiology department to perform an invasive electrophysiological investigation with subsequent catheter ablation of the tachycardia.

## Invasive Electrophysiological Investigation (Fig. 19.3)

> […] eccentric type ventriculo-atrial retrograde conduction. Early retrograde conduction in left posterolateral region (CS 5–6). Induction by programmed atrial pacing of an orthodromic atrioventricular reciprocating tachycardia (concealed accessory pathway). During tachycardia a catheter ablation of the posterolateral accessory pathway with termination of tachycardia was performed. […]

The final diagnosis was *atrioventricular reciprocating tachycardia from posterolateral concealed accessory pathway.*

## What Is the Meaning of ST Segment Depression During Supraventricular Paroxysmal Tachycardia? Can It Be a Sign of Atherosclerotic Coronary Disease?

This patient had a high cardiovascular risk profile (type 2 diabetes mellitus, arterial hypertension, dyslipidemia, overweight, past smoking status), and he was symptomatic for chest pain during tachycardia. Echocardiography and a treadmill test were performed.

Echo: Normal atrial size. Normal left ventricle size and systolic function (EF 60 %). Normal right ventricle size and systolic function (TAPSE 19 mm). Normal diastolic function. Mild mitral regurgitation. Mild tricuspid regurgitation with normal pulmonary arterial pressure. No pericardial effusion.

The exercise test was interrupted at the end of Bruce's protocol 5th stage. The patient reached

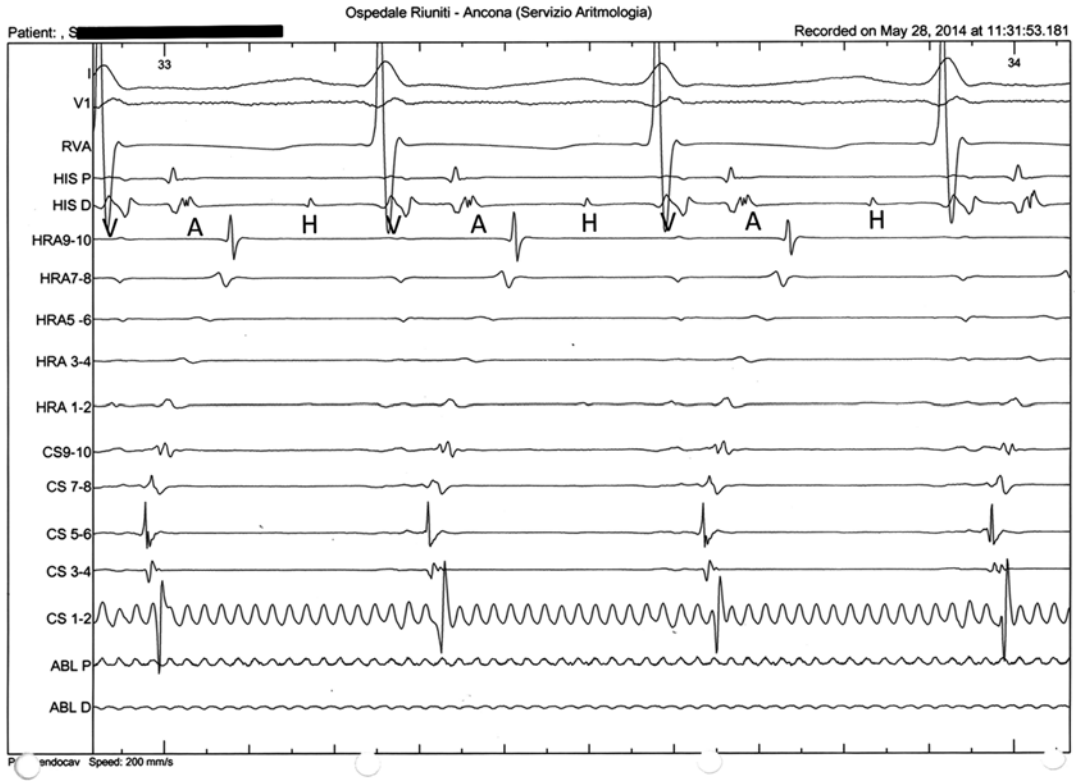


**Fig. 19.3** Electrophysiological study. Endocavity ECG during tachycardia. *A* atrium, *H* His, *V* ventricle

the peak exercise with a heart rate of 154 bpm (96 % of theoretical maximum heart rate) and a systolic blood pressure of 170 mmHg (double product: 26,180). Heart rate and blood pressure showed a normal behavior. There were no symptoms of ischemic ECG changes.

We conclude for nonischemic ST alteration. The abnormal repolarization may be the consequence of a distinct pattern of retrograde atrial activation through accessory pathway. This phenomenon (ST depression >2 mm) is more frequent in AV reentrant tachycardia than in AV nodal reentrant tachycardia. Indeed, in orthodromic AV tachycardia, atrial retrograde activation, as assessed by intracardiac electrograms, occurs during the ST segment on the surface ECG. In contrast, in AV nodal reentrant tachycardia, the retrograde atrial activation is most frequently simultaneous with the QRS complex and therefore does not interfere with repolarization [1–4].

