22 Arrhythmias

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22

Origin of Cardiac Arrythmia

The normal heartbeat is initiated in the sinus node (impulse formation) and travels over the conduction system (atrium, atrio-ventricular [AV] node, His–Purkinje system) to the ventricles (impulse conduction). *Sinus rhythm* is defined by presence in the electrocardiogram (ECG) of a sinus P wave, which is positive in lead II, and is followed by a QRS complex (except in case of an AV block). *Ectopic rhythms* are those having an origin outside of the sinus node. Normal sinus rhythm is not completely regular. Physiologic variations in heart rate can be associated with irregular respiration or with vagotonic/sympathicotonic stimulation.

Bradyarrhythmias arise from an absence of the normal impulse formation (e. g., sinus node arrest) or from a blocking of the normal impulse propagation (e. g., AV block). *Tachyarrhythmias* occur when an abnormal impulse is formed (focal arrhythmias) or an abnormal conduction is present (reentrant tachycardias), or from a combination of both (i. e., atrial fibrillation). Arrhythmias of all types and frequencies can result (i. e., from slow to fast and from regular to completely irregular). Many arrhythmias occur without an identifiable cause (primary arrhythmias). In secondary arrhythmias an underlying cardiac disease or systemic disease is arrhythmogenic.

22.1 Differential Diagnosis of Arrhythmias

Medical History

A detailed medical history is vital to any diagnosis, including that of arrhythmias.

Family History. The family history can reveal inherited causes of sudden cardiac death (long QT syndrome, Brugada syndrome, arrhythmogenic right ventricular cardiomyopathy, hypertrophic obstructive cardiomyopathy, etc.). If the cause of a sudden death of any family member is unclear, review of the ECG or medical records can be extremely helpful to identify the nature of an inherited disorder.

Personal History. In patients with structural heart disease (e. g., myocardial infarction, cardiomyopathy), cardiac arrhythmias and syncope have a serious impact on prognosis. On the other hand, in structurally normal hearts most arrhythmias are not associated with a worse prognosis.

> The prognosis of arrhythmias depends on the presence or absence of structural heart disease. In normal hearts arrhythmias are not dangerous, but in the presence of structural heart disease they can be life-threatening.

The *age* at first presentation of the arrhythmia can also provide clues to the etiology. Reentrant tachycardia over an accessory pathway is often symptomatic in infancy and adolescence, whereas atrial tachycardias often arise in the elderly and AV nodal reentrant tachycardia can become manifest at any age.

Paroxysmal supraventricular tachycardias have a *sudden onset* and a *sudden end*, like a light switch, and are often accompanied by diuresis provoked by release of atrial natriuretic peptide (ANP). In focal tachycardias (i. e., in atrial tachycardia) a gradual acceleration (warming up) and deceleration (cooling down) of the arrhythmia can be observed.

If the patient experiences *syncope*, especially in the presence of structural heart disease, a potentially dangerous condition must be assumed and hospitalization is required for evaluation (see Chapter 31). Caution: *epilepsy* can also be a secondary phenomenon caused by cardiac arrhythmia (asystole or ventricular fibrillation).

Clinical Examination

Central and Peripheral Pulse. In the arrhythmic patient counting the pulse (centrally with a stethoscope and peripherally) can provide useful information of hemodynamic relevance (e. g., pulse deficit) and about the synchronicity of atrial and ventricular contraction. If the atrium and ventricle are not synchronized, as during ventricular tachycardia, the first heart sound varies in intensity and character. The clinical examination can also uncover signs of underlying valvular cardiac disease, which can be directly relevant to the arrhythmia. A regular resting pulse with single pauses is a sign of extrasystoles leading to a compensatory pause, which are generally insignificant.

If an absolute pulse arrhythmia is present, together with a pulse deficit (i. e., a higher pulse at heart auscultation than at palpation of the radial artery), then the diagnosis of atrial fibrillation can be made even without the ECG.

The heart rate itself does not provide any information about the type of the arrhythmia and its criticality.

Although ventricular tachycardias are often fast and can lead to syncope, particularly in patients with structural heart disease, they can also be slower and hemodynamically well tolerated over hours and days. In every case, however, they require urgent treatment and monitoring. Conversely, supraventricular tachycardias, especially in patients with a normal heart, are usually better tolerated and may be terminated by the patient with coughing, pressure, or carotid sinus massage.

Carotid Sinus Massage. Carotid sinus massage is an integral component of the physical examination and should always be performed with ECG monitoring. Gentle pressure on the carotid sinus, in the middle of the neck, provokes vagotonic stimulation, which slows impulse frequency from the sinus and AV node. This has diagnostic significance and is also a therapeutic maneuver, which can stop an arrythmia (e. g., during supraventricular tachycardia where the AV node is part of the reentrant circuit). Focal arrhythmias can be slowed (atrial tachycardia) or flutter waves unmasked by slowing conduction over the AV node.

Carotid sinus massage (Tab. 22.**1**) should also be performed in syncopal patients to detect bradycardia-induced AV block, a dangerous condition with a poor prognosis (warranting immediate pacemaker implantation; see Chapter 31).

Table 22.**1** Procedure for carotid sinus massage

- Auscultation on both carotids (a murmur or sign of stenosis is a contraindication for CSM)
- Gentle pressure on one side, the ipsilateral pulse of the temoral artery should be palpable
- Continuous ECG monitoring
- Duration of massage approximately 5 s
- Emergency treatment for hypotension, bradycardia, and cardiac arrest available

Electrocardiogram (ECG)

The systematic analysis of the ECG begins with the analysis of the frequency of atrial (P waves) and ventricular (QRS complex) excitations and their relation. Then the size, width, and axis of P waves and QRS complexes should be analyzed. The repolarization phase (ST and T segments) and the presence of U waves can provide important information on the origin of arrhythmias. The duration of the longest QT interval should also be measured and corrected for the heart rate, since this can also play a major role in arrhythmias. In addition to the diagnosis of arrhythmias, the ECG can give important information about previous myocardial infarction, ischemia, pulmonary or systemic hypertension, pericarditis, cardiomyopathy, and congenital anomalies.

