Cardiac Arrythmias

(Tachyarrythmias & Bradyarrythmias)

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Overview of cardiac arrhythmias

Summary

Cardiac arrhythmias are accelerated, slowed, or irregular heart rates caused by abnormalities in the electrical impulses of
the myocardium. Bradyarrhythmias include sinus node dysfunction and atrioventricular block, and are characterized by a resting heart rate < 60/minutes.

Tachyarrhythmias (heart rates > 100/minute) are classified as supraventricular arrhythmias or ventricular arrhythmias. Supraventricular arrhythmias originate between the sinus node and the atrioventricular node. Ventricular arrhythmias originate below the atrioventricular node, on the ventricular level.

Classification

Bradyarrhythmias

Definition: heart rates of **< 60/min**

Tachyarrhythmias

Definition: heart rate of **> 100/min**

Supraventricular arrhythmias

Definition: arrhythmias that originate in the sinoatrial node, atrial myocardium, or atrioventricular node (regular QRS complex)

Ventricular arrhythmias

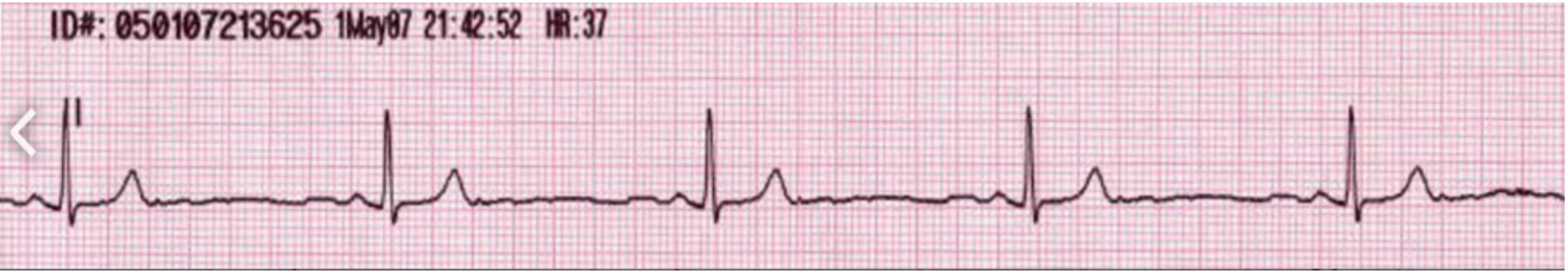
Definition: arrhythmias that originate below the atrioventricular node (wide QRS complex)

Bradyarrhythmias

Type of bradyarrhythmia	Causes and mechanisms	Main ECG findings
Atrial origin		
Respiratory sinus arrhythmia	• Physiological, particularly in youths	• Minor changes in the R-R interval during respiration: reduction during inspiration and increase during expiration
Sinus bradycardia	• Physiological, particularly in athletes • Sinus node dysfunction (sick sinus syndrome) • Drugs: beta blockers, calcium channel blockers	• Rate < 60 bpm • Normal P wave before every QRS complex

Type of bradyarrhythmia	Causes and mechanisms	Main ECG findings
Atrial origin		
Sinoatrial pause or arrest	• May occur in healthy individuals • Underlying cardiovascular disease (sick sinus syndrome)	• Transient absence of the P wave
Tachycardia-bradycardia syndrome	• Abnormal supraventricular impulse generation and conduction (sick sinus syndrome)	• Intermittent tachyarrhythmias and bradyarrhythmias

Type of bradyarrhythmia		Causes and mechanisms	Main ECG findings
AV node origin			
Atrioventricular block	First-degree block	• Physiological response • Increased vagal tone • Drugs: beta blocker or calcium channel blocker	• PR interval > 200 ms
	Second-degree block	• Drugs: digoxin, beta blocker, calcium channel blocker • Increased vagal tone • Sinoatrial conduction disease • Right coronary infarction	• Mobitz type I/Wenckebach: progressive lengthening of the PR interval until a beat is dropped • Mobitz type II: irregular dropped beats
	Third-degree block	• Complete block: no communication between the atria and ventricles	• AV dissociation: no relationship between P waves and QRS complexes

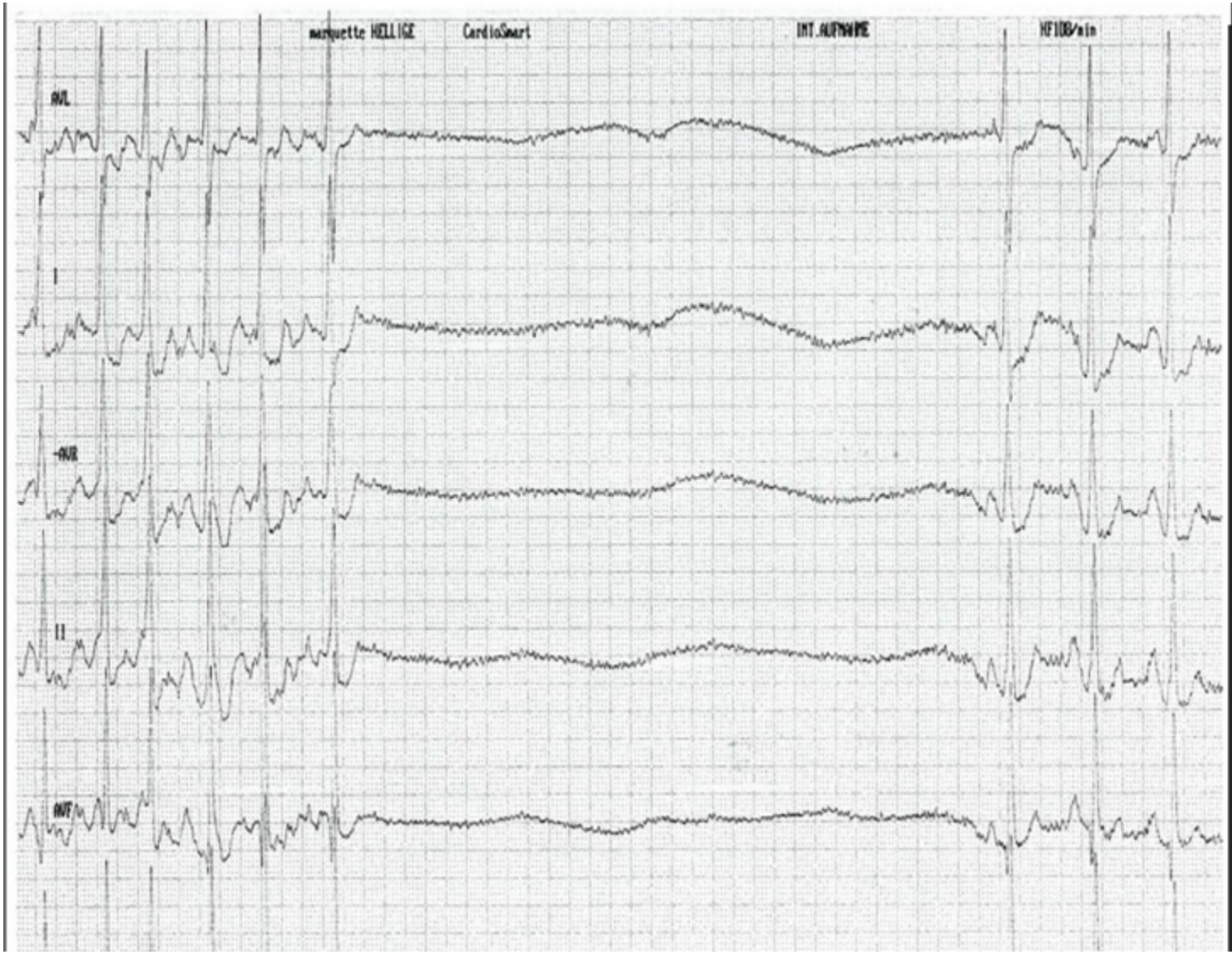


Sinus bradycardia

ECG (paper speed: 25 mm/s)

Regular sinus rhythm with a heart rate of 37/min.
PR interval normal (approx. 160 ms), QRS normal (approx. 110 ms), QT interval normal (approx. 480 ms; QTc = 380 ms).
Morphologies of P wave, QRS complex and T wave are physiologic. Note the U waves (occurring after the T waves).
No signs of ischemia.

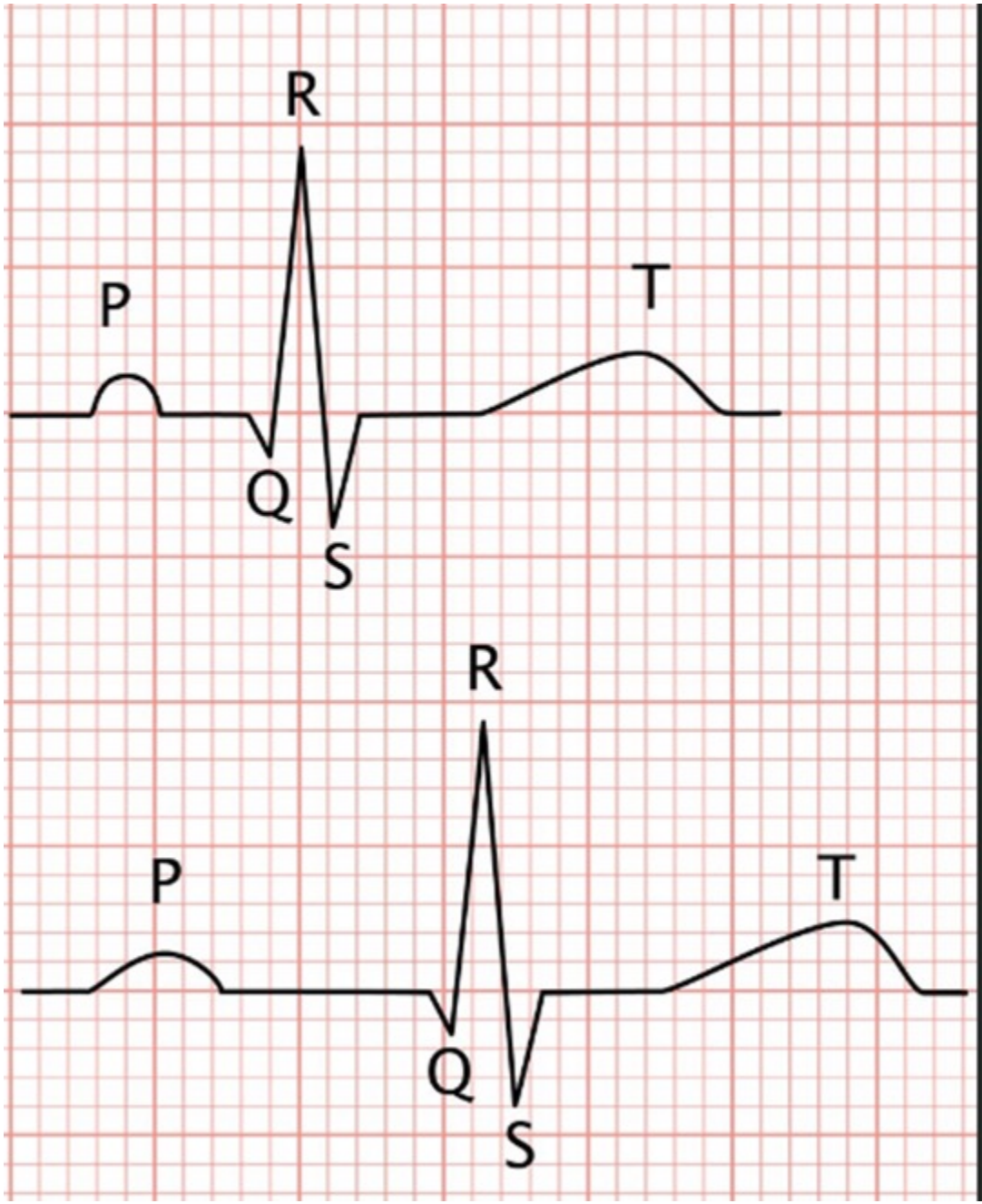
Diagnosis: sinus bradycardia



ECG with tachycardia–bradycardia syndrome (variant of sick sinus syndrome)

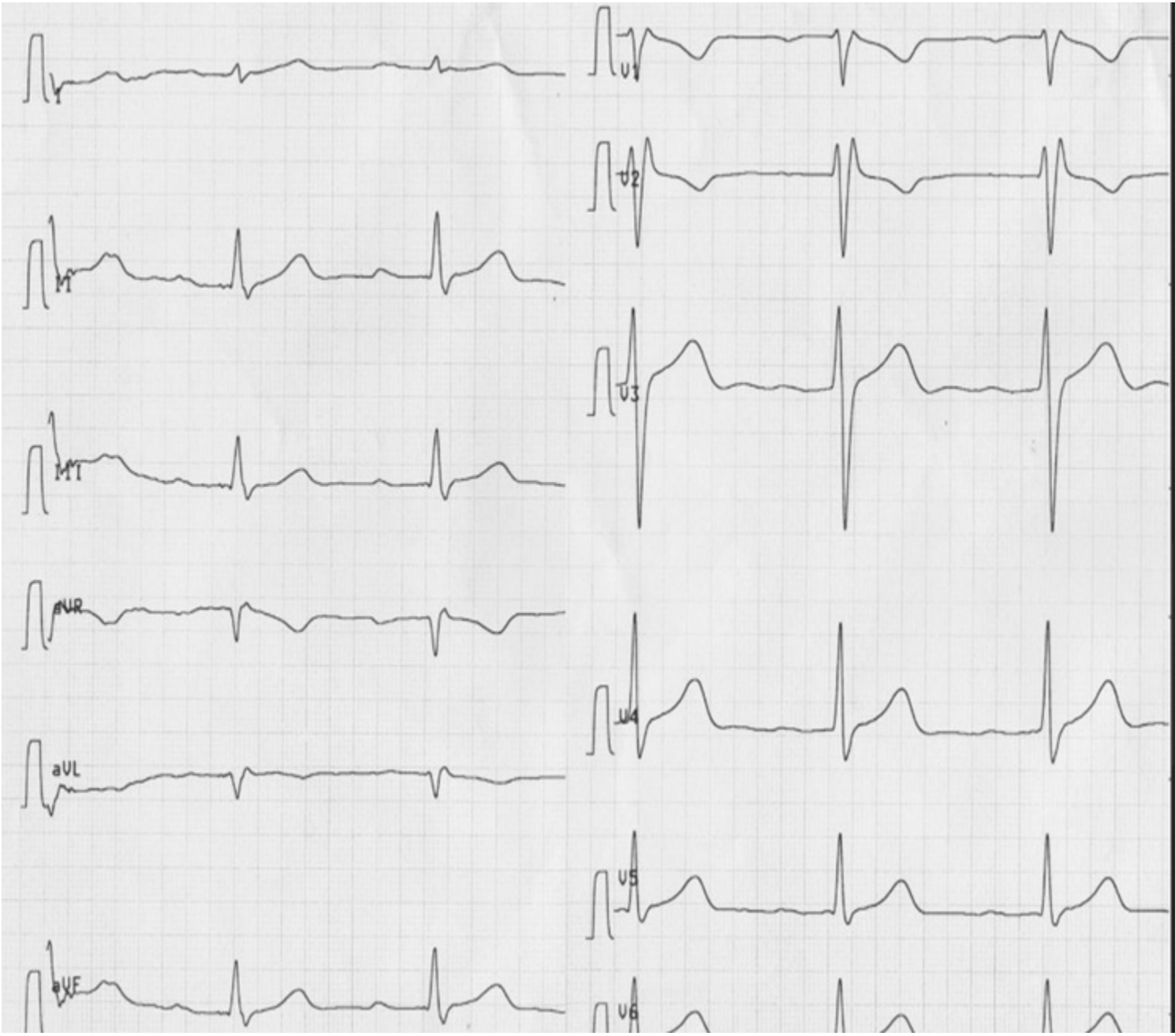
12-lead ECG: supraventricular tachycardia (most likely tachycardic atrial fibrillation) of approx. 110 bpm, one relevant pause of 6.6 s