## 19.2 Atrioventricular Nodal Reentrant Tachycardia (AVNRT)

### Definition and Epidemiology

Atrioventricular nodal reentrant tachycardia is the most common form of regular paroxysmal supraventricular tachycardia. Although it was classically described more often in the young and women, it can be detected at any age and sex. The overall prevalence of atrioventricular nodal reentrant tachycardia is 2–3 cases per 1000 persons. Atrioventricular nodal reentrant tachycardia is not usually associated with structural heart disease [5–7].

### Physiopathology and Classification

The reentrant circuit is confined not only in the compact AV node, but the perinodal atrial tissue has an important role too. Atrioventricular nodal reentrant tachycardia is related to the presence of two functionally and anatomically distinct AV nodal pathways (dual AV nodal pathways). The fast pathway, which conducts more rapidly (PR interval 100–150 ms and AH <220 ms) and has a long refractory period, appears to be located near the apex of Koch's triangle. The slow pathway, which conducts more slowly (PR interval >220 ms) and has a short refractory period, extends inferoposterior to the compact AV node tissue and extends along the septal margin of the tricuspid annulus at the level of the coronary sinus. The fast pathway constitutes the normal, physiological, AV conduction axis [6].

AVNRT has been categorized into typical or atypical. This categorization is based on the retrograde limb of the circuit: if it is the fast pathway, it is defined typical; if it is the slow pathway, it is atypical. Typical atrioventricular nodal reentrant tachycardia is the more common type (90 %) and involves the slow pathway for antegrade conduction and the fast pathway for retrograde conduction (slow-fast variant). Atypical atrioventricular nodal reentrant tachycardia is less common (10 %) and includes fast-slow and slow-slow variants [6].

An atrial premature complex that is blocked in the fast pathway and is conducted through the slow pathway generally initiates typical AVNRT. If the fast pathway has recovered excitability, the impulse can conduct retrogradely over the fast pathway, resulting in an atrial echo. If the impulse reenters into the slow pathway, typical AVNRT may be initiated. Seldom it is initiated by a ventricular premature complex (Fig. 19.4).

Atypical AVNRT (fast-slow variant) initiates with a ventricular premature complex that is blocked in the fast pathway. The impulse then conducts to the atrium via the slow pathway and returns to the ventricle via the fast pathway. Seldom an atrial premature complex initiates tachycardia (Fig. 19.5).

In AVNRT, neither the atrium nor the ventricle has a critical role in the reentrant circuit [6].

### Clinical Presentation and Diagnosis

AVNRT usually has a sudden onset and termination, without a warm-up period. Its symptoms

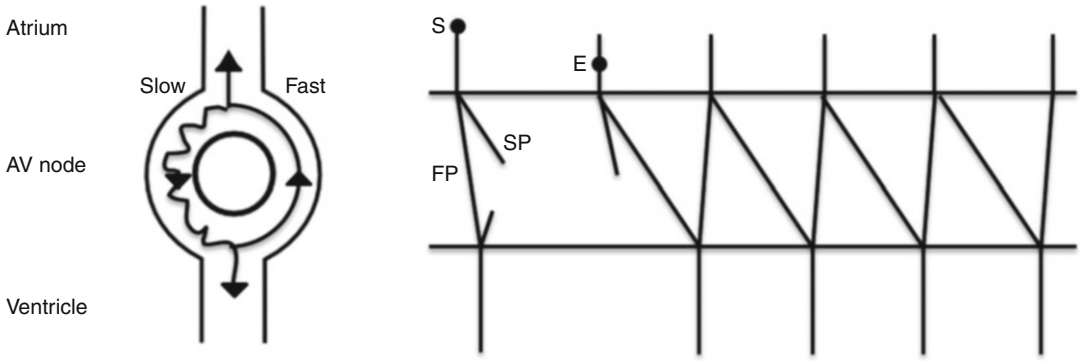


**Fig. 19.4** Typical AVNRT (slow-fast) mechanism. *FP* fast pathway, *SP* slow pathway, *S* sinus beat, *E* extrasystoles

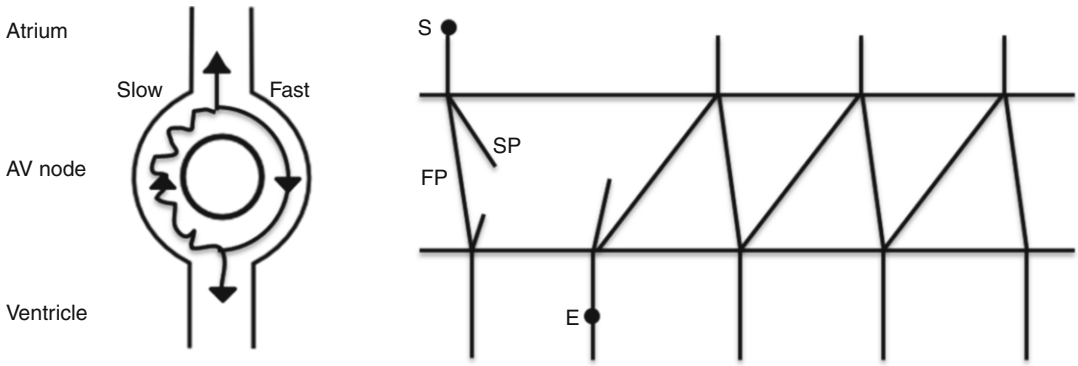


**Fig. 19.5** Atypical AVNRT (fast-slow) mechanism. *FP* fast pathway, *SP* slow pathway, *S* sinus beat, *E* extrasystoles