Additional Tools for the Diagnosis of Arrhythmias

Stress ECG. A continuous ECG recording during physical exercise (treadmill, bicycle) can provoke stress-induced arrhythmias or provide important information about increased sinus node rates (chronotropic competence) and the conduction properties of accessory pathways (delta waves in Wolff-Parkinson-White [WPW] syndrome).

Holter ECG. Continuous ECG monitoring over 24 or 48 hours can diagnose intermittent (paroxysmal) arrhythmias, provided that the symptomatic arrhythmia actually appears again during this time. Checking the correlation between the ECG and the symptoms logged is also important. In addition, the Holter ECG can give information about the function of the sinus node, the heart rate variability, and detect pauses which can be important for the patient's prognosis.

Event Recorder. If the arrhythmia occurs rarely and is unlikely to be captured by a Holter ECG, then a longer recording using an event recorder is preferable. These recorders can either function as a continuous loop recorder or can be held over the heart in case of symptoms. If potentially dangerous arrhythmias must be detected (i. e., in case of syncope), a loop recorder can be implanted subcutaneously for several months. This can then record the ECG automatically above and below programmable pulse limits (i. e., below 40 beats/min and above 180 beats/min). In addition, patient-initiated recordings are also possible (e. g., a normal ECG during suspicious symptoms points to an extracardiac origin).

Electrophysiologic Investigation. The electrophysiologic study is an invasive procedure for diagnosis of unclear arrhythmias and patients with syncope. It is also employed therapeutically for radiofrequency ablation treatment of supraventricular and ventricular arrhythmias (i. e., thermal ablation of the responsible focus or reentrant circuit). However, documentation of the arrhythmia by the noninvasive methods described above should always be carried out beforehand to match induced arrhythmias with clinical ECG tracings.

Further Diagnostic Tools. A tilt table, which suddenly shifts the patient from a recumbent to an upright position, can provoke a neurocardiogenic syncope, which if correlated with the clinical symptoms, has diagnostic significance. However, unspecific findings are frequent. Further analysis of ECG intervals can noninvasively identify patient groups at increased risk for arrhythmias or sudden death (e. g., heart rate variability as a measure of autonomic innervation, signal-averaged ECG, T-wave alternans, etc.). Unfortunately, none of these tests has proved to be of sufficient sensitivity and specificity in unselected patients to justify routine clinical use.

22.2 Bradyarrhythmias

Bradycardias are frequent and can be as low as 30 beats/min in healthy individuals during the night or in athletes, even during the day at rest. Treatment of bradycardia is indicated only in case of correlating symptoms.

Sinus Node Dysfunction

Particularly in elderly patients, the function of the sinus node can be impaired (sick sinus syndrome). This is usually expressed by the absence of heart rate increase as a response to physical exercise (chronotropic incompetence), sinus node pauses of over 3 seconds, or a marked increase in beat to beat variation (over 15% of cycle length). If an inappropriate sinus or atrial tachycardia is found in the same patient, then the diagnosis of a *brady–tachycardia syndrome* can be made.

The differentiation of the exact nature of the *sinus node dysfunction* (i. e., conduction block [*sinoatrial block*] versus absent impulse formation [*sinus arrest*]) is of little prognostic or therapeutic interest. More important is the correlation of symptoms with the ECG findings, because only the symptoms (e. g., syncope, exertional dyspnea, fainting) justify invasive therapies (e. g., pacemaker implantation).

A *secondary sinus node dysfunction* can result from hypothyroidism, drugs, electrolyte imbalances, sleep apnea syndrome, etc. (Tab. 22.**2**).

In about 20% of patients with sinus node dysfunction, concomitant AV node dysfunction can be observed, since the causes are similar (Tab. 22.**2**). A transient suppression of sinus node function can be observed after conversion of rapid atrial arrhythmias (e. g., atrial fibrillation or flutter [prolonged sinus node recovery time]).

Treatment of sinus node dysfunction is indicated only when corresponding symptoms are present.

Table 22.**2** Causes of bradyarrhythmias

- Drugs (beta-blockers, digoxin, calcium antagonists, antiarrhythmics, neuroleptics, sedatives, narcotics, etc.)
- Vagal tone (e. g., valsalva, carotid massage, athletes)
- Electrolyte imbalances (e. g., hyperkalemia)
- Coronary artery diseases (with involvement of AV conduction system)
- Myocarditis (e. g., rheumatic fever)
- Infectious endocarditis
- Other cardiac infections (borreliosis, tuberculosis, Chagas disease, toxoplasmosis)
- Systemic disorders (amyloidosis, sarcoidosis, hemochromatosis)
- Mechanical (cardiac surgery, trauma)
- Metastatic cancer
- Degenerative changes
- Congenital heart block
- Neuromuscular disease

Atrioventricular Block

Atrioventricular conduction block is classified according to three degrees. In first degree AV block conduction is slowed, but present for every heartbeat. In second degree AV block some P waves are conducted, whereas others are blocked. In third degree AV block all P waves are blocked.

First Degree AV Block

In first degree AV block the PQ interval is prolonged over 200 ms. Intermittent first degree AV block can be observed with increased vagal tone, in advanced age, and with drugs. If first degree AV block persists during sympathetic activation (exercise), then pathologic AV node function or a congenital anomaly must be suspected. First degree AV block, especially with coincident bundle branch block, can be a sign of severe conduction system disease below the AV node. If the PQ interval becomes very long (up to 300 ms) the atrial systole can occur during ventricular systole, against closed AV valves, leading to palpitations, fainting, and limited exercise tolerance.

Second Degree AV Block

In second degree AV block fewer QRS complexes than P waves are seen, but an association between the two is maintained. Depending upon the origin, and particularly for prognostic reasons, second degree AV block is classified into two types:

Type 1. In second degree AV block type 1 (*Wenckebach type*) there is a progressive prolongation of PQ intervals, until a single P wave block (Fig. 22.**1**). The diagnostic criterion is the shortened PQ interval after the blocked P wave. The progressive reduction of the RR intervals, which is known as Wenckebach periodicity, results from the slowed progression of the PQ prolongation. The place of origin is generally the AV node, which has a conduction delay due to increased vagal tone. Progression to a higher degree AV block is rare.