followed by a normal SR with a frequency of approx. 75 bpm



First-degree atrioventricular block

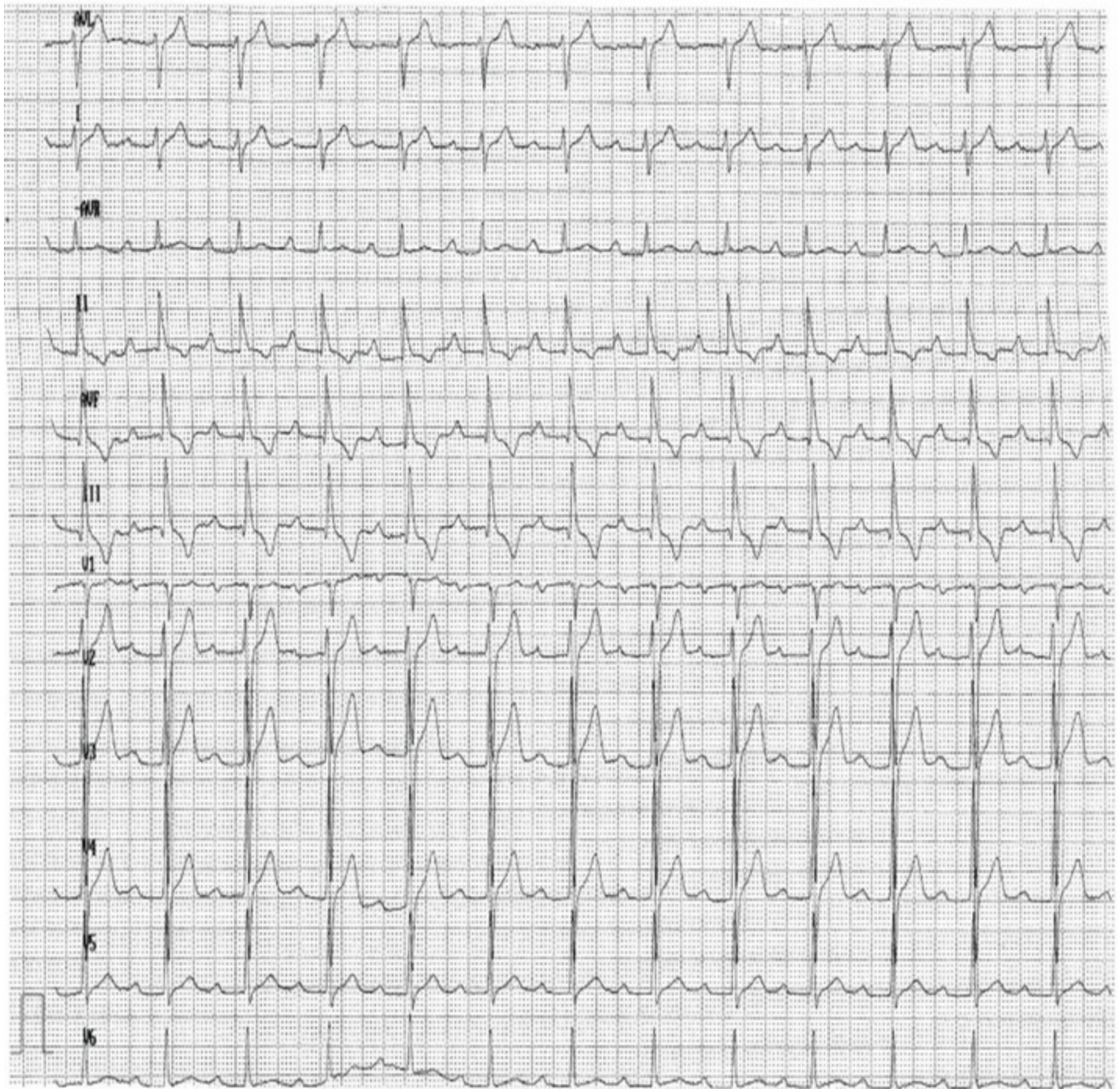
Top: normal ECG. Bottom: PR interval > 200 ms (paper speed of 50 mm/s). Normal QRS complex configuration. No indication of repolarization abnormalities.



First-degree AV block and incomplete RBBB in ECG

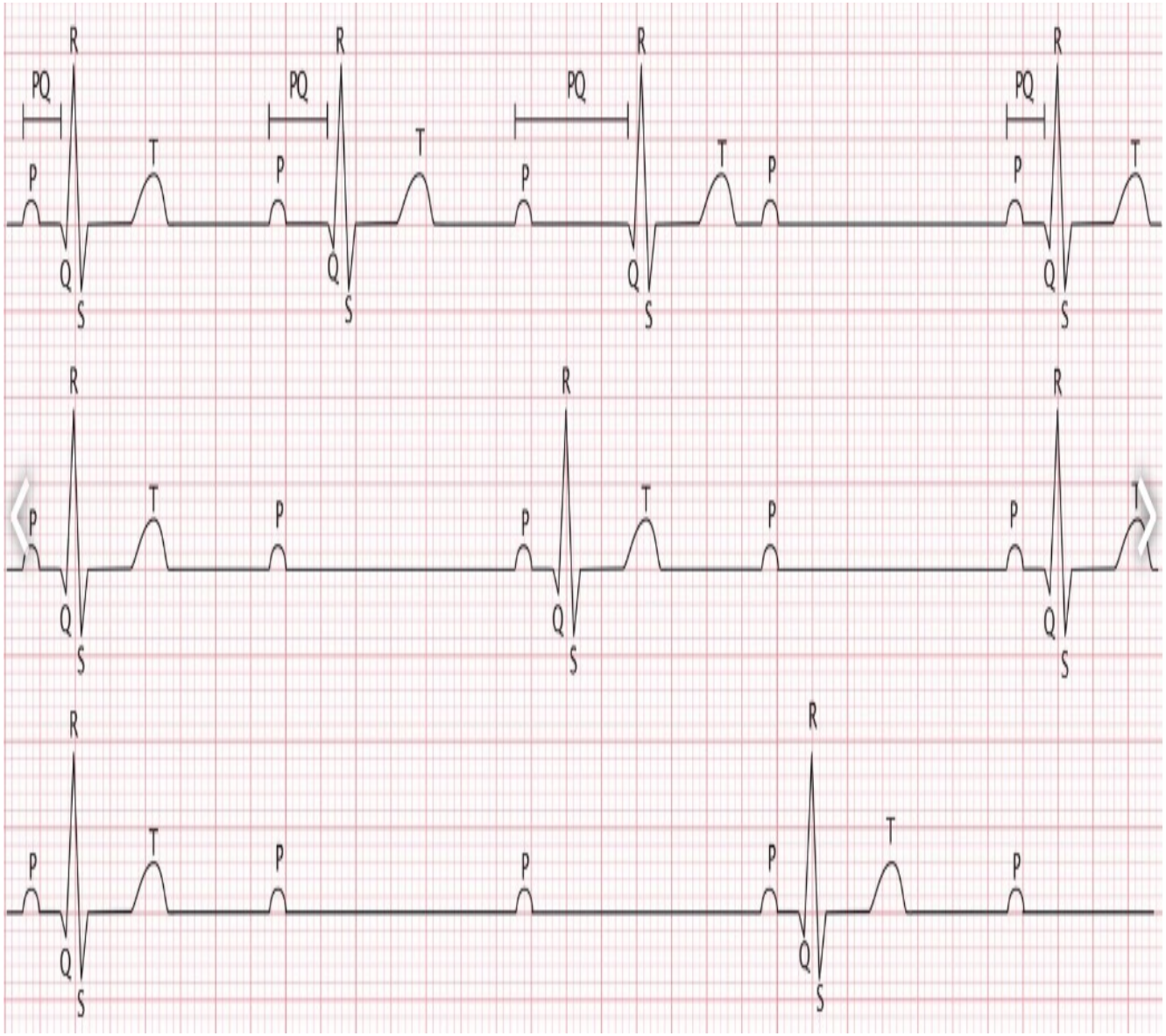
Sinus rhythm with a heart rate of approx. 70/min.

Normal axis; PQ interval of 220 ms (> 0.2 s), consistent with first-degree atrioventricular block; incomplete right bundle branch block with characteristic RSR' in V1–V2 and QRS complex of 0.11 s.



First-degree atrioventricular block

Sinus rhythm with a heart rate of approx. 75/min. Borderline right axis deviation (R ≈ S in I, R > S in aVF). PR interval prolonged (280 ms); borderline positive Sokolow-Lyon index for left ventricular hypertrophy (3.2 mV); preterminal negative T wave in II, III, and AVF.



Second-degree atrioventricular blocks

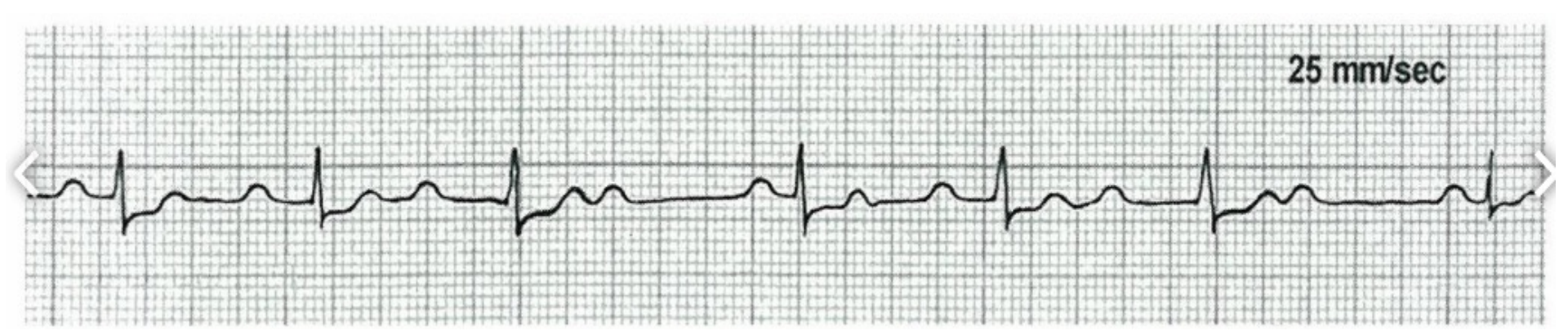
ECG illustration of AV blocks (paper speed: 50 mm/s)

Regular P waves with a frequency of 85/min in all tracings.

Top: second-degree AV block (Mobitz I/Wenckebach). Progressive lengthening of the PR interval with a blocked QRS complex following the fourth P wave. Ventricular rate approx. 74/min.

Middle: second-degree AV block (Mobitz II). PR interval is constant but only every second P wave is conducted to the ventricle, where it triggers a QRS complex (2:1 block). Ventricular rate is approx. 42/min.

Bottom: second-degree AV block (Mobitz II). PR interval is constant but only every third P wave is conducted to the ventricle, where it triggers a QRS complex (3:1 block). Ventricular rate is only approx. 28 bpm.

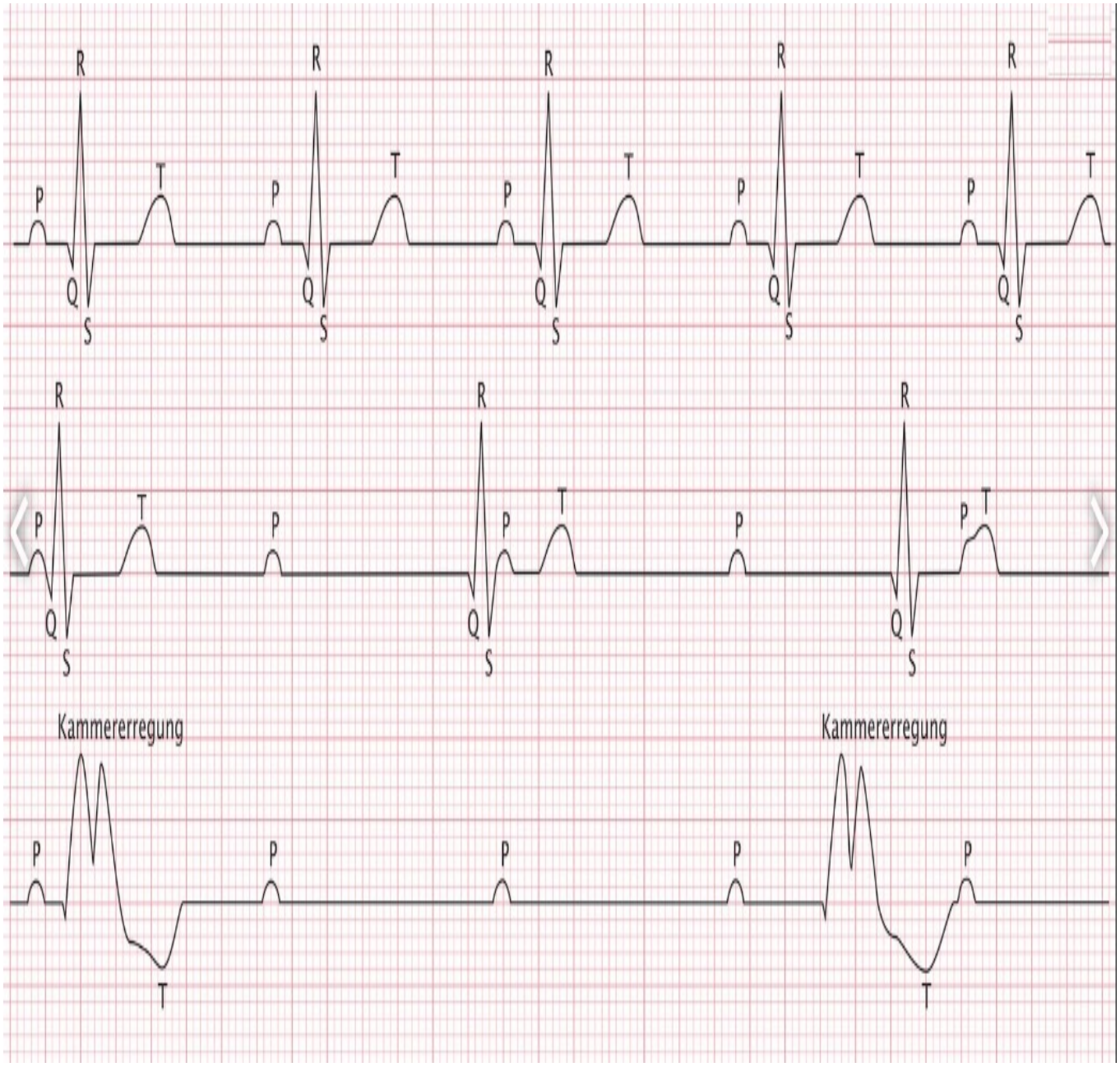


Second-degree AV block (Mobitz I, Wenckebach)

ECG strip (paper speed 25mm/s)

Regular P waves with a frequency of 75–80/min. The PR interval increases steadily until, after the fourth P wave, the QRS complex is missing.
The QRS complexes have normal morphology but occur irregularly (frequency approx. 65/min).

Diagnosis: second-degree atrioventricular block (Mobitz I, Wenckebach)



Third-degree atrioventricular block

ECG illustration of AV blocks (paper speed: 50 mm/s)

Top: normal ECG with a heart rate of approx. 90/min.

Middle: third-degree AV block with a ventricular escape rhythm (site of origin at the AV node) with a rate of approx. 50/min. QRS complexes are narrow but within normal limits.

Bottom: third-degree AV block with a ventricular escape rhythm (site of origin at the bundle of His or the bundle branches) with a rate of approx. 27/min. QRS complexes are widened, resembling bundle branch block.