include palpitations, neck pulsations, dizziness, and in some cases chest pain or dyspnea. Tachycardia can last a few minutes or hours, and the termination can be spontaneous or not. Sometimes precipitating factors (drugs, physical and psychological stress, menstruation, anemia, hyperthyroidism, etc.) can be found [5–7].

A 12-lead ECG is fundamental for the diagnosis. ECG, during AVNRT, shows in most cases a regular narrow QRS complex tachycardia; sometimes the QRS complex can be wide with the typical bundle branch block morphology (LBBB or RBBB). Rate of tachycardia is more often between 140 and 250 per minute. The AV relationship is practically in all cases 1:1. Atrial activation is concentric type because the retroconducted stimulus comes from the AV node, so the retroconducted P wave is narrow and negative in inferior leads. In typical AVNRT, the impulse conducts to the atrium via the fast pathway so the atrium and ventricle are activated simultaneously during tachycardia; the retrograde P wave is either hidden within the QRS or embedded in the terminal portion of the QRS, resulting in RP<PR (usually RP is less or equal to 70 ms) with pseudo-r waves in lead V1 and pseudo-s waves in inferior leads. Comparison with the QRS in sinus rhythm can help in identifying pseudo-r and pseudo-s waves. In atypical AVNRT, the conduction to the atrium is through the slow pathway; the atrium is activated late relative to the ventricle, so the retroconducted P wave is away from the previous QRS and it is near the following QRS (RP>PR) [6].

Invasive electrophysiological investigation (EPS) allows to detect the presence of dual AV nodal pathways, to induce and identify AVNRT (typical or atypical), and to perform catheter ablation of tachycardia. Dual pathway can be demonstrated with delivery of a premature atrial

complex that results in a "jump" in AH conduction curve (defined as an increase >50 ms in AH interval with a 10 ms increase in atrial extrastimulus prematurity). This "jump" is due to antegrade block in the fast pathway with the conduction through the slow pathway. The electrophysiological characteristic of AVNRT is the concentric retrograde activation of the atrium, with a short ventriculoatrial (VA) interval or with a "A on V" (simultaneous activation of the atrium and ventricle) in typical AVNRT or with a long VA time in atypical (fast-slow) AVNRT [6].

## Differential Diagnosis

Surface ECG can make differential diagnosis between AVNRT and the other narrow QRS complex tachycardia during tachycardia, by the induction and termination of tachycardia and by the response to vagal maneuvers [5].

ECG features to assess are shown in Fig. 19.6.

The response to vagal maneuvers or to adenosine can help to distinguish between the different types of supraventricular tachycardia. Gradual slowing then reacceleration of rate is typical of sinus tachycardia or focal atrial tachycardia. Sudden termination is typical of AVNRT and AVRT. Persisting tachycardia with transient high grade of AV block is typical of atrial flutter or atrial tachycardia [5].

Termination of tachycardia is due to block in slow pathway in AVNRT and in AV node in AVRT. Thus, typical AVNRT ends with a retroconducted P wave, atypical AVNRT ends with a QRS complex, and orthodromic AVRT ends with a retroconducted by accessory pathway P wave [5, 6].

## Treatment

Acute treatment: patients with hemodynamic compromise should be electrically cardioverted without delay. Usually, AVNRT does not cause significant hemodynamic compromise. Vagal maneuvers (Valsalva maneuver, dive reflex, carotid sinus massage) can be used to terminate the tachycardia. These maneuvers increase the vagal tone resulting in slow pathway conduction blockage. If vagal maneuvers are ineffective, medications known to prolong refractoriness of the slow pathway of the AVNRT circuit can be used. These agents include adenosine, calcium channel blocker (verapamil or diltiazem), and beta-blockers (metoprolol, atenolol, propranolol, esmolol). Intravenous administration of these drugs should be performed with continuous ECG monitoring. These agents are very effective in terminating the AVNRT [5, 6].