Second degree AV block type 1 (Wenckebach) is defined by a shorter PQ interval after the pause than before the pause. In half of the patients progressive PQ prolongation can also be observed, leading to Wenckebach periodicity of RR intervals. The prognosis is usually good.

Fig. 22.**1** Second degree AV block type 1 (Wenkebach). Progressive PQ prolongation until AV block with subsequent shortening of PQ interval.

Type 2. In second degree AV block type 2 (Mobitz) P waves are blocked in a more or less fixed ratio (2:1, 3:1, or 4:1) *without prolongation* of the PQ interval and *without shortening* of the PQ interval after the pause. As the origin of type 2 Mobitz block is below the AV node, usually within the His–Purkinje system, progression to complete AV block is frequent. The PQ intervals are usually short because of increased sympathetic tone and show no variation at all.

Differentiation. In the case of second degree AV block with 2:1 conduction, a differential diagnosis of the two types can be difficult and usually becomes possible only when the beginning and end of the block, as well as further signs are taken into consideration. In Wenckebach type 1 nodal block there are frequently signs of increased vagal activity (sinus bradycardia), while in Mobitz type 2 infranodal block there is, on the contrary, increased sympathetic tone (sinus tachycardia).

Third Degree AV Block

Third degree AV block is defined by a lack of conduction between the atria and the ventricles. Fortunately, in most cases an escape rhythm below the AV node takes over. Otherwise, the patient develops syncope due to asystole (Adams–Stokes). Escape rhythms are usually regular and are narrow if they arise high in the His bundle, wider if they arise more distally in the Purkinje system. If they have typical left bundle branch block morphology, they arise from the right bundle and vice versa.

Caution: AV blocks are also associated with atrial flutter and atrial fibrillation. In the case of atrial flutter, a healthy AV node conducts the flutter waves in the ratio of 2:1, which results in a typical ventricular rate of around 130–150 beats/min. Slower rates at 3:1 or 4:1 conduction are usually the result of slowed AV node conduction, caused by drugs or propagation system disorders. If regular ventricular rhythm suddenly occurs, instead of absolute arrhythmia with atrial fibrillation, then a total AV block must be assumed.

> Regular ventricular rhythm in atrial fibrillation is a sign of total AV block with escape rhythm!

High grade AV block (intermittent third and second degree block) is most often idiopathic or caused by degeneration of the normal conduction system. Secondary causes should be investigated in line with the clinical findings (see Tab. 22.**2**).

Differential Diagnosis of Vagotonic (Functional) Versus Organic AV Block

Vagotonic AV Block. Typically in patients with a healthy heart, increased vagal tone can produce a bradycardia and even pauses of several seconds in the sinus node, as well as in the AV node. Since the vagal influence is intensified by the sympathetic nervous system, such bouts of syncope primarily occur during emotional excitement, pain, or intense heat. In the ECG the vagal influence can be detected (i. e., *simultaneous slowing of the sinus nodes and* AV blocking [first degree or second degree Wenckebach type 1]). This is the key to the diagnosis in the ECG (Fig. 22.**2**). After the pause, reflex tachycardias can appear. Classical symptoms are a protracted hypotension after the event, which leads to feeling unwell, pallor, sweating, and sickness. These episodes are distressing but not dangerous.

Organic AV Block. In contrast to organic AV block, the pauses induced by a pathologic state (i. e., a disease of the propagation system) are followed by a strongly increased sympathetic tone. For instance, organic AV block due to hypotension leads to *stimulation of the sympathetic system* and a *simultaneous sinus tachycardia* (reduction of the PP interval during the AV block; Fig. 22.**3**). After termination of the AV block, the patients have high to normal blood pressure and feel well (Tab. 22.**3**).

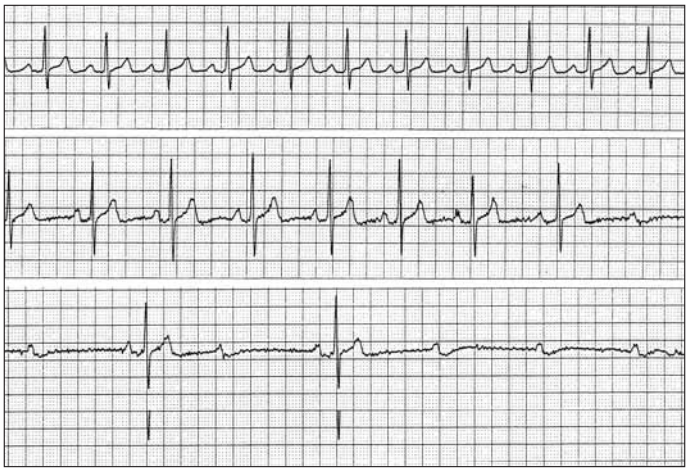


Fig. 22.**2** Vagotonic AV block during venous puncture, with syncope. Initially, there is sinus tachycardia (emotional stress), which slows down (vagotonic due to pain from puncture) and is followed by third degree AV block. The sinus rate during AV block is slower than before the block.