In both types of third-degree AV block, the P waves occur at a regular rate of approx. 90/min (the rate of the sinoatrial node). However, the QRS complexes occur in accordance with the rhythm of the abnormal site of origin.

Tachyarrhythmias
Supraventricular arrhythmias

Type of tachyarrhythmia	Causes and mechanisms	Main ECG findings
Atrial origin		
Supraventricular premature beats	• Physiological response in healthy individuals • Electrolyte imbalances • Underlying cardiovascular disease	• P wave abnormalities or absent P waves

Type of tachyarrhythmia	Causes and mechanisms	Main ECG findings
Atrial origin		
Sinus tachycardia	• Sympathetic activation or vagal withdrawal on the SA node	• Gradual onset • Regular rhythm • Rate: max. rate usually 180 bpm • P wave: normal morphology • Narrow QRS complex

Type of tachyarrhythmia	Causes and mechanisms	Main ECG findings
Atrial origin		

Atrial flutter	• Macroreentrant rhythms within the atria	• Regular rhythm
		• Rate: atrial 250–350; ventricular < 200
		• P waves
		  • Occur before every QRS complex
		  • Sawtooth appearance of regular P waves (flutter waves) especially in leads II, III, and aVF
		• Narrow QRS complex

Type of tachyarrhythmia	Causes and mechanisms	Main ECG findings
Atrial origin		
Atrial fibrillation	• Multiple mechanisms which are not completely understood	• Rhythm: irregularly irregular • P-waves are indiscernible • Narrow QRS complex

Type of tachyarrhythmia		Causes and mechanisms	Main ECG findings
Atrial origin			
Atrial tachycardia (~ 5%)	Focal atrial tachycardia	• Discharge from a single ectopic focus in the atrium	• Very abrupt onset • Regular rhythm • Rate: 150–250 • P wave: morphology varies depending on the site of the ectopic focus • Occurs before the QRS complex • Narrow QRS complex
	Multifocal atrial tachycardia (MAT)	• Discharge from multiple ectopic foci in the atrium • Associated with pulmonary disorders (e.g., COPD exacerbation, pulmonary embolism), cardiac conditions (CHF exacerbation), and treatment with theophylline	• Very abrupt onset with rate variation • Rhythm: irregularly irregular • Rate: 150–250 • Discernible P waves with ≥ 3 different P wave morphologies; no single morphology is predominant • Narrow QRS complex

Type of tachyarrhythmia	Causes and mechanisms	Main ECG findings
AV node origin		

Atrioventricular reentry tachycardia (AVRT)	• A form of paroxysmal supraventricular tachycardia
	• Tachycardia caused by an accessory pathway between the atria and ventricles
		• Very abrupt onset
		• Regular rhythm
		• Rate: 150–250
		• P wave
		  ◦ Inverted (downgoing in II, III and aVF and/or upright in aVR)
		  ◦ Occur after the QRS complex
		• RP interval is shorter than PR interval
		• QRS complex
		  ◦ Orthodromic AVRT: narrow QRS complex
		  ◦ Antidromic AVRT: wide QRS complex with delta waves (WPW syndrome)

Type of tachyarrhythmia	Causes and mechanisms	Main ECG findings
AV node origin		
AV nodal reentry tachycardia (AVNRT)	• A form of paroxysmal supraventricular tachycardia • A dysfunctional AV node that contains two electrical pathways	• Regular rhythm • Rate: 150–250 • P waves occur during (i.e. are not visible) or after the QRS complex • RP interval is shorter than PR interval • Narrow QRS complex

Type of tachyarrhythmia	Causes and mechanisms	Main ECG findings
AV node origin		
Junctional tachycardia	• The AV node takes over the pacemaker function • Digitalis toxicity • Myocarditis • Myocardial infarction	• Regular rhythm • Rate: 100–130 • P waves occur before, during, or after the QRS complex • P waves are inverted • AV dissociation usually occurs • Narrow QRS complex

Ventricular arrhythmias

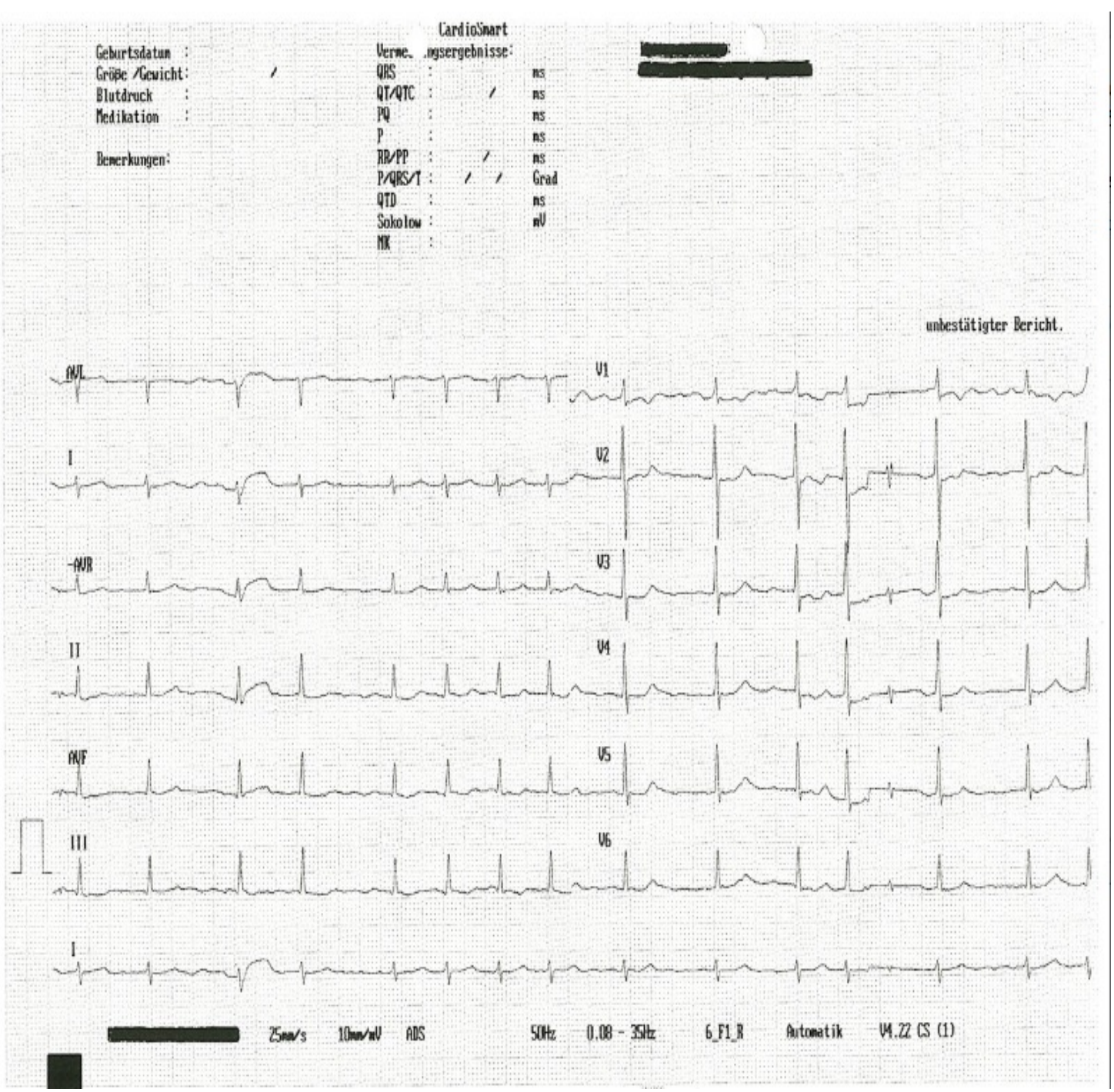
Type of arrhythmia	Causes and mechanisms	ECG findings
Premature ventricular beats (PVCs)	• Ectopic beat that originates from a ventricular focus • Due to hypoxia, hyperthyroidism, electrolyte abnormalities, ischemia or other heart pathology.	• Premature, wide QRS complex that is not preceded by a P wave • Compensatory pause after the premature beat

Type of arrhythmia	Causes and mechanisms	ECG findings

Ventricular tachycardia	• Coronary artery disease
	• Myocardial infarction
	• Structural heart diseases
		• Regular, rapid rhythm
		• Wide QRS complexes ≥3 consecutive premature ventricular beats)
		  • Monomorphic VT (most common): single QRS morphology
		  • Polymorphic VT: multiple QRS morphologies
		• AV dissociation (P waves may or may not be discernible)

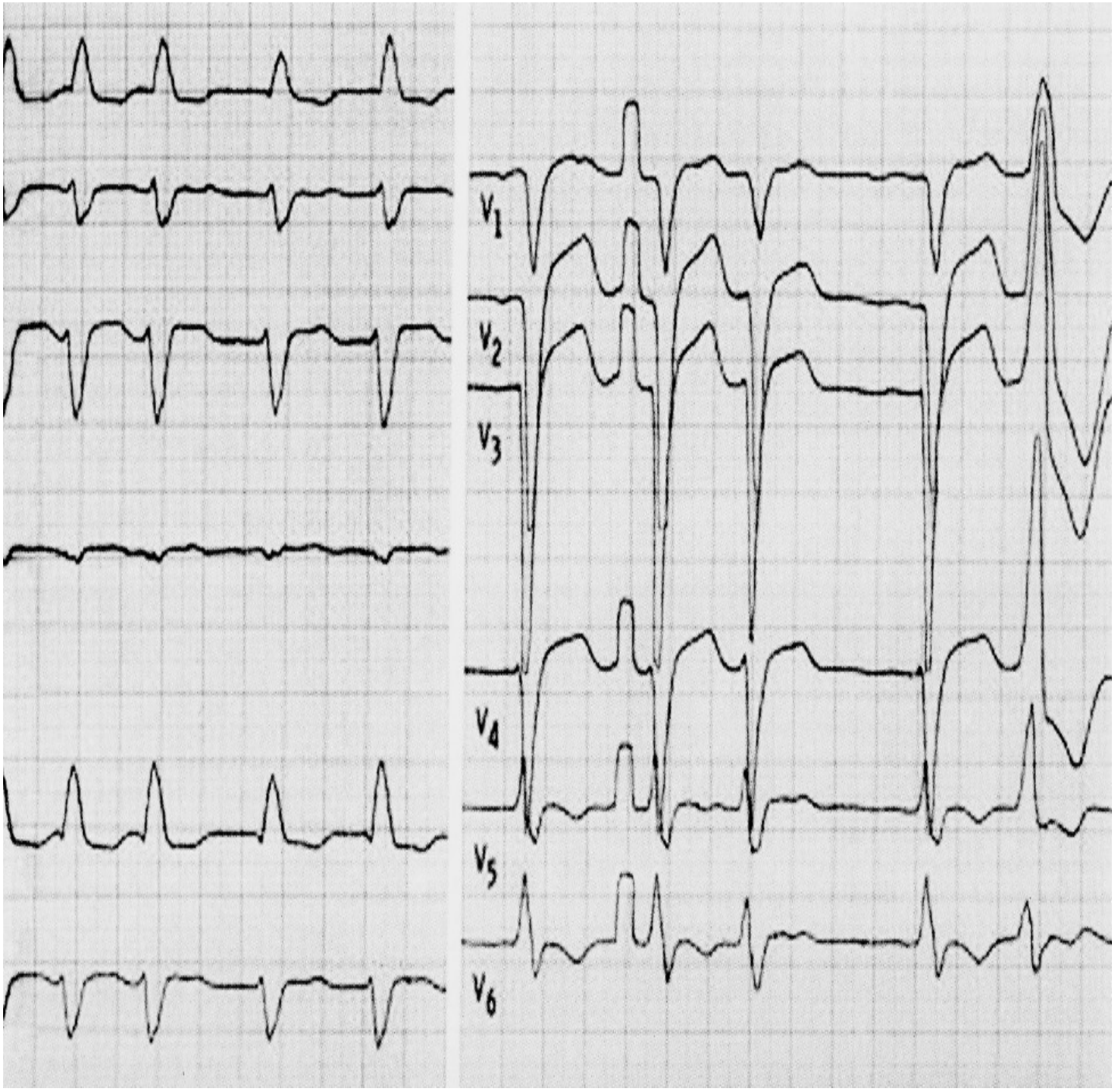
Type of arrhythmia	Causes and mechanisms	ECG findings
Torsade de pointes tachycardia	• Associated with Long QT syndrome • Proarrhythmic drugs • Electrolyte abnormalities (hypokalemia)	• Polymorphic ventricular tachycardia with QRS complexes that appear to twist around the isoelectric line

Type of arrhythmia	Causes and mechanisms	ECG findings
Ventricular fibrillation	• Myocardial infarction • Structural heart diseases	• Arrhythmic, fibrillatory baseline, usually > 300 bpm • Erratic undulations with indiscernible QRS complexes



ECG with tachycardic atrial fibrillation

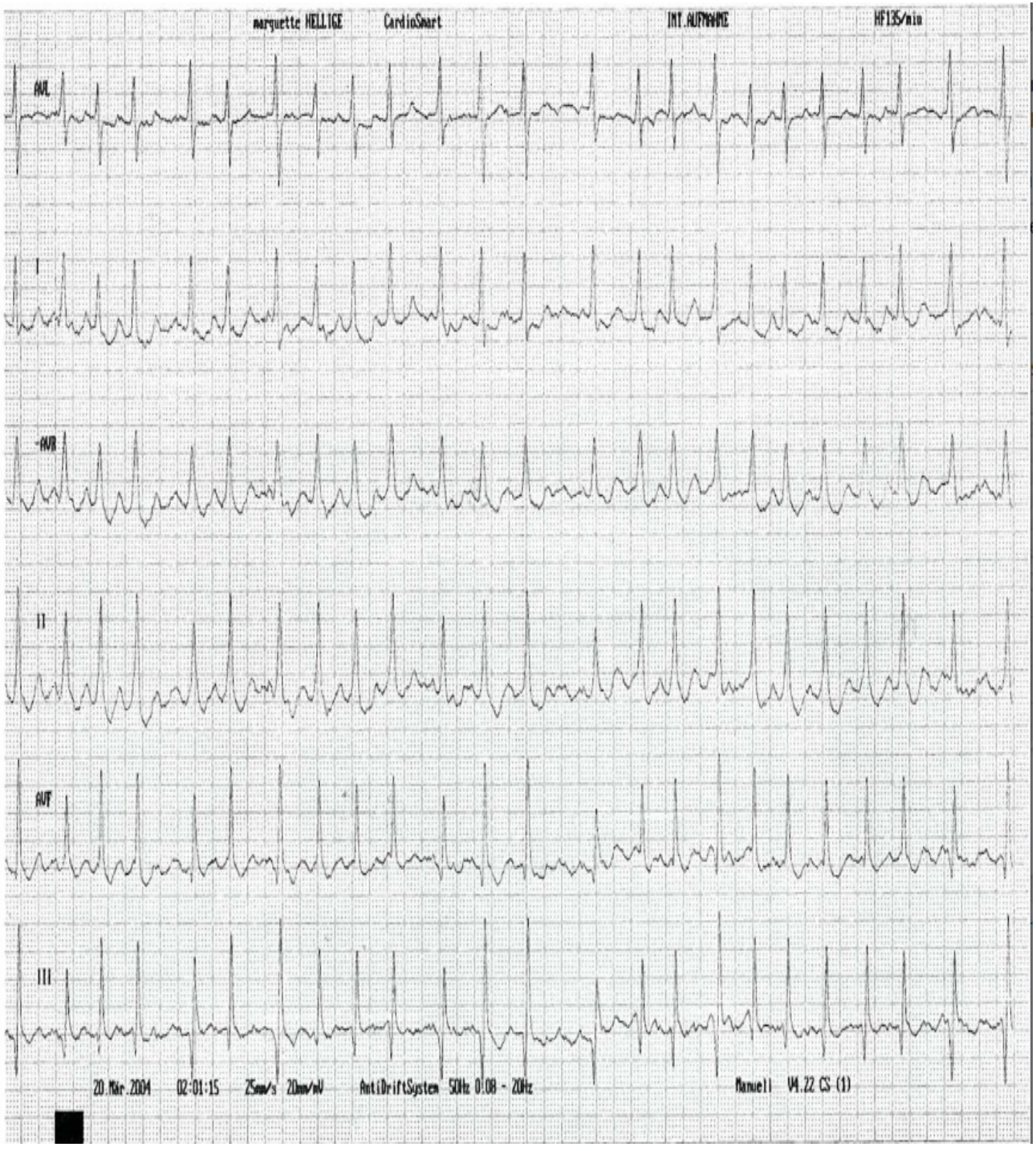
Findings: tachycardic atrial fibrillation with a ventricular rate of approx. 110 bpm (paper speed of 25 mm/s), transition from vertical heart to right axis deviation, no relevant repolarization abnormalities