Long-term treatment: in patients with recurrent sustained episodes of AVNRT, we have two strategies of long-term treatment, catheter ablation and oral drug therapy. For oral therapy, the first choice includes AV nodal blocking agents like nondihydropyridine calcium channel blockers (verapamil, diltiazem) or beta-blockers. The use of other antiarrhythmic agents like class IC (flecainide, propafenone) or class III (sotalol, amiodarone) is not common for treating AVNRT

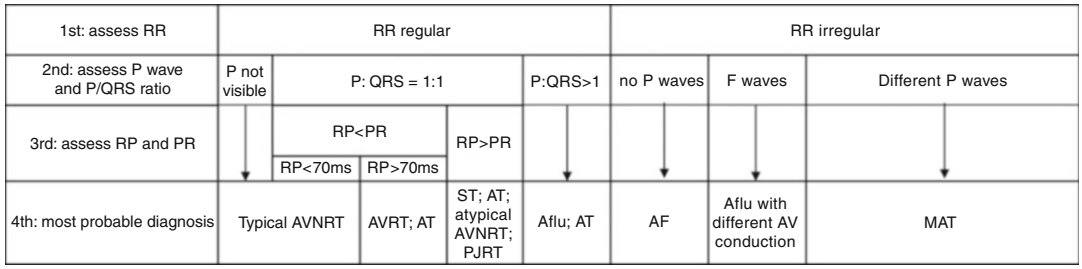


**Fig. 19.6** ECG features to assess. *AVNRT* atrioventricular node reentrant tachycardia, *AVRT* atrioventricular reentrant tachycardia, *AT* atrial tachycardia, *ST* sinus tachycardia, *PJRT* permanent junctional reciprocating tachycardia, *Aflu* atrial flutter, *AF* atrial fibrillation, *MAT* multifocal atrial tachycardia

today; these are used only when the AV nodal blocking agents have failed to prevent recurrence of tachycardia and patient refuses catheter ablation. Radiofrequency catheter ablation is the non-pharmacologic treatment option. Success rates for this technique are approximately 90 %. The major procedural risk is the AV complete block. The ablation of slow pathway, rather than the ablation of the fast pathway, reduces the risk of AV complete block (1 % vs 8 %) and results in the absence of hemodynamic consequences of marked prolongation of PR interval that derives from fast pathway ablation. Therefore, slow pathway ablation represents the first choice today [5].

### Prognosis

Atrioventricular nodal reentrant tachycardia has a good prognosis, and it is not usually associated with structural heart disease. In some patient, AVNRT coexists with atrial fibrillation, and recent reports suggest that it may serve as a trigger for atrial fibrillation and its eliminations can help to treat atrial fibrillation [6].

## 19.3 Ventricular Preexcitation and Atrioventricular Reentrant Tachycardia (AVRT)

### Definition and Epidemiology

Ventricular preexcitation is a congenital alteration of the normal conduction of the electrical impulse in the heart. In this pathology, an accessory pathway is present through which the electrical impulse can activate an area of the myocardium more rapidly bypassing the normal conduction system. In 1930, Louis Wolff, John Parkinson, and Paul Dudley White described a series of young patients who experienced paroxysms of tachycardia and had characteristic abnormalities on electrocardiography (ECG) of ventricular preexcitation. When ventricular preexcitation is associated to symptoms and signs of tachycardias, it is defined as Wolff-Parkinson-White (WPW) syndrome. The expression WPW pattern defines the ventricular preexcitation in the absence of tachycardias' symptoms and signs [5, 7].

The prevalence in the general population is about 1–3 per 1000 people. Men have an higher incidence of ventricular preexcitation than women (M/F = 2.2:1), and there is a higher incidence of multiple accessory pathways in men, too. Some cases of WPW are inherited. Parents who have accessory pathways may pass them onto their children. The incidence of preexcitation in first-degree relatives could be as high as 5.5 per 1000 people. In a recent clinical study, a mutation to the gene that codifies for PRKAG2 (γ-2 regulatory subunit of AMP-activated protein kinase) was identified to be responsible for a syndrome associated with ventricular preexcitation and early onset of atrial fibrillation and conduction disease. About 7–20 % of patients with WPW also have congenital defects within the heart [5–7].

### Accessory Pathways' Anatomy

The anatomical substrate of ventricular preexcitation is an accessory pathway. There are four different types of them. The most common accessory pathway is the bundle of Kent (more of 96 % of cases). This is an atrioventricular bypass: the electrical impulse from the atria directly reaches the ventricles. The bundle of Kent is made of common myocardial cells. They are "sodium-dependent cells," and for this reason, they have a faster conduction of the electrical impulse than the specialized cells of the heart conduction system that are "calcium dependent." The electrical conduction through the bundle of Kent is often frequency-independent and occurs with an "all-or-nothing" mode (non-decremental conduction). It means that an electrical impulse can be conducted through the accessory pathway or blocked, and when conducted, it isn't slowed down. The bundle of Kent can conduct the electrical impulse in different ways: both directions (anterograde and retrograde, about 68 % of cases), only in retrograde direction (from ventricles to atria, about 21 % of cases), or only in anterograde direction (from atria

to ventricles, about 11 % of cases). In addition to the bundle of Kent, there are three other types of accessory pathways grouped under the name of "fibers of Mahaim": atriofascicular tract (from atria to one of the bundle branches, sometimes also called Brechenmacher tract), nodoventricular tract (from AV node to one of the bundle branches or to ventricles), and fasciculoventricular tract (from the bundle of His or one of the bundle branches). Finally, there are some rare cases where multiple accessory pathways can be found [8, 9].