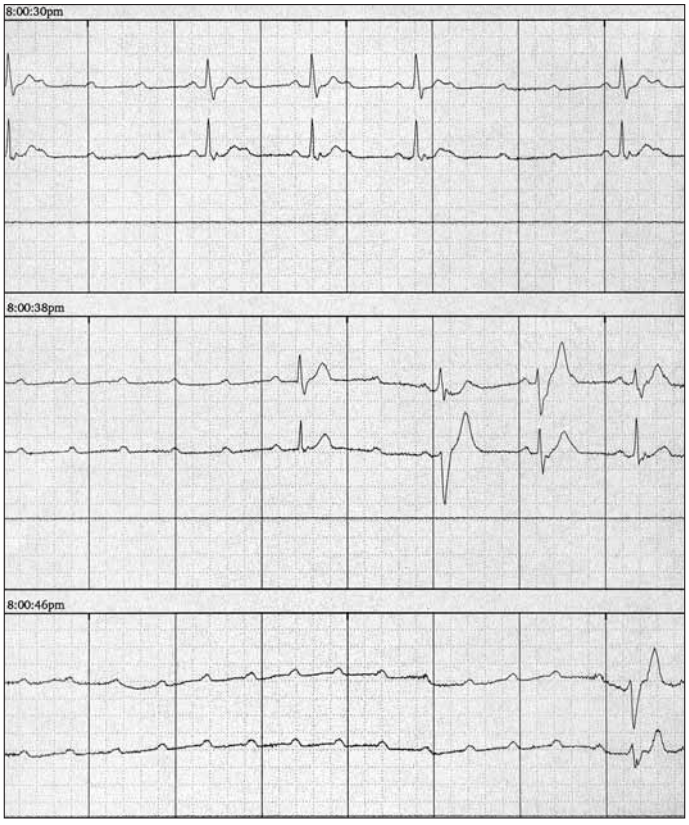


Fig. 22.**3** Organic AV block. Initially, 2:1 and 4:1 followed by third degree AV block. A vagotonic block can be excluded because of the high sinus rate (150 beats/min) during the block.

Table 22.**3** Differential diagnosis of functional (vagotonic) versus organic AV Block

	Vagotonic AV block	Organic AV block
Location of block	AV node	His–Purkinje system
Most frequent etiology	Vagotonic	Organic (ischemia, degenerative)
Prognosis	Good	Worse (progression frequent)
Sinus rate during block	Lower	Higher than before/after
Conduction at increased heart rate	Better	Worse
Conduction during carotid massage	Worse	Better
Retrograde conduction (VA)	Never	May be present
Variation of PQ intervals	If > 100 ms	If < 50 ms

Bradyarrhythmias with Acute Myocardial Infarction

Bradyarrhythmias (sinus bradycardia up to third degree AV block) can be provoked by vagotonia after morphine administration or pain, and are common during reperfusion, especially in inferior infarctions. Rarely, acute ischemia is responsible for sinus node dysfunction. In this event a very proximal occlusion of the right or circumflex coronary artery is present. The first branches of the left anterior descending (LAD) coronary artery supply the His bundle and the right and left Tawara bundle. Therefore, a *newly acquired bundle branch block* (suggested by a septal Q wave) indicates a very proximal LAD occlusion with high risk of progression to complete AV block.

22.3 Junctional Rhythms

The AV junction can serve as a secondary pacemaker in case of sinus arrest or can generate accelerated junctional rhythms faster than the sinus rate. The latter is mostly due to abnormal impulse formation (Digoxin, excessive catecholamines, fibrinolysis and reperfusion, cardiac surgery). Retrograde P waves are grounds to assume a junctional rhythm. A gradual deceleration is observed after carotid sinus massage and an acceleration is possible (Fig. 22.**4**). Accelerated junctional rhythms may also be observed, particularly in adolescents. However, the diagnosis can be confirmed only by electrophysiologic studies. Retrograde P waves are typically narrow and negative in inferior leads (II, III, aVF).

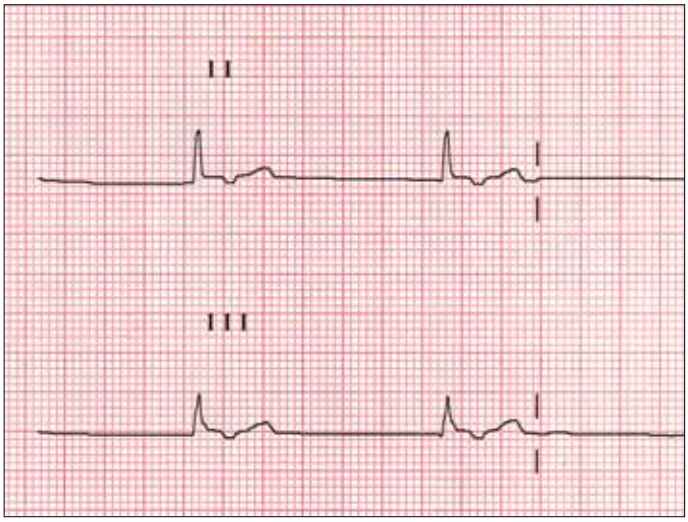


Fig. 22.**4** Junctional rhythm with retrograde P waves (negative, narrow P waves indicate atrial activation from the lower septum).

22.4 Extrasystoles

Extrasystoles are single beats caused by abnormal impulse generation anywhere in the heart (atria, ventricles, conduction system). Extrasystoles are frequently observed in healthy hearts. They can occur as single beats, alternating with one normal sinus beat (bigeminus) or two sinus beats (trigeminus) or can occur in series of two (couplet) or three (triplet) beats. More than three ventricular extrasystoles in a row define nonsustained ventricular tachycardia. In most patients extrasystoles are asymptomatic. However rarely, they can evoke disturbing symptoms such as palpitations, panic attacks, dyspnea, or hyperventilation.

Supraventricular Extrasystoles

Supraventricular extrasystoles originate in the atrium, in the atrial myocardium, or in the AV node. They manifest as an early P wave, which has a morphology corresponding to the origin of the focus (negative if lower atrial focus, biphasic if left atrial, etc.). The QRS complex can be normal or widened because of aberrancy. Frequently, the conduction of the supraventricular extrasystoles is blocked, so that a pause occurs, which is further extended by the subsequent sinus

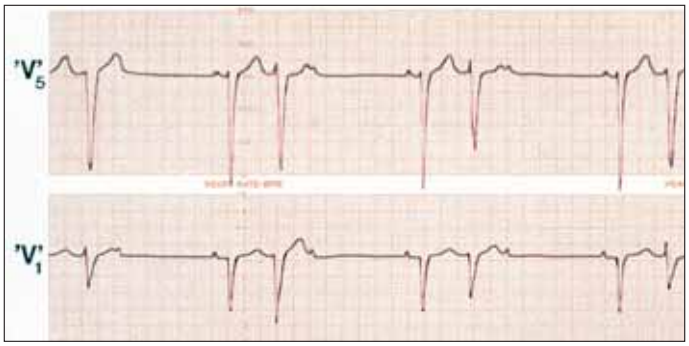


Fig. 22.**5** Supraventricular extrasystoles with physiologic block.