Absolute arrhythmia in atrial fibrillation

12-lead ECG (paper speed: 25mm/s)

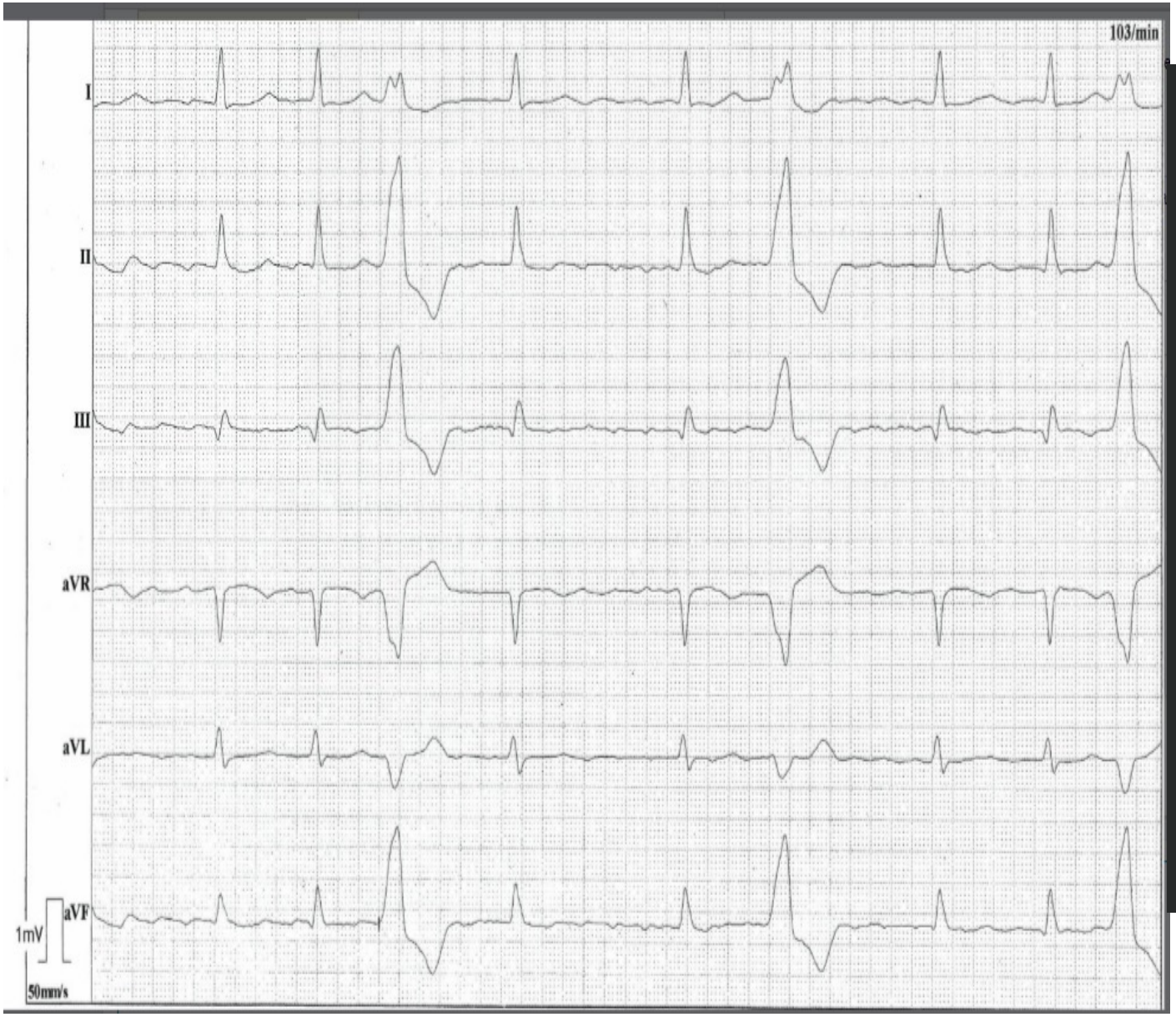
Approx. 115 bpm, left anterior fascicular block with severely delayed intraventricular conduction, ventricular extrasystole.



ECG with tachycardic atrial fibrillation

6-lead ECG (paper speed: 25 mm/s)

Tachycardic atrial fibrillation with a heart rate of approx. 135 bpm, vertical heart, no relevant repolarization abnormalities.

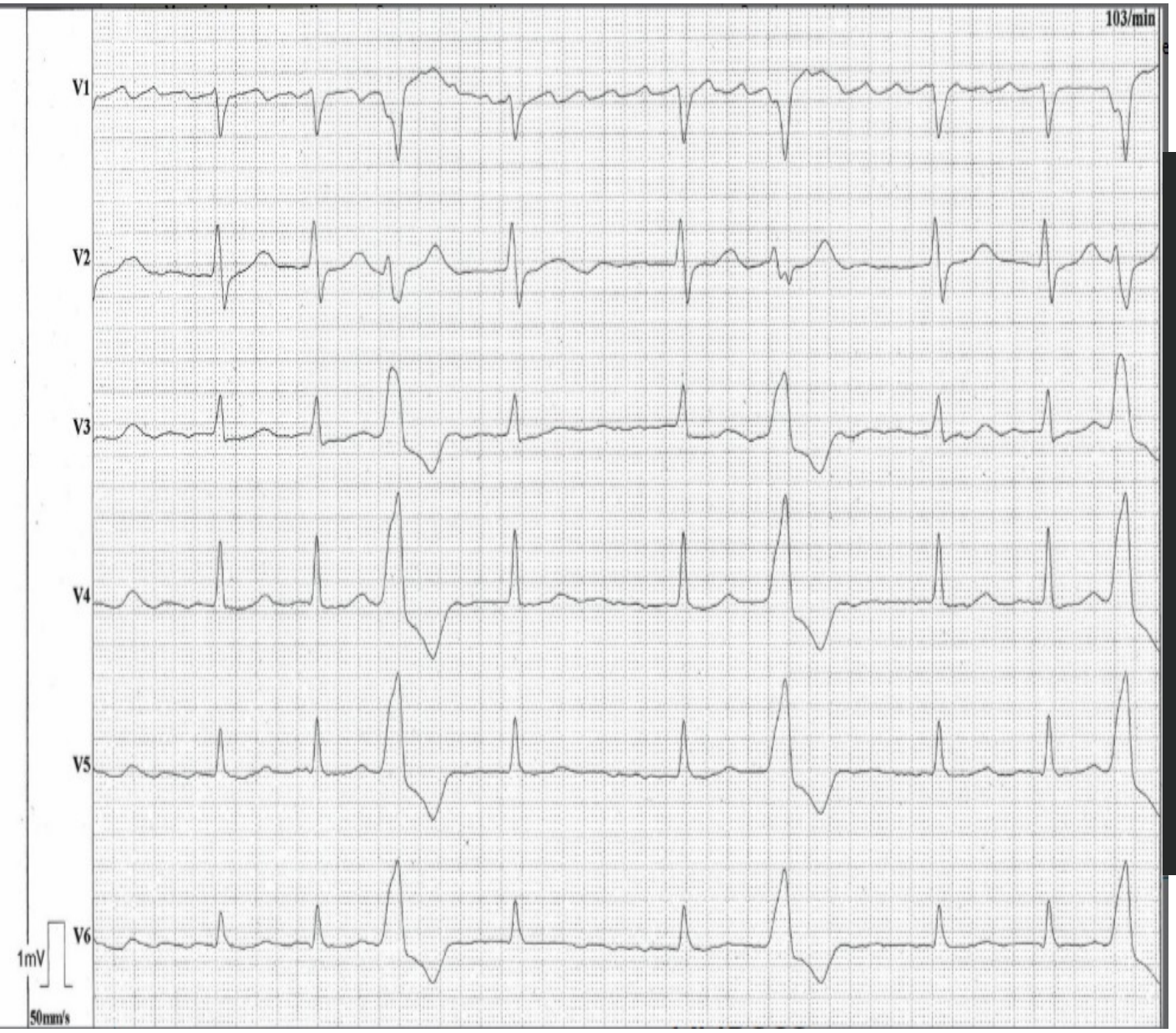


Atrial fibrillation and trigeminy in ECG

12-lead ECG (paper speed: 50 mm/s)

Absolute arrhythmia with an heart rate of approx. 103/min (according to the automated analysis; however, this is difficult to determine using the actual ECG). Fibrillations seen in V1. Normal heart axis (QRS complex positive in I–III with I > III). Two normally configured QRS complexes are followed by a premature ventricular beat; unspecific Q in III.

Diagnosis: tachyarrhythmia in atrial fibrillation with trigeminy

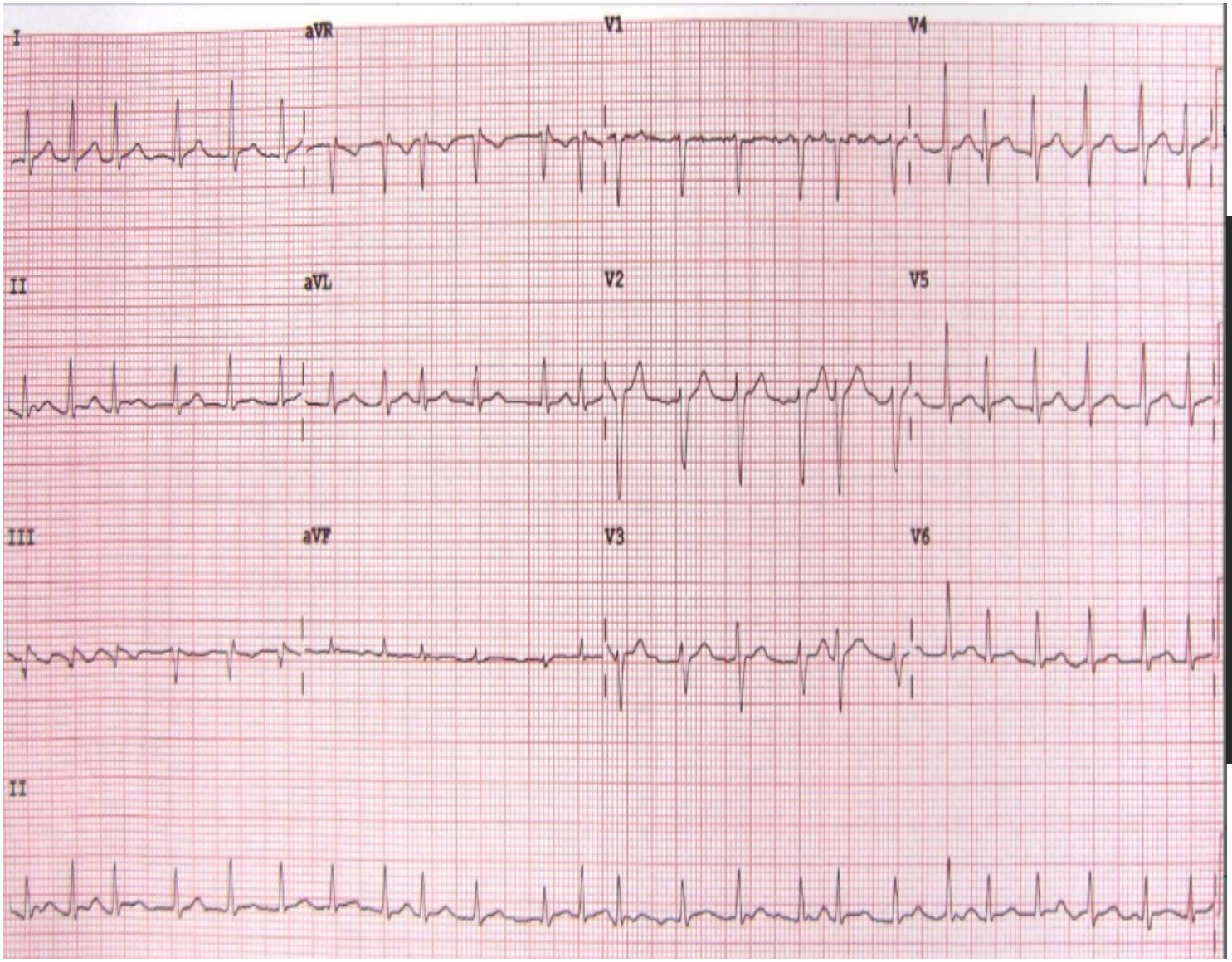


Atrial fibrillation and 2:1 extrasystole

6-lead ECG (paper speed: 50 mm/s)

Irregularly irregular rhythm with a ventricular rate of approx. 100/min.
No regular P waves are discernible (pronounced fibrillatory waves in V1).
2:1 extrasystole – monomorphic ventricular extrasystoles follow two normal QRS complexes.
No signs of ischemia.