## Ventricular Preexcitation's ECG Pattern

The WPW ECG pattern with full preexcitation contains the following elements:

1. Short PR: a PR interval equal or less than 0.12 s, with a normal P wave. When the electrical activation from the atria reaches the ventricles through the bundle of Kent (by a faster pathway), as a consequence, the PR interval reduces its length.
2. Wide QRS complex: abnormally wide QRS complex with a duration of 0.11 s or more. The QRS complex represents a fusion's beat made of two wavefronts: one from the normal pathway and the other from the accessory. Each of them depolarizes a portion of the ventricular mass.
3. Delta wave: the presence of initial slurring of the QRS complex. This is the most important finding in WPW pattern. Delta wave is in the initial part of the QRS complex and is visible as a very slow initial deflection. The duration of the delta wave varies between 0.02 and 0.07 s. Theoretically, the delta wave should be present in all leads, but it may become isoelectric and be easily overlooked in the leads with the lead axis, which is nearly perpendicular to the initial QRS forces. The end of delta wave joins to the QRS complex (sometimes a little notch can be found between the end of the delta wave and the QRS complex). The entity of the preexcitation depends on the difference of the times of conduction of the accessory and normal pathways. Indeed, a larger preexcitation will be the consequence of a rapid conduction through the accessory pathways. This fact depends on the anatomical position of the accessory pathway: one, near to the sinus-atrial node thus achievable rapidly from the electrical impulse, will show a large preexcitation; another, far from the sinus-atrial node thus achievable slowly from the electrical impulse, will show a little preexcitation.
4. Alterations of repolarization: secondary ST segment and T-wave changes. The altered sequence of ventricular activation in patients with the WPW pattern results in secondary repolarization abnormalities. Most commonly, the direction of the ST segment displacement and the T-wave polarity is opposite to the direction of the delta wave and the major deflection of the QRS complex. In some cases, any abnormalities are seen. The alteration of repolarization can persist after disappearance of preexcitation, too. This is the phenomenon of the "heart electrical memory." They completely regress within several weeks after ablation of the accessory pathway [8, 9] (Fig. 19.7).

## Classification

Ventricular preexcitation can be classified by the direction (anterograde, retrograde, or both) of the accessory pathways and by the presence or absence of signs of WPW on the ECG:

- Manifest preexcitation: the basal ECG shows the WPW pattern. The direction of the conduction through the accessory pathways must be necessarily anterograde, but can be possibly retrograde conduction too.
- Not manifest preexcitation: the basal ECG doesn't show the WPW pattern. The direction of the conduction through the accessory pathways must be necessarily anterograde, but can be possibly retrograde conduction too.
- Concealed preexcitation: the basal ECG doesn't show the WPW pattern. The direction of the conduction through the accessory pathways must be only retrograde [8, 9].

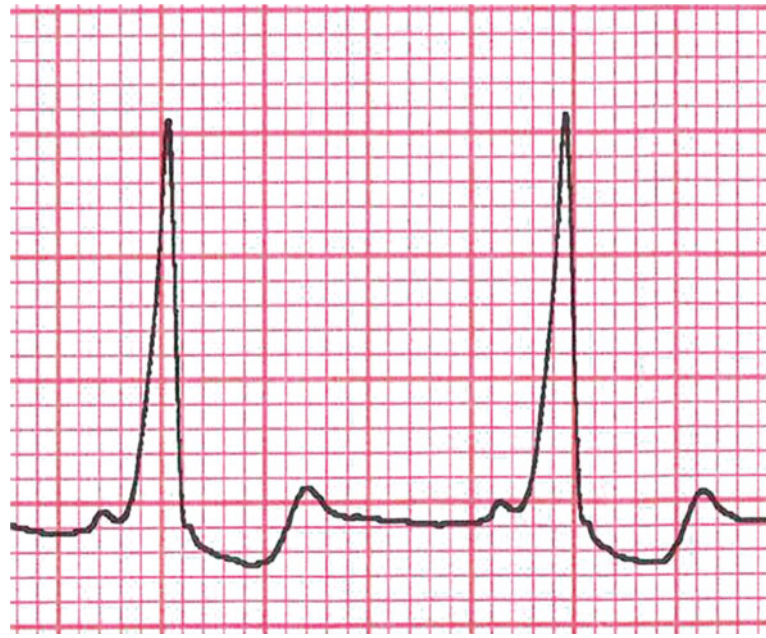

**Fig. 19.7** ECG WPW pattern. Short PR interval, delta wave, wide QRS complex, ST-T alteration

## Localization of Accessory Pathway Using ECG

Sometimes with the standard 12-lead ECG, it is possible to identify the position of the accessory pathway on the patient's heart. An almost correct localization can be reached only when delta wave is visible on the ECG. A perfect localization can be made only with EPS. The possible localization of accessory pathway and their characteristics on the ECG is listed in Table 19.1 (Fig. 19.8a, b).