Fig. 22.**6** Ventricular extrasystoles in a Holter ECG (lead V_5). Isolated monomorphic ventricular extrasystoles in rows 1–7; couplets in row 7 and triplets in row 6.

pause (Fig. 22.**5**). Supraventricular extrasystoles have no prognostic significance but can induce sustained arrhythmias.

The most frequent cause of pauses is supraventricular extrasystoles with physiologic block. The symptoms are caused by the pause rather than the extrasystole.

Ventricular Extrasystoles

Ventricular extrasystoles can be differentiated from supraventricular extrasystoles by the following (Fig. 22.**6**):

- no preceding P wave (no constant PQ interval)
- different QRS morphology, width, and axis
- altered repolarization (T wave opposite to QRS vector)
- compensatory pause.

Single ventricular extrasystoles have no proven prognostic value, especially in the absence of structural heart disease. More than three consecutive ventricular extrasystoles are considered to be ventricular tachycardia, which represents a risk factor in patients with coronary heart disease or hypertrophic cardiomyopathy.

22.5 Tachyarrhythmias

Tachyarrhythmias are classified as *supraventricular* or *ventricular*, depending on the origin of the arrhythmia (Tab. 22.**4**). The symptoms in all tachyarrhythmias are similar (e. g., light-headedness, palpitation, presyncope, and syncope). If a tachyarrhythmia is hemodynamically stable, it is usually supraventricular, but ventricular tachyarrhythmias may also present without hemodynamic compromise. Termination of a tachyarrhythmia with vagal maneuvers (such as drinking cold water, carotid sinus massage, Valsalva maneuver) suggests involvement of the AV node in the tachycardia (AV nodal reentrant tachycardia, AV reentrant tachycardia).

The medical history is particularly useful for the differentiation of supraventricular and ventricular tachyarrhythmias. History of myocardial infarction increases the likelihood of a ventricular tachycardia. The Q waves may also be visible during the tachycardia. On the other hand, if a patient has a very long history of palpitations, the tachycardia is most likely of supraventricular origin.

In daily practice, an initial differentiation is based on the duration of the QRS complex during the tachycardia.

Table 22.**4** Clinical differentiation of tachycardias

	Paroxysmal supraventricular tachycardia	**Ventricular tachycardia (VT)**
Symptoms	Palpitations Diaphoresis Nausea Sweating Dyspnea Diuresis Rarely syncope	Palpitations Diaphoresis Nausea Sweating Dyspnea – Syncope common
Structural heart disease	Rare	Common
Hereditary causes	Very rare	Occasional
Adenosine and vagal stimulation	Termination common	Termination seldom
QRS vector	Similar to that seen in sinus rhythm	Different from sinus rhythm
Q waves during tachycardia	Narrow	often > 140 ms
QRS width	Sometimes aberrant	Very rarely narrow (septal VT)
Fusion beats	never	If present, diagnostic
AV synchrony	almost always	facultative

Narrow-Complex Tachycardia

In narrow-complex tachycardia the QRS width is, by definition, less than 120 ms and these arrhythmias are, with rare exceptions, almost always of supraventricular origin (Fig. 22.**7**).

Sinus Tachycardia

In sinus tachycardia the morphology and axis of the P wave are similar to that observed during sinus rhythm. The rhythm typically speeds up and slows down gradually. Sinus tachycardia is a common finding and is typically *secondary* to other causes, such as heart failure, pain, pulmonary emboli, as well as central nervous system and other disturbances, which are associated with increased adrenergic stimulation. In addition, some *drugs* (e. g., antihypertensives) or withdrawal of some drugs (e. g., beta-blockers) may cause sinus tachycardia. Rarely, the sinus node may show a hypersensitive response to endogenous catecholamines, causing inappropriate sinus tachycardia. This is a diagnosis of exclusion when other possible causes for sinus tachycardia are eliminated and when the sinus node reacts very rapidly after minimal exercise (e. g., pulse > 150 beats/min after 10 knee bends).