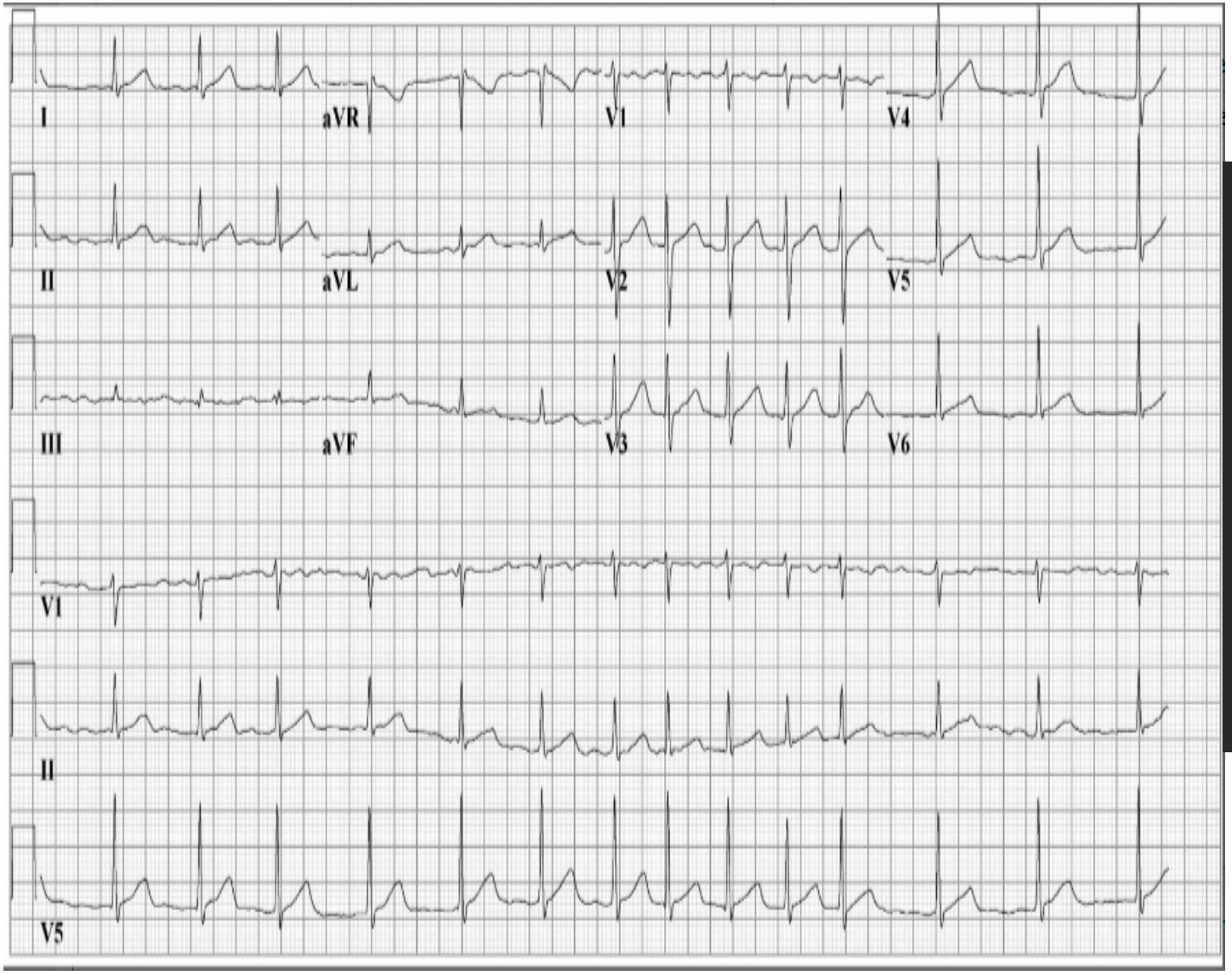
Diagnosis: atrial fibrillation



Atrial fibrillation

12-lead ECG (paper speed: 25 mm/s)

Irregularly irregular rhythm with a ventricular frequency between approx. 107/min and 166/min.
No P waves are discernible and there is no proper isoelectric baseline. QRS complexes are narrow (approx. 80 ms).

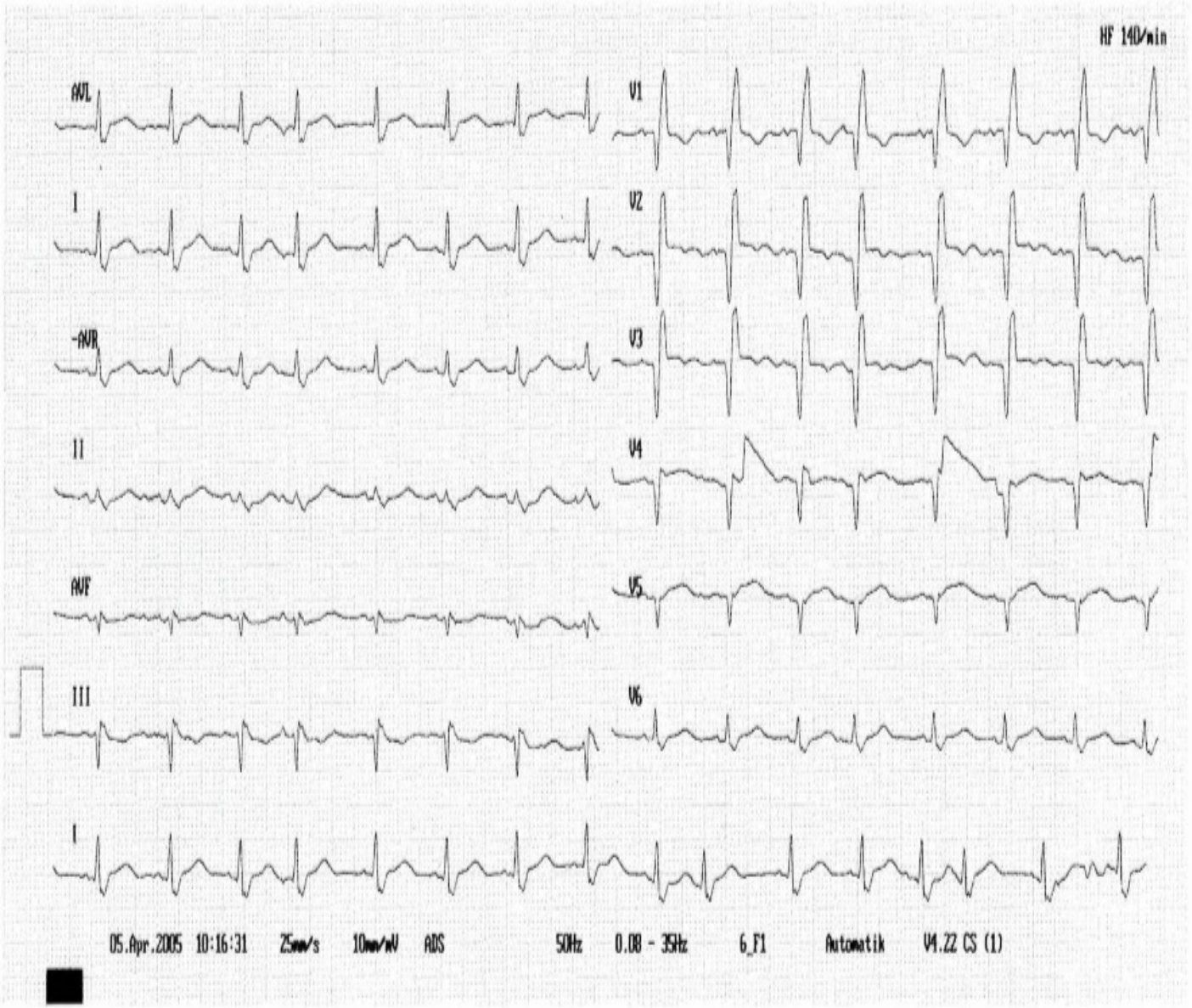


Atrial fibrillation

12-lead ECG (paper speed: 25mm/s)

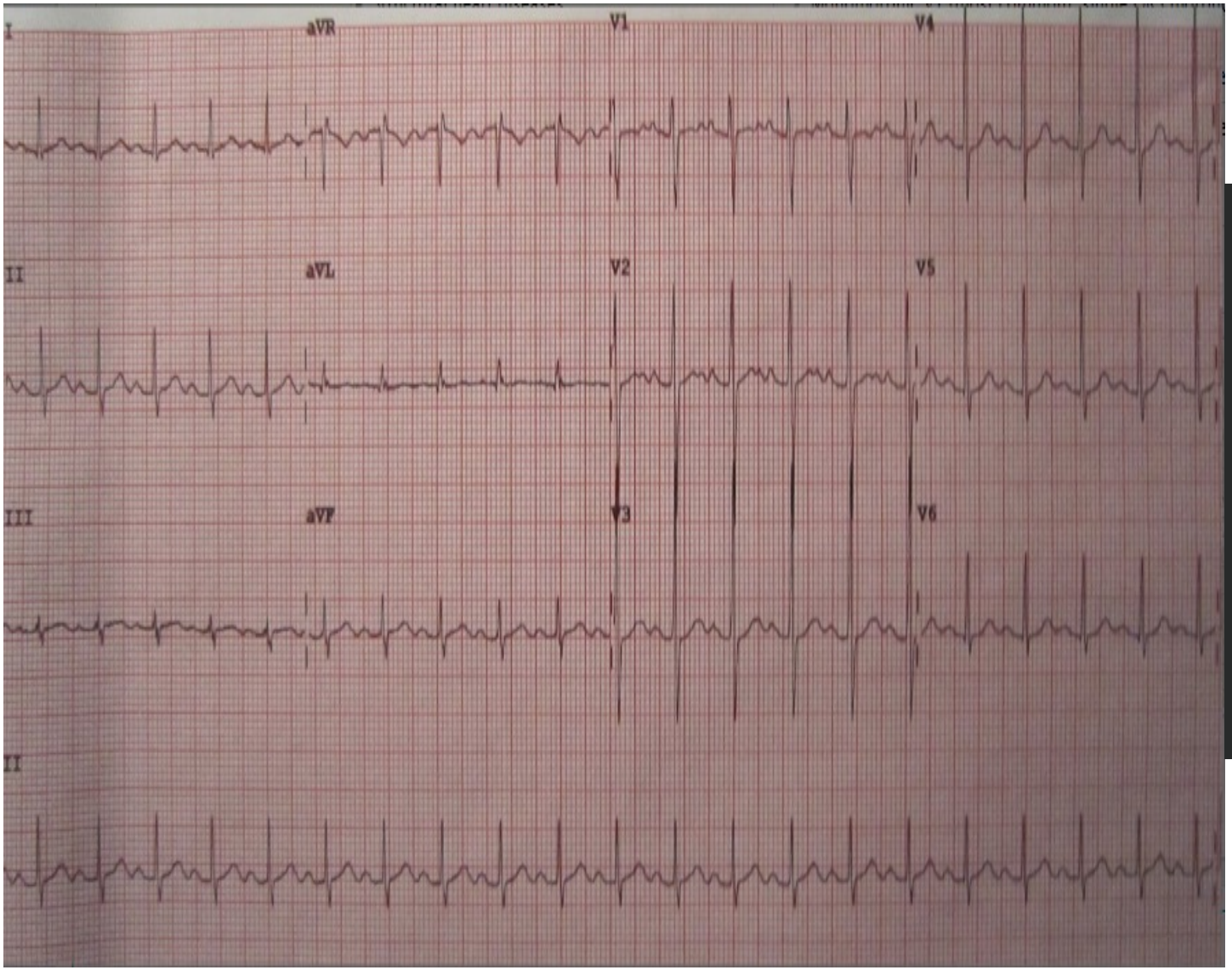
Irregularly irregular rhythm with a ventricular frequency between approx. 66/min and 120/min.
No P waves are discernible and there is no isoelectric baseline. QRS complexes are narrow (approx. 80ms).

Diagnosis: atrial fibrillation



Supraventricular premature beats

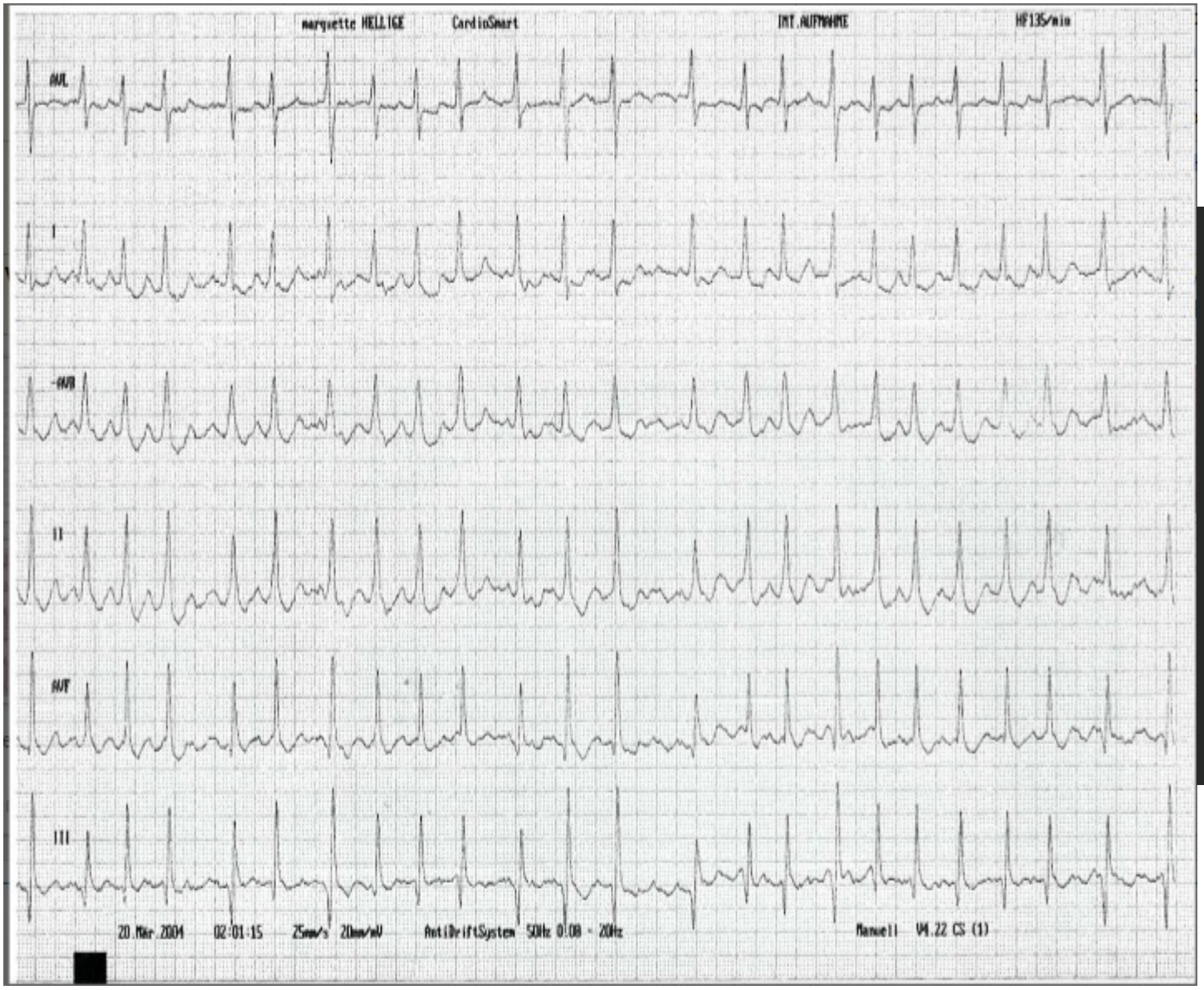
12-lead ECG (paper speed: 25 mm/s) in a 70-year-old female 5 days after a coronary intervention in myocardial infarction: Stable sinus rhythm with supraventricular premature beats in a rhythm strip (below, indicated as a red square). Heart rate of 140/min.
Marked left axis deviation.
Minor signs of infarction: mild ST elevation in leads V2-V4, widened and depressed Q wave in V1-V5, terminal negative T wave in V2 and V3, loss of R wave progression over the anterior wall