## Physiopathology of Arrhythmias

Paroxysmal tachycardias are the most important clinical manifestations in patients with WPW syndrome. They were recorded in about 47 % of patients with the WPW pattern. The possible arrhythmias in the patient with an accessory pathway are:

- Orthodromic atrioventricular reentrant tachycardia: the tachycardia is based on an electrical loop made up of the atrium, AV node, His-Purkinje system, and ventricular myocardium in the anterograde direction, returning to the atrium via accessory pathway in the retrograde direction. In this arrhythmia, the delta wave is not visible during the tachycardia, and the duration of QRS complex is normal (narrow). The average heart rate is 140–250 beats/min. Tachycardia is often initiated by premature atrial or ventricular complexes. Diagnostic criteria on the ECG are (1) not sinus P wave (negative in DI), (2) PR longer than RP, (3) P wave after the QRS complex (at least after 0.07 s), (4) occasional occurrence of electrical alternation of different voltages of QRS complex, and (5) P/QRS ratio always 1:1; an AV block during tachycardia rules out the AV reentrant one (Fig. 19.9).
- Antidromic atrioventricular reentrant tachycardia: the tachycardia is based on an electrical loop made up of the atrium, accessory pathway, and ventricular myocardium in the anterograde direction, returning to the atrium via the His-Purkinje system and AV node. In this arrhythmia, the QRS complex is fully preexcited and completely made of delta wave. The duration of the QRS complex is increased (wide complex tachycardia). Tachycardia is often initiated by premature

**Table 19.1** Main accessory pathway ECG features

| | Delta wave's axis | QRS's axis | Delta wave in V1 | Delta wave in V6 | Transition on precordial leads |
|---|---|---|---|---|---|
| Lateral left | +90° to +150° | +90° to +150° | + | ± | V1 |
| Posterolateral | −150° to −90° | −150° to −90° | + | + | V1 |
| Lateral right | −30° to +45° | +45° to −45° | − | + | V3–V4 |
| Anteroseptal | +30° to +90° | 0° to +90° | − | + | V3–V4 |
| Posteroseptal left | −30° to −90° | −30° to −90° | + | + | V1 |
| Posteroseptal right | −30° to −90° | −30° to −90° | − | + | V2 |

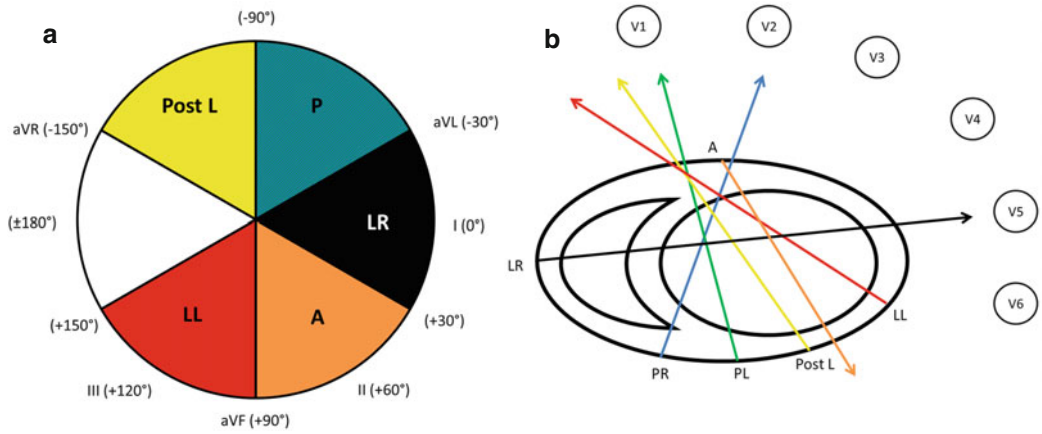


**Fig. 19.8** Accessory pathway axis. Peripheral leads (**a**) and precordial leads (**b**). *LL* lateral left, *PostL* posterolateral, *P* posteroseptal, *PL* posteroseptal left, *PR* posteroseptal right, *LR* lateral right, *A* anteroseptal