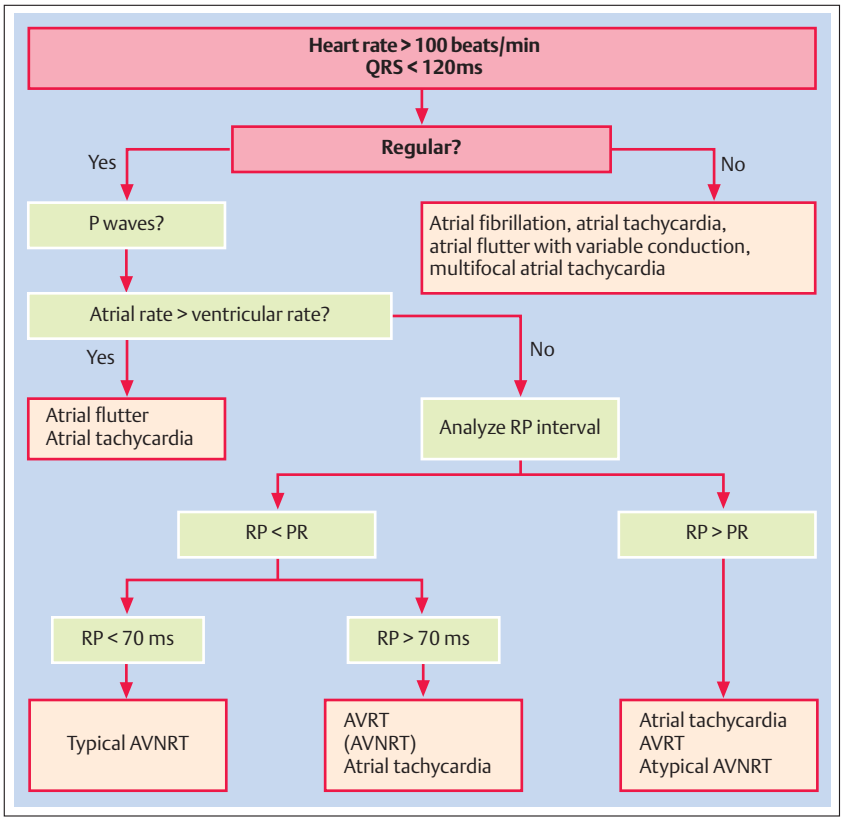


Fig. 22.**7** Differential diagnosis of narrow complex tachycardia. HR: heart rate, RP: Interval between R peak to P waves during the tachycardia, AVNRT: AV nodal reentrant tachycardia, AVRT: AV reentrant tachycardia.

Atrial Tachycardia

In atrial tachycardia the atrial rate is usually 150–250 beats/min and the P wave morphology is different from that observed during sinus rhythm (Fig. 22.**8**). The ECG derivation with a negative initial P wave suggests the origin of the tachycardia (V_1 right atrium; aVL left atrium). Atrial tachycardia may show some irregularity and patients with this arrhythmia usually have atrial ectopic beats originating from the same focus with similar P-wave morphology.

Atrial tachycardia may be a sustained arrhythmia and may have a single or multiple origins. In the latter case, the so-called *multifocal atrial tachycardia*, there are often secondary causes, and the atria are often dilated due to pressure or volume overload of the atria, hypertension, or hyperthyroidism. Atrial tachycardia can also arise from the pulmonary veins and may trigger atrial fibrillation. In this case, short bursts of fast, irregular beats arising from the pulmonary veins are commonly observed.

Atrial Flutter

The atrial rate during atrial flutter is usually 220–350 beats/min, and the atrial rhythm is more or less regular. The mechanism of atrial flutter is always an *abnormal pulse propagation* (macroreentry), which in 80 % of cases involves the entire right atrium. The flutter waves typically have a saw-tooth appearance and since some parts of the atria are constantly electrically active, there is no isoelectric line in between these waves, as opposed to the flat isoelectric line observed in atrial tachycardia. In the most common form of atrial flutter, the flutter waves are negative in inferior derivations (II, III, aVF) suggesting a caudo–cranial (counterclockwise) activation pattern along the atrial septum (Fig. 22.**9**). In patients who have surgical scars in the atria, the reentry may occur around these scars and different flutter wave patterns may arise.

AV Conduction. The conduction over the AV node to the ventricles is usually 2:1. Therefore, the typical pulse during atrial flutter is usually 130–150 beats/min. Under catecholaminergic influence or in patients receiving class I antiarrhythmic therapy (e. g., flecainide), the tachycardia may show 1:1 conduction, usually with wide complexes due to rate-dependent aberration, and become life-threatening due to its rapid rate. Conversely, the AV conduction may slow down (e. g., 3:1 or 4:1) with carotid massage or by application of various

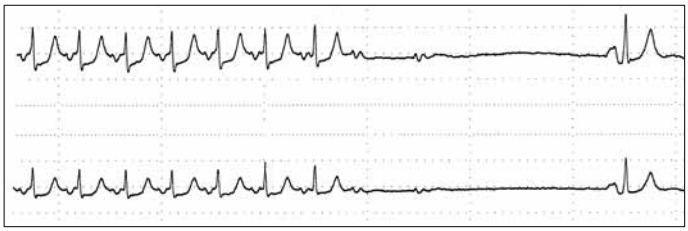


Fig. 22.**8** Atrial tachycardia. The ectopic atrial tachycardia has a different P wave morphology from that of the sinus beat. Adenosine can terminate focally-induced atrial tachycardias. In this example, adenosine terminates the tachycardia shortly after causing AV block. Note the two P waves without conduction to the ventricles prior to termination of this tachycardia.

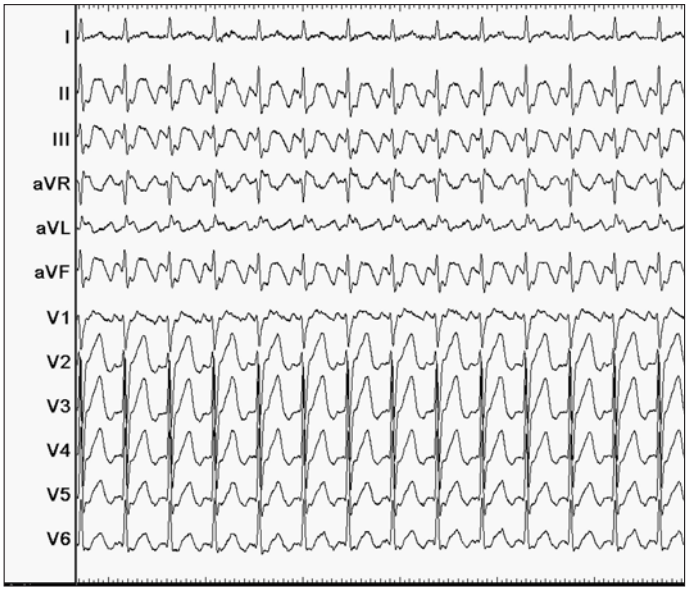


Fig. 22.**9** Typical atrial flutter. The negative flutter waves in inferior derivations (II, II, aVF) suggest a counterclockwise activation within the right atrium.