Sinus tachycardia

12-lead ECG

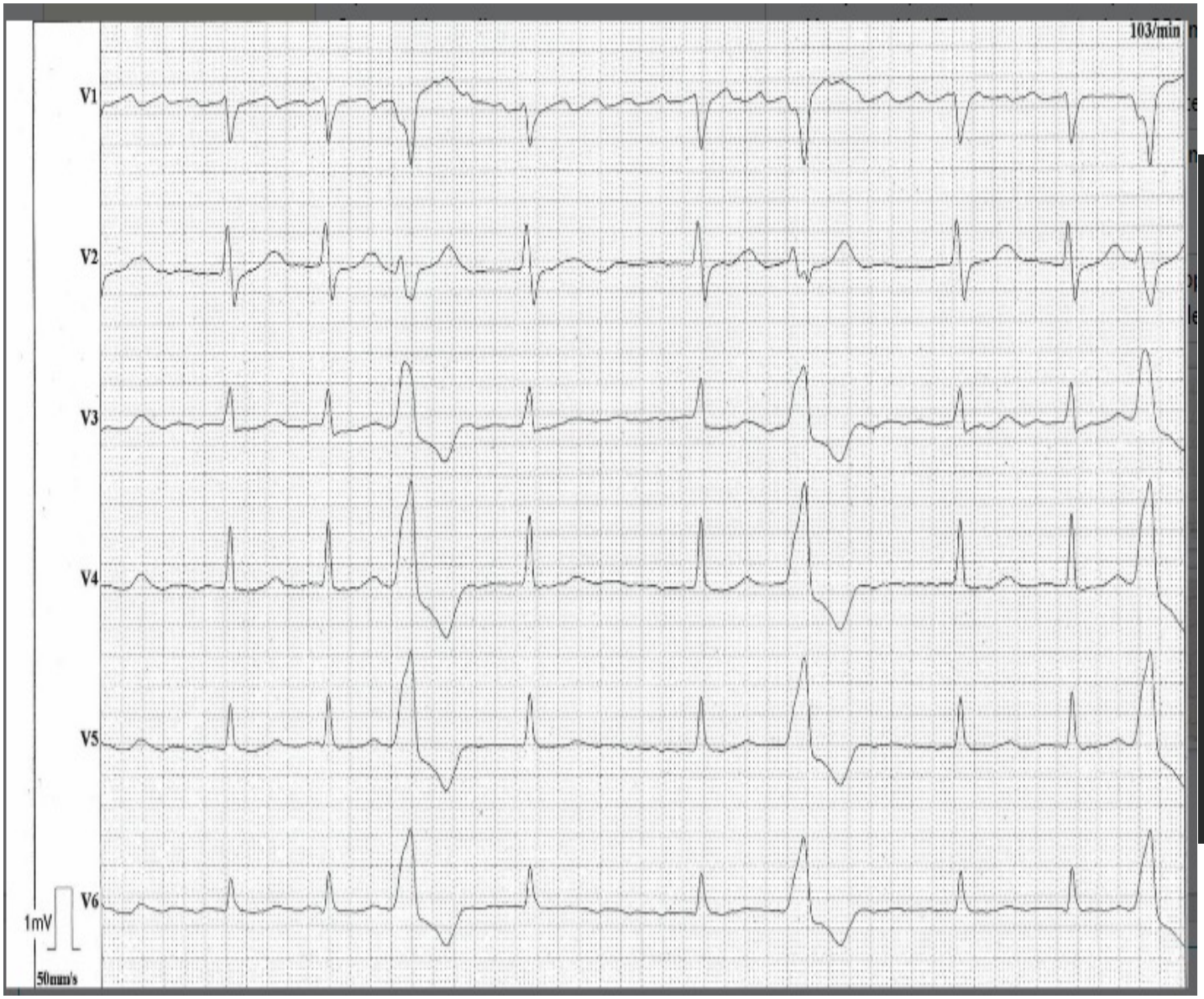
Regular sinus rhythm with a heart rate of approx. 120/min. Morphology and orientation of P waves is normal. PR interval within normal limits (approx. 180 ms). QRS axis normal, QRS duration normal (approx. 70 ms). QT interval normal (QT approx. 280 ms, QTc 396 ms). No evidence of ventricular hypertrophy, repolarization abnormalities, or ischemia.



ECG with tachycardic atrial fibrillation

6-lead ECG (paper speed: 25 mm/s)

Tachycardic atrial fibrillation with a heart rate of approx. 135 bpm, vertical heart, no relevant repolarization abnormalities.

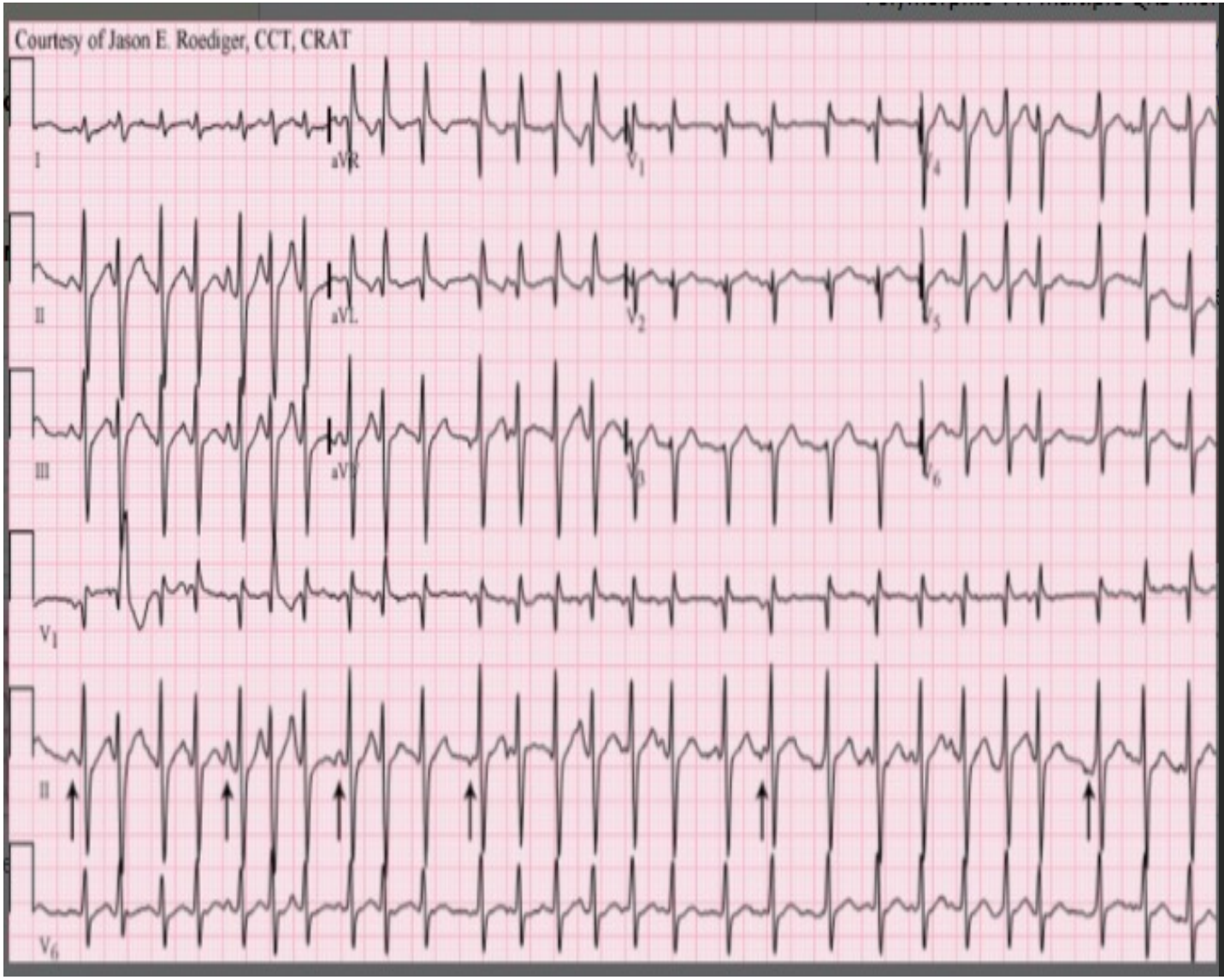


Atrial fibrillation and 2:1 extrasystole

6-lead ECG (paper speed: 50 mm/s)

Irregularly irregular rhythm with a ventricular rate of approx. 100/min.
No regular P waves are discernible (pronounced fibrillatory waves in V1).
2:1 extrasystole – monomorphic ventricular extrasystoles follow two normal QRS complexes.
No signs of ischemia.

Diagnosis: atrial fibrillation

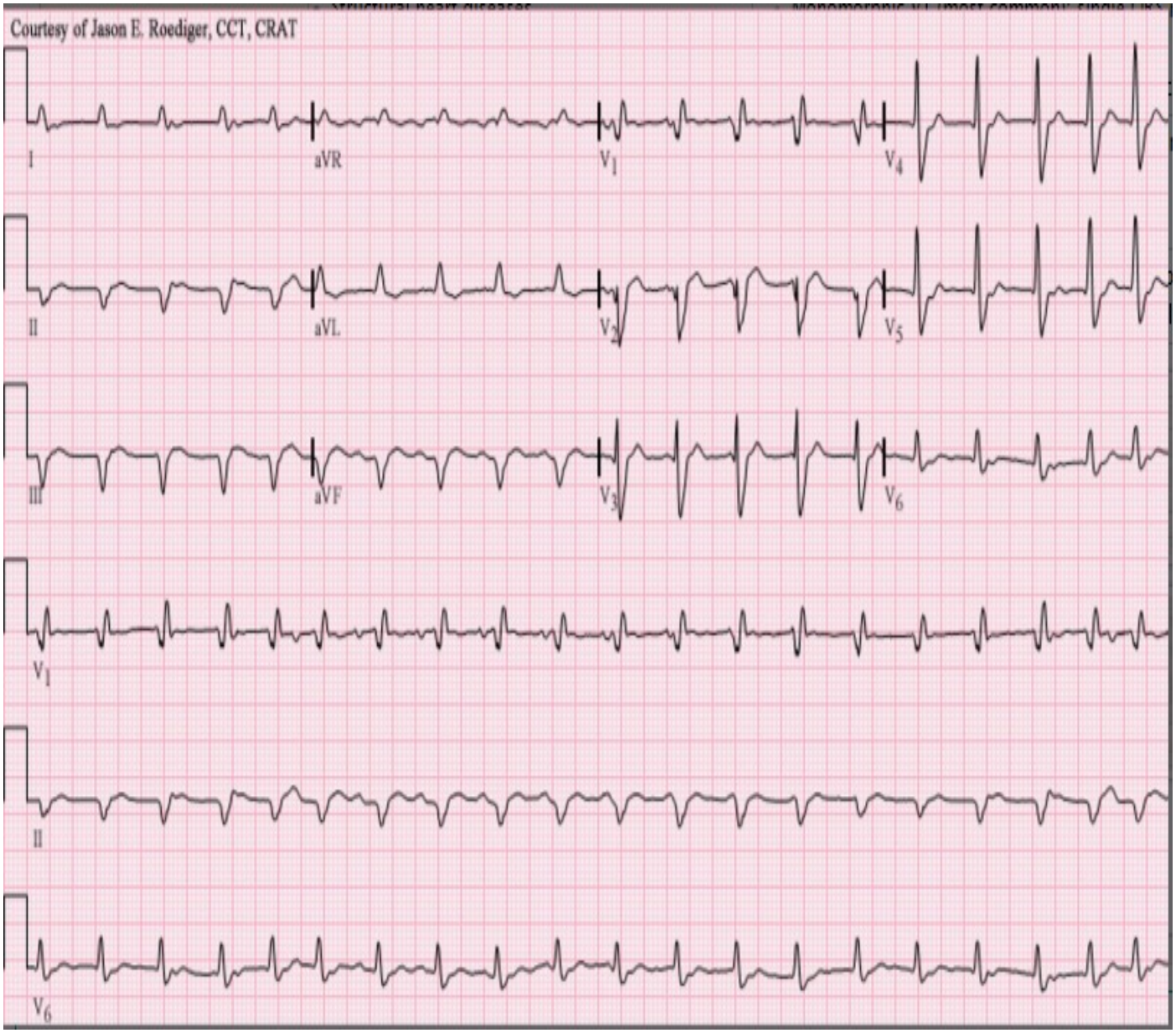


Multifocal atrial tachycardia

12-lead ECG (paper speed: 25 mm/s)

Irregularly irregular tachycardia with both atrial and ventricular rates of approx. 120–200/min (see examples of longer and shorter RR intervals in the overlay).
Left axis deviation (R > S in I, S > R in aVF).
P waves with different morphologies (including normal, biphasic, and inverted waves) in the same lead (arrows). P waves return to the isoelectric line (thus there is no atrial flutter/fibrillation).
PP, PR, RR, and QT intervals are variable. QRS length and morphology are largely normal. No signs of ischemia (however, interpretability is limited).

In summary: Irregularly irregular tachycardia with variable P-wave morphology, consistent with the diagnosis of multifocal atrial tachycardia.

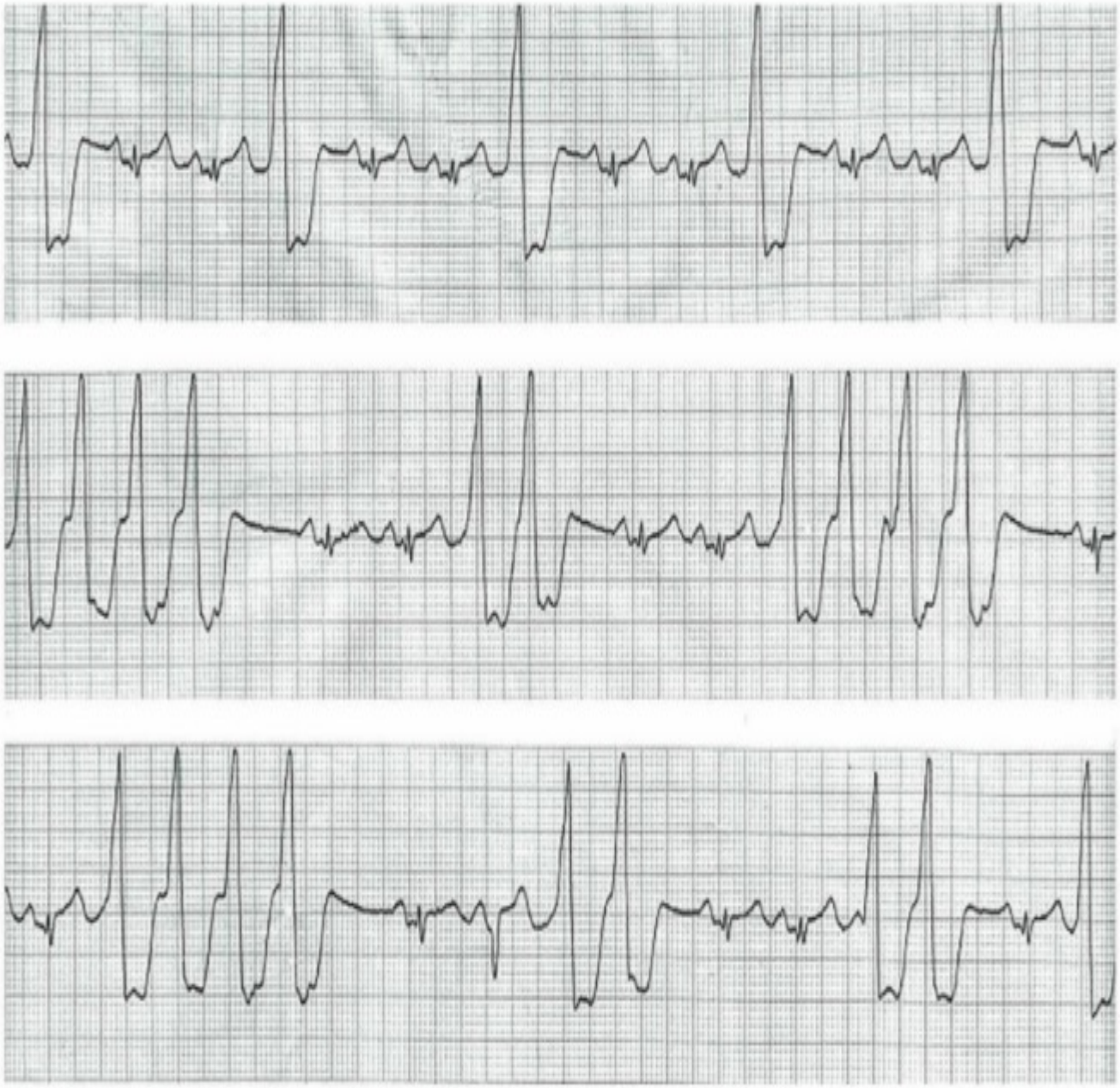


Junctional tachycardia

12-lead ECG (paper speed: 25 mm/s)

Ventricular rate of approx. 120/min
P waves occur before, during, and after the QRS complexes at a rate of approx. 105/min
Variable PR intervals, wide QRS complex (approx. 140 ms), normal QT interval (approx. 280 ms; QTc = 396 ms)
QS waves in inferior leads (II, aV_F, III), rsR' waves in V_1, slurred S waves in precordial leads (V_2–V_6)
Left axis deviation (R > S in I, S > R in aVF)
No evidence of ventricular hypertrophy