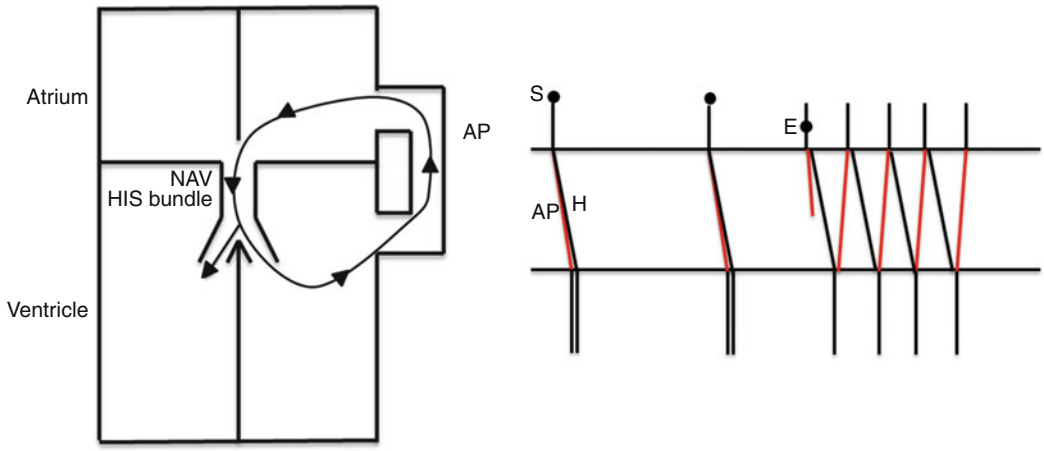


**Fig. 19.9** Orthodromic AVRT mechanism. *NAV* atrioventricular node, *AP* accessory pathway, *S* sinus beat, *E* extrasystoles, *H* His bundle

atrial or ventricular complexes. Diagnostic criteria on the ECG are (1) wide QRS regular tachycardia; (2) QRS morphology identical to that of the fully preexcited complex; (3) not sinus P wave, when visible; and (4) P/QRS ratio always 1:1; an AV block during tachycardia rules out the AV reentrant one (Fig. 19.10).

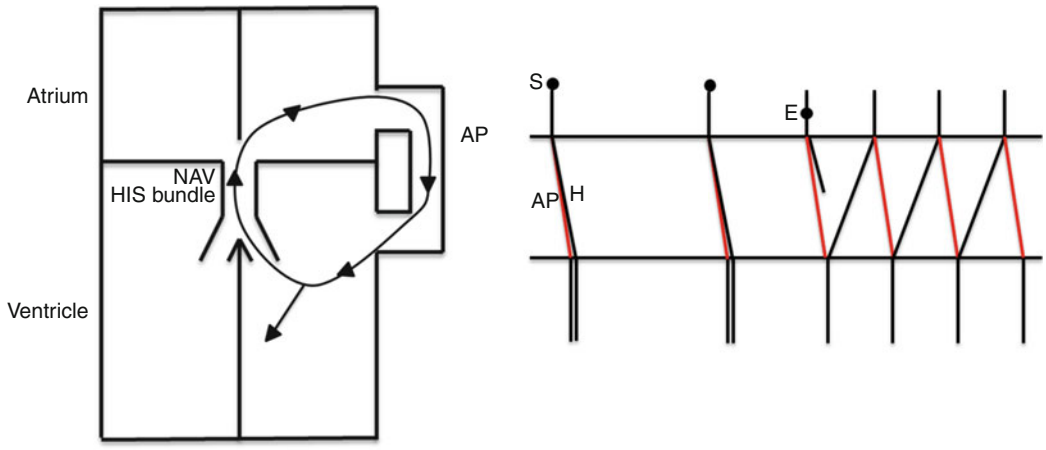


**Fig. 19.10** Antidromic AVRT mechanism. *NAV* atrioventricular node, *AP* accessory pathway, *S* sinus beat, *E* extrasystoles, *H* His bundle

- Preexcited atrial fibrillation/atrial flutter: atrial fibrillation and flutter are less common than AV reentrant tachycardia in the WPW syndrome. Atrial impulses during atrial fibrillation and atrial flutter are conducted to the ventricles through the accessory pathway. The result is a wide complex tachycardia with irregular RR in the case of atrial fibrillation and regular in the case of atrial flutter. This tachycardia may be as rapid as 220–360 beats/min. This high rate is possible in the presence of a short, effective refractory period of the accessory bundle. Because of the rapid ventricular response during atrial fibrillation, patients with the WPW syndrome may develop ventricular fibrillation that results in sudden death. In the case of atrial flutter, the AV conduction ratio may be 2:1 or 1:1 (Fig. 19.11).
- Permanent junctional reciprocating tachycardia (Coumel type): the tachycardia is based on an electrical loop made up of the atrium, AV node, His-Purkinje system, and ventricular myocardium in the anterograde direction, returning to the atrium via accessory pathway in the retrograde direction. In this case, the accessory pathway has retrograde decremental conduction properties. The accessory pathway is located near to the His bundle (para-Hisian). It is usually a long-lasting tachycardia [5, 6, 8, 9].

## Clinical Presentation

Patients with WPW can remain completely asymptomatic, and the preexcitation can be discovered with an ECG made for other reason. Usually, patients become symptomatic when paroxysmal arrhythmias occur. In this case, common symptoms are palpitations, fatigue, lightheadedness, chest discomfort, dyspnea, presyncope, or syncope.

Arrhythmic episodes usually have sudden onset and termination. The episode may have different duration, from few minutes to several hours. It is important to investigate all the symptoms, number of episodes, duration, frequency, mode of onset, and possible triggers [5, 7, 10].