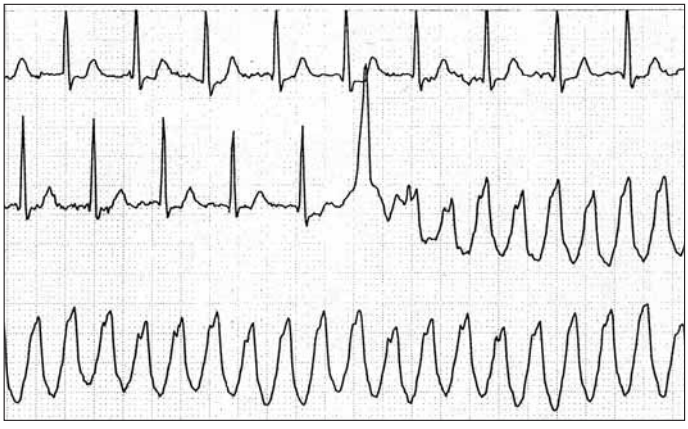


Fig. 22.**10** Atrial flutter with 2:1 conduction converting into 1:1 conduction with aberration. The initial tachycardia is regular at a rate of 150 beats/min and has a narrow complex. The second tachycardia is exactly twice as fast as the initial tachycardia, which is unlikely to occur by chance. If the initial tachycardia were not documented, it would be difficult to differentiate atrial flutter from ventricular tachycardia in this patient.

drugs resulting in the unmasking of the flutter waves (Fig. 22.**10**). The ventricular rate during atrial flutter has a certain regularity, differentiating it from atrial fibrillation, which has a totally irregular rhythm. Atrial flutter is responsive to curative therapy with radiofrequency catheter ablation in which the macroreentry is interrupted at a narrow electrical isthmus.

Atrial Fibrillation

Atrial fibrillation is one of the *most common arrhythmias*, affecting 7% of the population at age 60 or older (Fig. 22.**11**). In contrast to atrial flutter, the ventricular rate during atrial fibrillation is absolutely irregular due to the chaotic fibrillatory activity in the atria at very high rates (> 300 beats/min). However, the arrhythmia may become somewhat regular at rapid ventricular rates. Every absolutely irregular rhythm is considered atrial fibrillation until proven otherwise, even in the absence of identifiable P waves and even with wide QRS

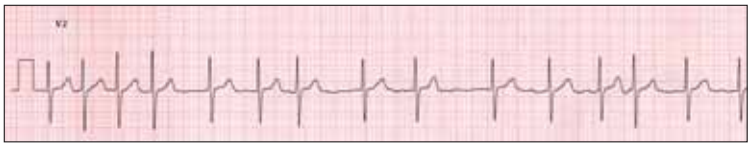
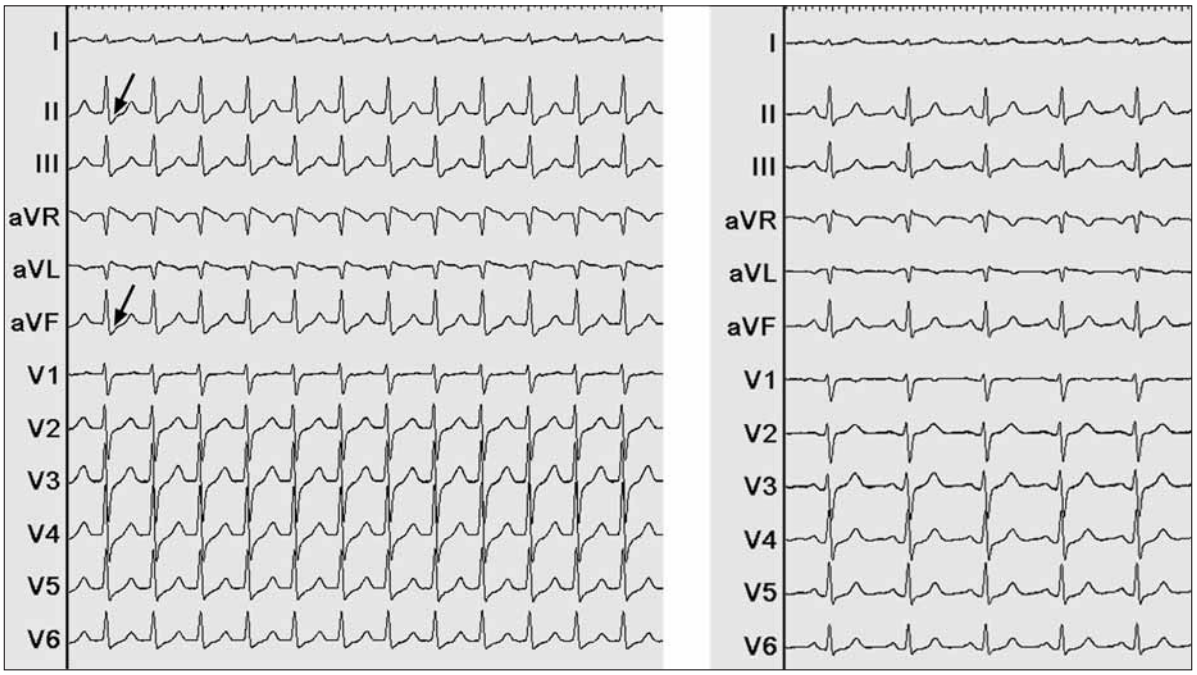


Fig. 22.**11** Atrial fibrillation. Note the absolutely irregular rhythm and the lack of P waves.



a b

Fig. 22.**12 a** AV nodal reentrant tachycardia. Note the narrow P waves that are negative in inferior (II, III, aVF) derivations (arrows). **b** In comparison, sinus rhythm ECG of the same patient.

Table 22.**5** Causes of atrial fibrillation

- Pulmonary vein tachycardia
- Hypertensive heart disease
- Heart failure
- Ischemic heart disease
- Sick sinus syndrome
- Cardiomyopathies
- Rheumatic valve disease
- Hyperthyroidism
- Myocarditis, pericarditis
- Preexcitation syndrome (WPW syndrome)
- Alcohol, caffeine
- Postcardiac surgery
- Pulmonary diseases

complexes. Conversely, a very regular tachycardia rules out the diagnosis of atrial fibrillation (DD: atrial flutter, junctional rhythm).