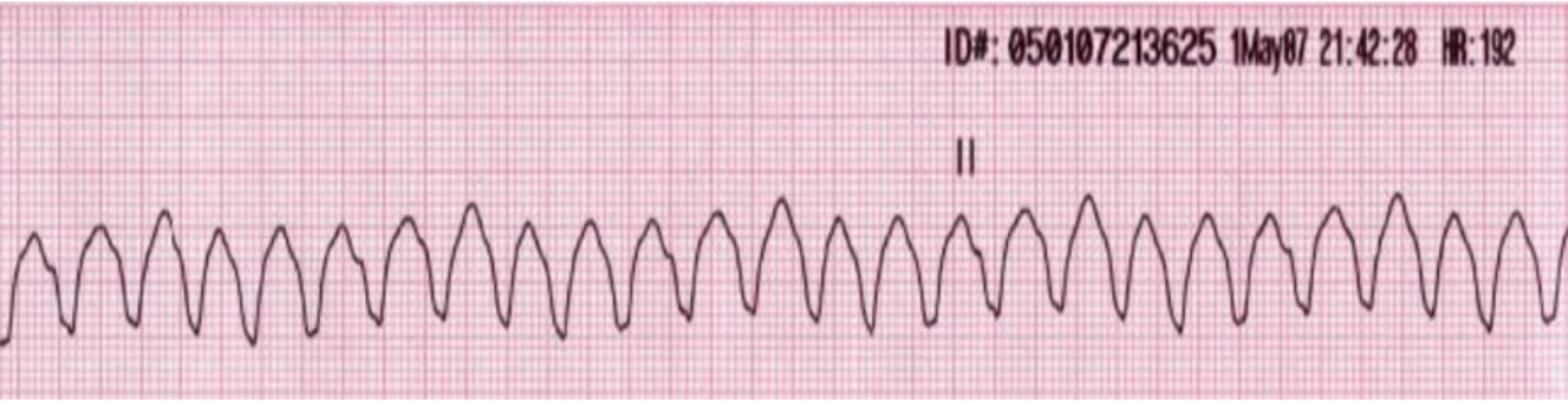
Impression: atrioventricular dissociation, junctional tachycardia (double tachycardia), right bundle branch block with left anterior fascicular block (bifascicular block), previous inferior wall infarct



Premature ventricular beats and trigeminy

Cardiac rhythm strip (paper speed: 25 mm/s)

- Sinus rhythm (rate approx. 95 bpm) with premature ventricular beats (PVB) and a wide QRS complex (approx. 300 ms)
- Top strip (trigeminy): a repeating pattern of one PVB following two sine beats
- Middle strip: two short runs of ventricular tachycardia interrupted by sinus rhythm and two PVBs (couplet)
- Bottom strip: two couplets following a short run of ventricular tachycardia

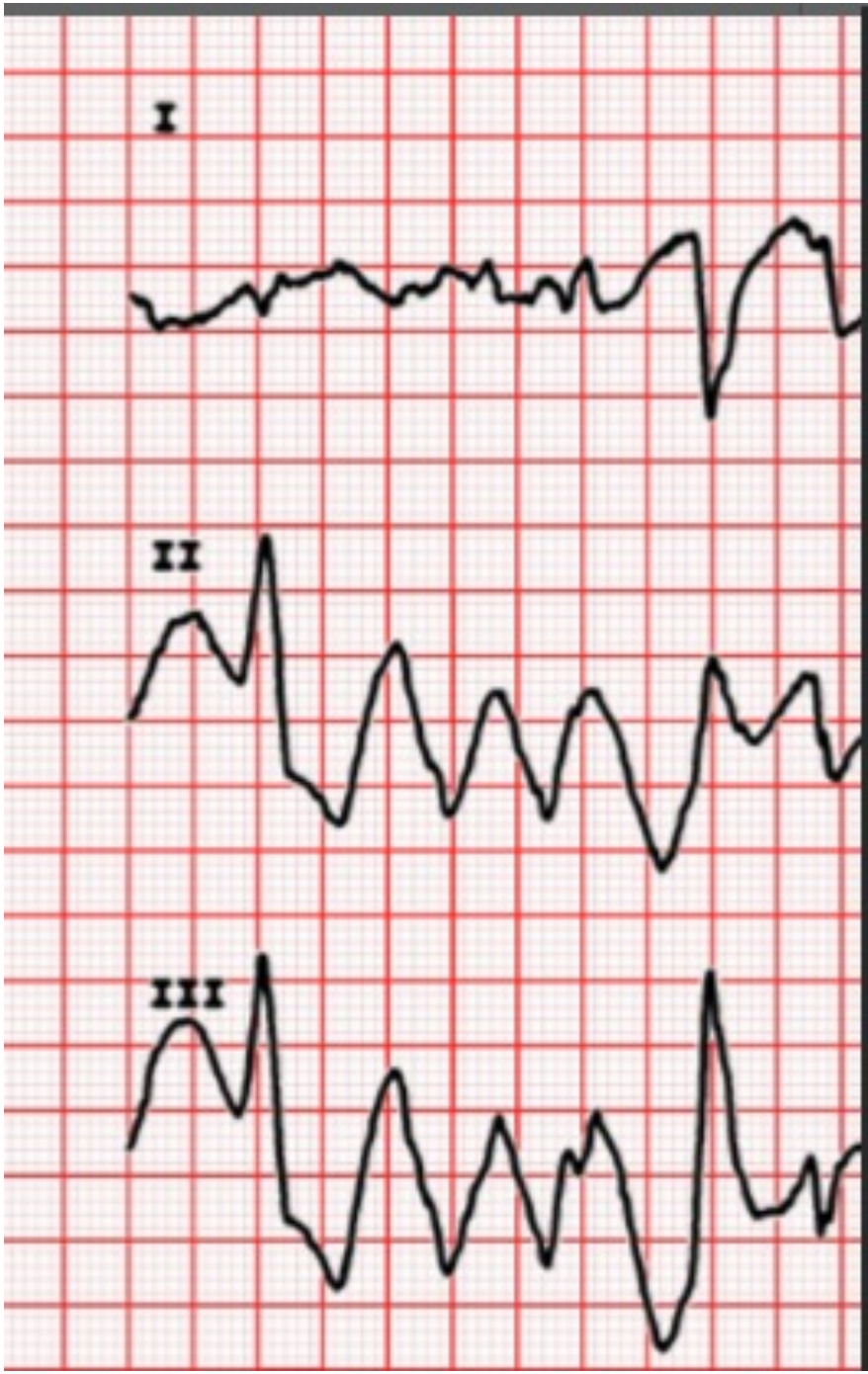


Monomorphic ventricular tachycardia

ECG (paper speed: 25 mm/s)

Broad QRS complexes (> 120 ms) that are monomorphic; ventricular rate of 192/min. Because of the high ventricular rate, P waves and T waves are not visible.

(Note: Tachycardia is considered "sustained" if it is present for at least 30 seconds. However, this strip does not show the full 30-second interval.)

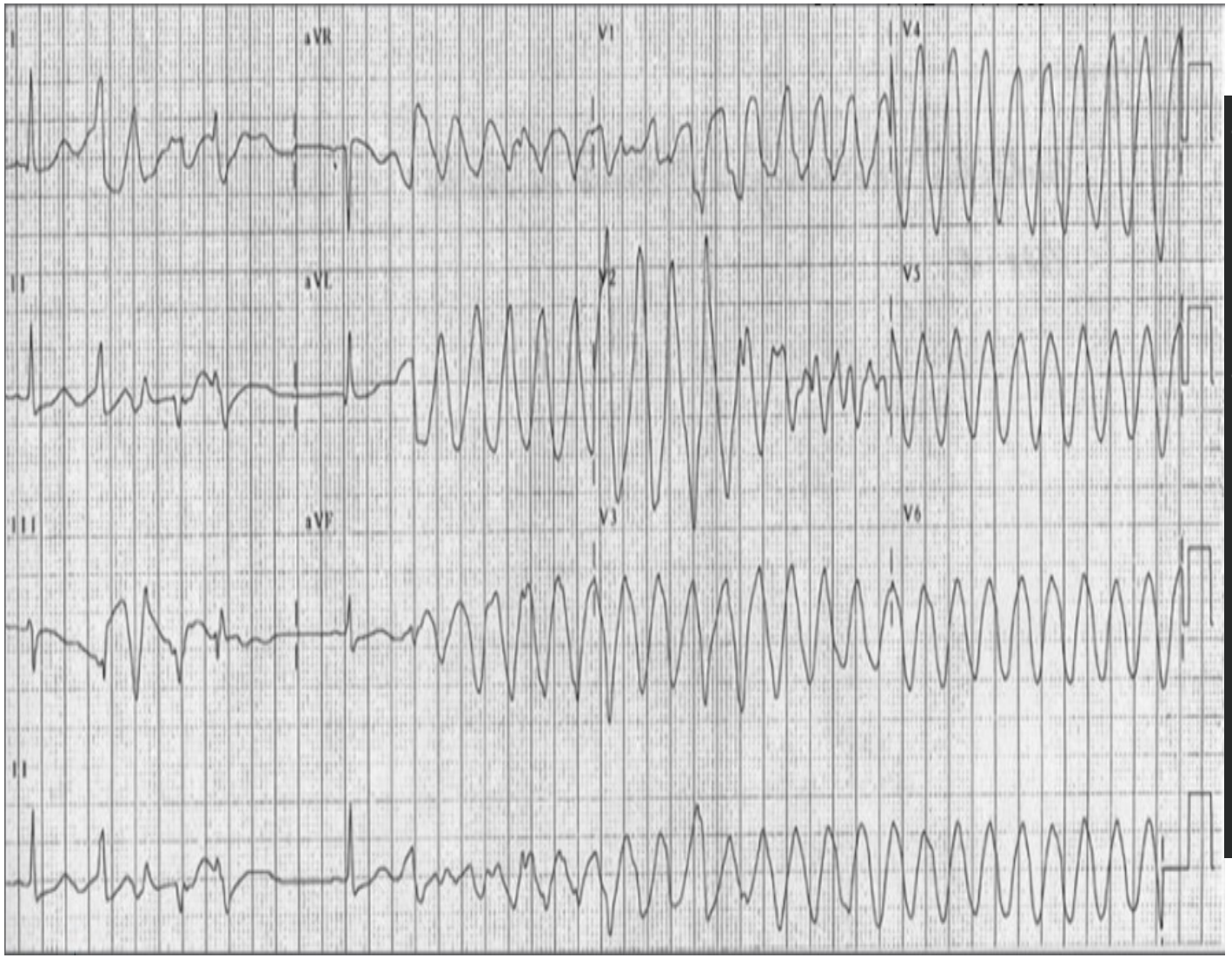


Polymorphic ventricular tachycardia

3-lead ECG (paper speed: 25 mm/s)

Irregularly irregular rhythm with a ventricular frequency of approx. 150–200/min.
No P or T waves are discernible.
Ventricular complexes occur at varying intervals and with varying but always abnormal morphology. The variance in QRS morphology separates this condition from monomorphic ventricular tachycardia.

Diagnosis: polymorphic ventricular tachycardia.

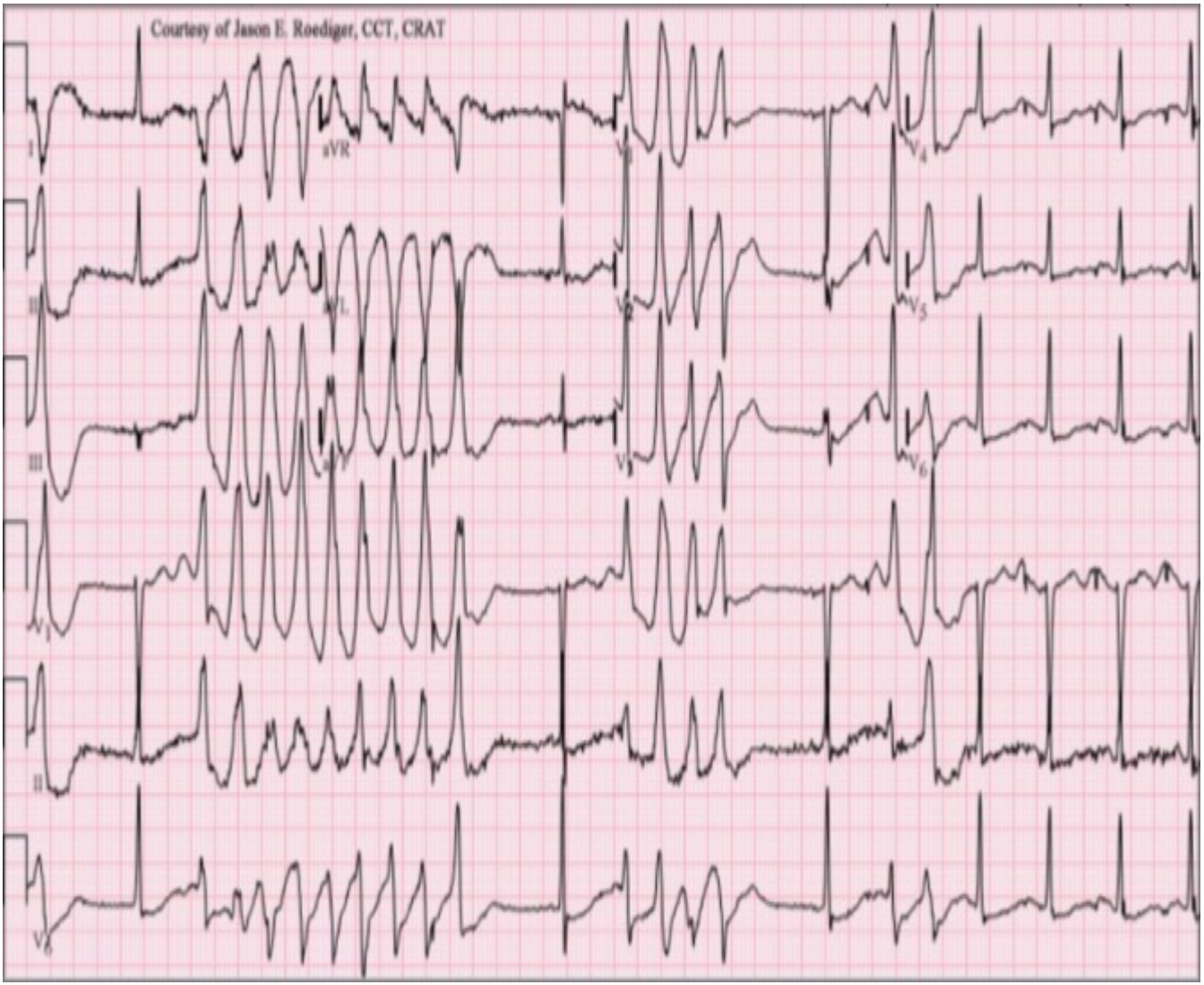


Torsade de Pointes tachycardia

12-lead ECG (paper speed: 25mm/s)

Conversion of sinus rhythm (P wave initially present; best seen in II) to Torsade de Pointes (TdP) tachycardia (QRS complexes twisting around the isoelectric line, producing a spindle-shaped pattern; best seen in V2) with a ventricular rate of approx. 200/min.

Note the R-on-T phenomenon (best seen in II): presumably due to a prolonged QT interval, the QRS complex falls into the latter half of the preceding T wave (vulnerable phase), eventually leading to TdP.