## Diagnosis and Differential Diagnosis

The diagnosis can be suggested in the presence of symptoms of the paroxysmal arrhythmias referred by the patient (previously discussed). The 12-element ECG is crucial for the diagnosis. Indeed, in case of preexcitation, the ECG shows the typical features: short PR, wide QRS complex, delta wave, and alterations of repolarization. In the case of strong clinical suspect, an EPS is recommended in order to identify the accessory pathway and/or to evocate possible arrhyth-

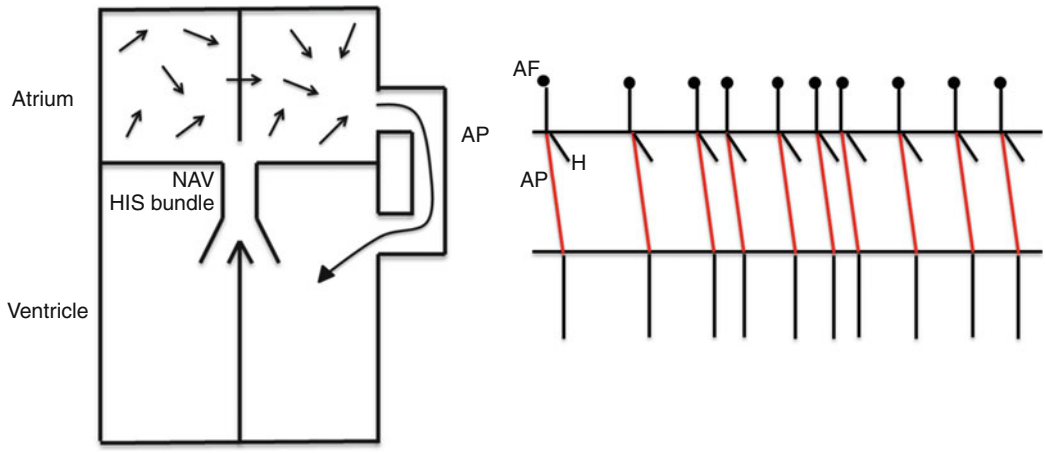


**Fig. 19.11** Preexcited atrial fibrillation mechanism. *NAV* atrioventricular node, *AP* accessory pathway, *AF* atrial fibrillation, *H* His bundle

mias. Invasive EPS allows overstimulating the atria to identify if there is another way (in addition to the AV node) for the electrical impulse to reach the ventricles and overstimulating the ventricles to identify if there is another way to reach the atria. In addition, studying the first activated and the last activated area is possible to localize exactly the position of the accessory pathway. In some cases, it is possible to induce tachycardia, too [5–7, 10].

The differential diagnosis has already been discussed in the AVNRT paragraph.

## Treatment

Acute treatment: if the patient, during the arrhythmia, is hemodynamically unstable, an immediate electrical cardioversion is mandatory. In the case of a regular narrow QRS complex tachycardia, in a hemodynamically stable patient, it is important to do vagal maneuvers (Valsalva, carotid sinus massage, and facial immersion) to terminate the arrhythmia. If these fail, IV antiarrhythmic drugs should be administered. Adenosine or nondihydropyridine calcium channel antagonists are the drugs of choice (the first one should be avoided in patients with severe bronchial asthma). Second-choice drugs are IV calcium channel blockers or beta-blockers. An ECG should be recorded during the maneuvers for the termination of the tachycardia. In fact a P wave at the end of the tachycardia is suggestive for AVRT or AVNRT. Continuation of tachycardia with AV block rules out AVRT. In case all the maneuvers for the termination of the tachycardia are not effective, electrical cardioversion should be considered. In patients with a regular wide QRS complex tachycardia, if a differential diagnosis of ventricular tachycardia (VT) is unclear, the arrhythmia should be treated as VT. In case of preexcited tachycardias, AV nodal blocking agents should be used cautiously. It is preferable to treat preexcited AF with IV ibutilide, flecainide, or procainamide.

Chronic treatment: in patients with WPW syndrome, the first-line therapy is catheter ablation of the accessory pathway. This approach is recommended in patients with preexcitation and symptomatic arrhythmias, mostly in the case of preexcited tachycardias or when the arrhythmic episodes are frequent or poorly tolerated. In these patients, when catheter ablation is not possible, a prophylactic antiarrhythmic therapy should be considered. The advised drugs are flecainide or propafenone or sotalol or amiodarone. AV-blockers (nondihydropyridine calcium channel antagonists or beta-blockers) should not be used as single therapy in patients with preexcited tachycardia (especially AF) because there is concrete risk to favor the conduction through the accessory pathway. In case of rare episodes of AVRT without preexcitation or in asymptomatic

patients with preexcitation, catheter ablation should be considered. In such cases, patient's choice should be taken into consideration. In this case, a pharmacological – pill in the pocket – therapy (taking an antiarrhythmic drug only at the onset of a tachycardia episode) could be made with verapamil, diltiazem, or beta-blocker. Sometimes none therapy could be another strategy, too [5, 6, 10].

## Prognosis

The risk of sudden cardiac death due to the presence of accessory pathway has been estimated to range from 0.15 to 0.39 % over 3–10 years follow-up. If the risk of sudden cardiac death after accessory pathway ablation is the same or less is debated yet [5, 10].

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