Atrial fibrillation is classified as being *paroxysmal* (self-terminating), *persistent* (sustained atrial fibrillation that can only be returned to sinus rhythm by cardioversion), or *permanent* (sustained atrial fibrillation that is either resistant to, or not appropriate for cardioversion). The causes of paroxysmal atrial fibrillation are listed in Tab. 22.**5**. Commonly, atrial fibrillation, atrial flutter, and atrial tachycardias occur in the same patient and can induce one another.

AV Nodal Reentrant Tachycardia

AV nodal reentrant tachycardia is the most common regular, paroxysmal, supraventricular tachycardia, which represents approximately 60% of cases. The mechanism is reentry that occurs in the region of the AV node using the so-called slow pathway, a second structure that is considered to be physiologic in many individuals and conducts at a lower rate as compared to the fast pathway. Typically, the tachycardia causes simultaneous contraction of the atria and the ventricles. Since the atrial contraction occurs as the atrioventricular valves are closed, patients commonly feel pulsation in the neck veins during the tachycardia and commonly report increased diuresis following the arrhythmia.

In the ECG, *retrograde P waves* may be identified within or shortly after the QRS complex. In the V_1 lead this pattern may mimic the presence of an incomplete right bundle branch block. Therefore, it is important to compare the QRS complexes during the tachycardia with those obtained during sinus rhythm (Fig. 22.**12**).

The P waves during the tachycardia are usually small and negative in inferior derivations due to the simultaneous activation of both atria from the region of the AV node.

In the less commonly observed atypical form of AV nodal reentrant tachycardia, the reentry occurs in the opposite direction and the P waves can be readily identified with RP intervals longer than the PR interval. Some patients with AV nodal reentrant tachycardia may have PQ intervals less than 120 ms in sinus rhythm.

AV nodal reentrant tachycardia is catecholamine-dependent and can typically be terminated with vagal maneuvers (Valsalva, carotid massage) or adenosine injection.

AV Reentrant Tachycardia with Antegrade Conduction over the AV Node

Patients with AV reentrant tachycardia have, in addition to the AV node, an *accessory electrical conduction* between the atria and the ventricles. These accessory pathways may be found in the left or right atrioventricular groove or may even be multiple in the same patient. Approximately 60% of these connections only have the ability for retrograde conduction (from the ventricles to the atria). The conduction is bi-directional in 30% and only antegrade in 10%. In WPW syndrome, there is antegrade conduction over the accessory pathway during sinus rhythm, which results in preexcited QRS complexes (delta waves).

Physiologically, an AV reentrant tachycardia using the AV node as the antegrade pathway and the accessory bundle as the retrograde pathway is far more common (Fig. 22.**13**). These tachycardias have a normal QRS morphology and the P wave is found at least 60 ms after the QRS complex. Occasionally, electrical alternans occurs with varying amplitude, but constant width of the QRS complexes.

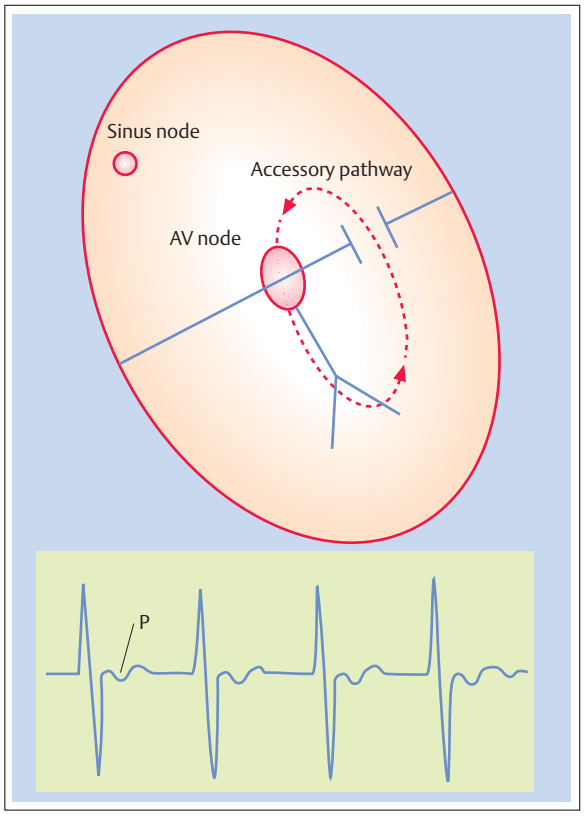


Fig. 22.**13** AV reentrant tachycardia with antegrade conduction over the AV node.

Wide-Complex Tachycardia

Differential Diagnosis of Wide-Complex Tachycardias

In 80% of wide-complex tachycardias the diagnosis is ventricular tachycardia. The remaining 20% are due to supraventricular tachycardia with aberration, pre-existing bundle branch block, or preexcitation (WPW syndrome). The differential diagnosis of a wide-complex tachycardia is extremely important. Depending on the diagnosis, the therapy and prognosis are very different. A structured approach is advisable for the differentiation of wide-complex tachycardias:

Medical History. In *postmyocardial infarction* patients, ventricular tachycardia is more likely to occur than other arrhythmias. Pathologic Q waves can be recognized even during the tachycardia on derivations corresponding to the scar left by a transmural myocardial infarction. A previous ECG can be very useful for identifying delta waves and possibly preexisting intraventricular conduction disturbances, bundle branch block, etc.

Ratio of Atrial and Ventricular Beats. It is important to carefully study the ratio of P waves to QRS complexes on tachycardia ECGs. Approximately one-third of ventricular tachycardias exhibit retrograde conduction over the AV node, producing regular associated P waves with a 1:1 AV relationship. Conversely, demonstration of AV dissociation (independent rhythm in the ventricles and the atria) is considered to be diagnostic for ventricular tachycardia. *Carotid sinus massage* may provide diagnostic information by slowing or interrupting the retrograde conduction over the AV node, thus demonstrating dissociation of the atrial and ventricular excitation. Intermittent occurrence of fusion or capture beats due to antegrade conduction over the AV node is also diagnostic of ventricular tachycardia.

Ventricular Activation Axis. The *axis of ventricular activation* helps to identify the origin of the arrhythmia. For