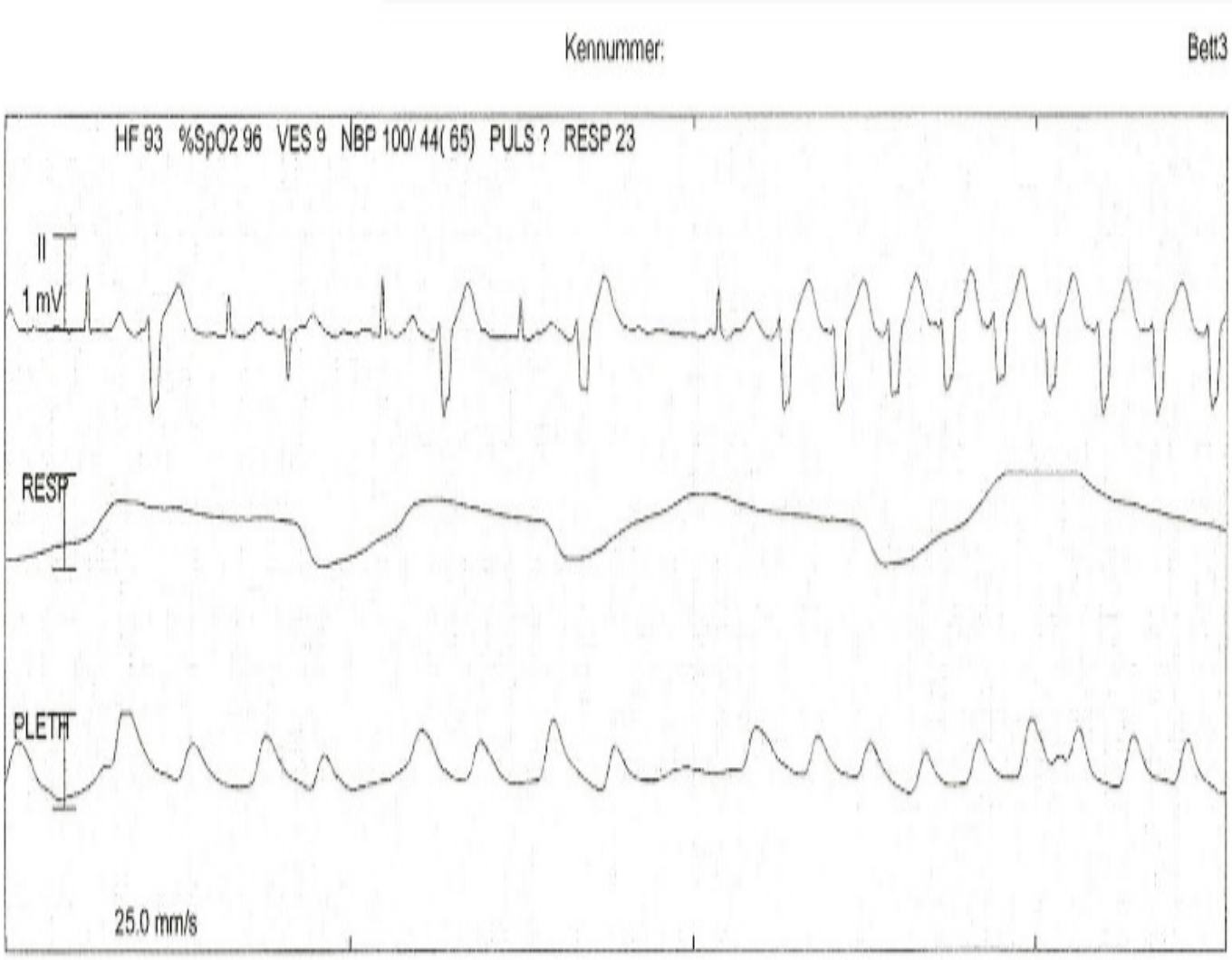


Torsade de Pointes tachycardia

12-lead ECG (paper speed: 25 mm/s)

P waves are initially present but are subsequently obscured by the high-frequency ventricular complexes (approx. 220/min) during the Torsade de Pointes (TdP) phases.
During TdP, QRS complexes twist around the isoelectric line and are borderline wide (approx. 120 ms). The QT interval (as measured after the second TdP phase in V6) is prolonged (approx. 440 ms; QTc = 568 ms).
Note the R-on-T phenomenon (best seen in V1).

Diagnosis: Torsade de Pointes tachycardia



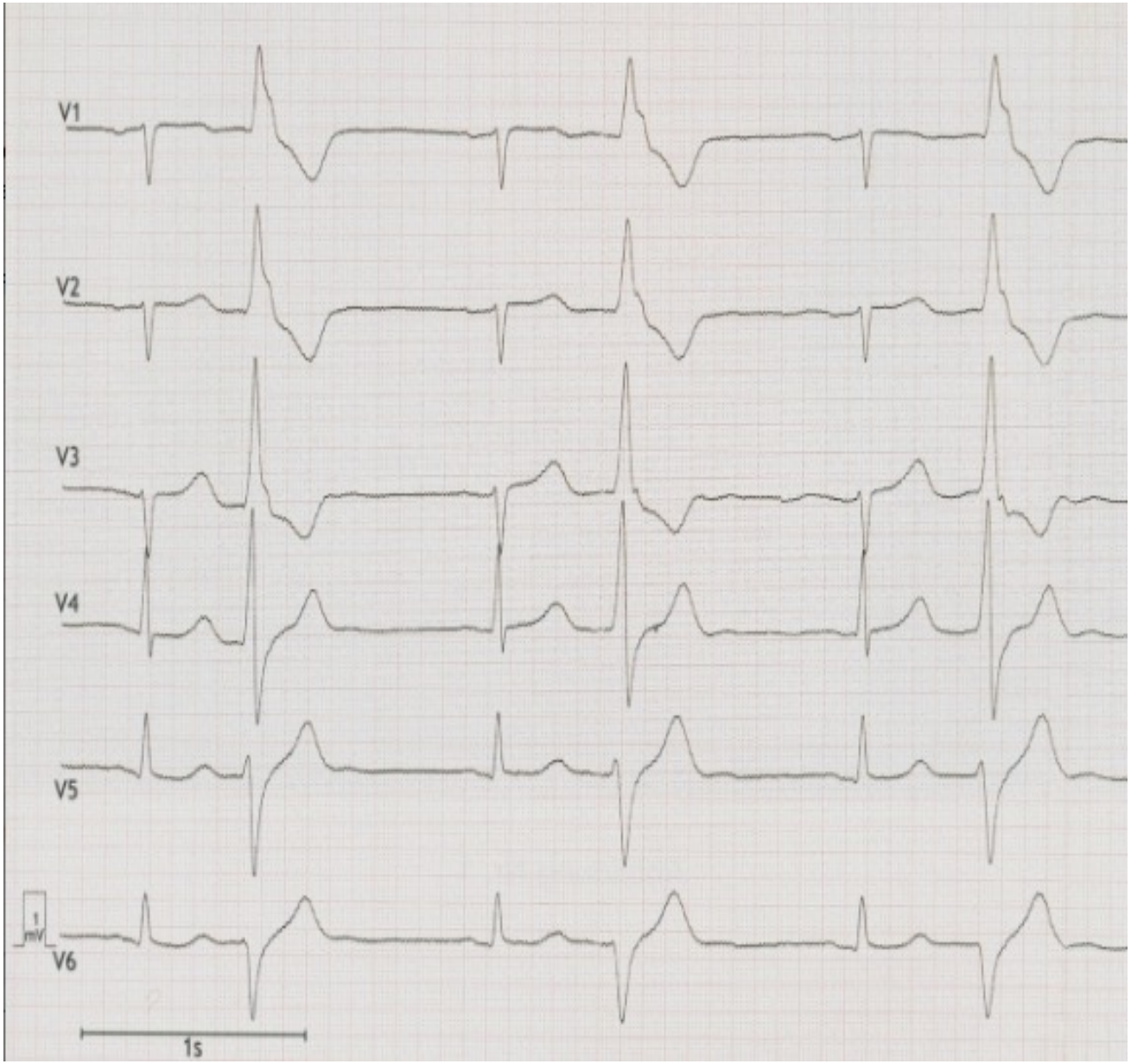
ECG with ventricular bigeminy and ventricular tachycardia

Findings:

1: ventricular bigeminy with a heart rate of approx. 100 beats/min.

2: ventricular tachycardia (9 successive PVC) with a heart rate of approx. 150 beats/min.

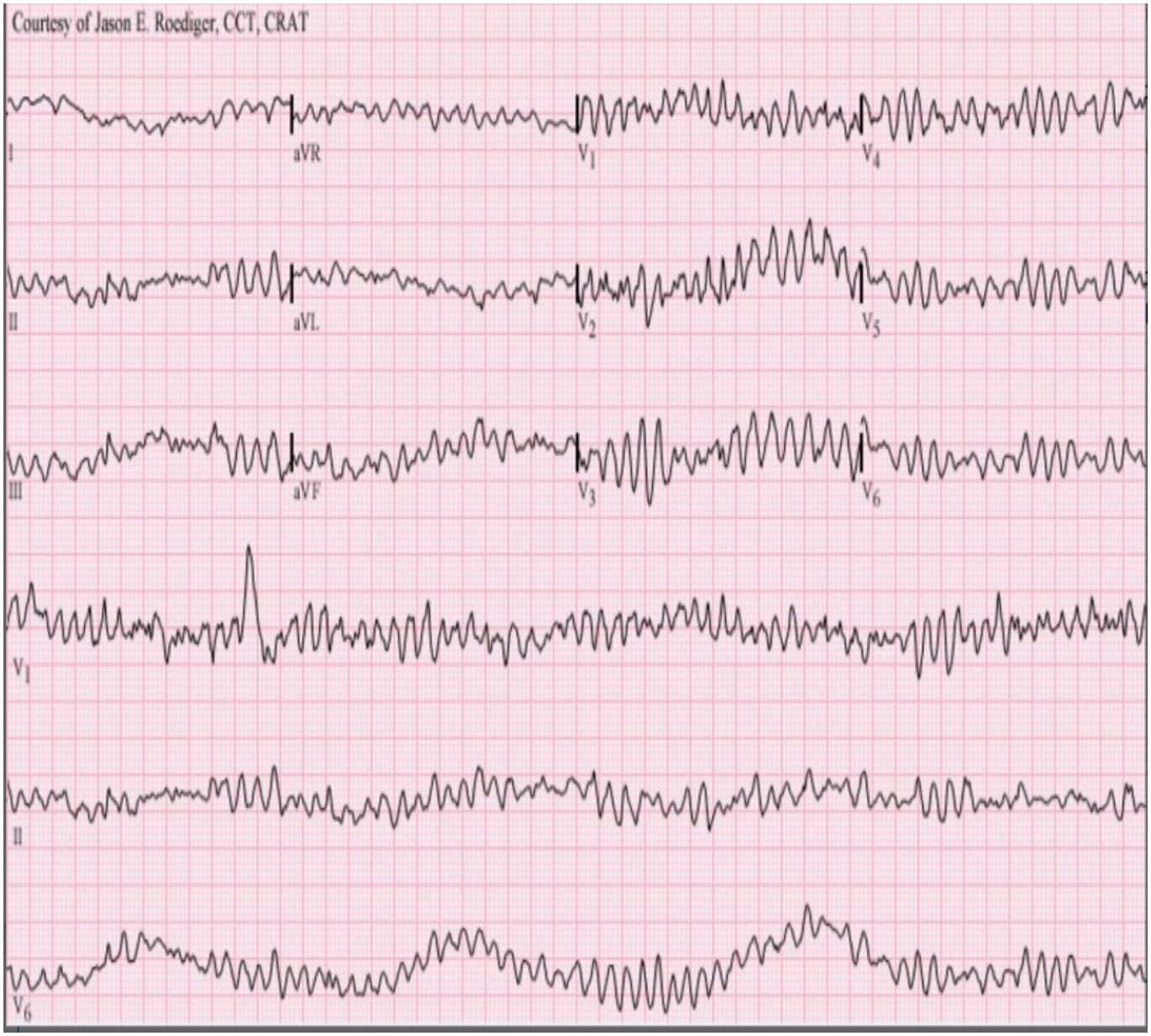
Axis can not be determined (only lead II is shown).



ECG with bigeminy

6-lead ECG (paper speed: 50 mm/s)

Sinus rhythm with a heart rate of approx. 75/min.
Ventricular bigeminy (with right bundle branch block morphology of the ventricular complexes; red markings) whose point of origin is most likely located in the left ventricle. As a result of the aberrant depolarization, there are repolarization abnormalities in the bigeminy contractions and in the branch blocks. Otherwise no repolarization abnormalities.



Ventricular fibrillation

12-lead ECG (paper speed: 25mm/s):

Tachyarrhythmia with a heart rate of approx. 375/min.
No discernible P waves, QRS complexes, or T waves. Absence of the normal isoelectric baseline.

Diagnosis: ventricular fibrillation

Atrial fibrillation

Summary

Atrial fibrillation (AF, Afib) is a common supraventricular tachyarrhythmia that is caused by uncoordinated atrial activation resulting in an irregular ventricular response.

A number of cardiac and noncardiac risk factors are associated with AF.

Patients are often asymptomatic but have an irregularly irregular pulse on physical examination. When present, symptoms usually include palpitations, lightheadedness, shortness of breath or features of embolic stroke. The relative stagnation of blood in the atria due to ineffective atrial emptying promotes clot formation, which in turn increases the risk of stroke and other thromboembolic complications.

The diagnosis is confirmed by an ECG that shows indiscernible P waves and a narrow but irregular QRS complex. A Holter monitor or event recorder is used to diagnose intermittent episodes. Echocardiography is useful in ruling out structural heart disease, and to detect the presence of atrial thrombi.

AF with symptoms of hemodynamic instability should always be treated with immediate synchronized cardioversion.

Treatment of AF among hemodynamically stable patients consists of anticoagulation therapy to prevent thromboembolic complications and the use of rate or rhythm control strategies to prevent the symptoms of AF and atrial remodeling. The need for anticoagulation therapy is determined based on the CHA_2DS_2-VAScscore.

Rate control therapy typically involves the use of beta-blockers or nondihydropyridine calcium channel blockers. Rhythm control strategies involve elective synchronized cardioversion and/or the use of antiarrhythmics such as flecainide, propafenone, ibutilide, dofetilide, or amiodarone.

Rate control therapy is usually preferred but the preferred treatment strategy may vary depending on the treatment center and the presence or absence of other comorbidities. Catheter directed or surgical ablation of the arrhythmogenic tissue is a newer modality used in refractory or severe AF.

Atrial flutter is another common supraventricular tachyarrhythmia that is usually caused by a single macroreentrant rhythm within the atria. The risk factors for atrial flutter are similar to those of AF. However, the atrial rate is slower, the QRS rhythm is usually regular, and characteristic saw-toothed P waves are seen on an ECG. Treatment is similar to that of AF, consisting of anticoagulation, and rate or rhythm control strategies. Atrial flutter frequently degenerates into atrial fibrillation.

Epidemiology

Most common sustained arrhythmia

Peak incidence: risk of AF increases with age

Etiolog: Risk factors for atrial fibrillation		
Cardiovascular risk factors	**Intrinsic cardiac disorders**	**Noncardiac disorders**
•Increasing age •Hypertension •Diabetes mellitus •Smoking •Obesity •Sleep apnea	•Coronary artery disease •Valvular heart disease (especially mitral valve disease) •Congestive heart failure (CHF) •Pre-excitation tachycardia (e.g., Wolff-Parkinson-White syndrome) •Sick sinus syndrome (tachycardia-bradycardia syndrome) •Cardiomyopathies •Pericarditis	•Pulmonary disease: COPD, pulmonary embolism, pneumonia •Hyperthyroidism •Catecholamine release and/or increased sympathetic activity • Stress: sepsis, hypovolemia, post-surgical state (especially following cardiac surgery), hypothermia • Pheochromocytoma • Cocaine, amphetamines •Electrolyte imbalances (hypomagnesemia, hypokalemia) •Drugs: e.g., adenosine, digoxin •Holiday heart syndrome: irregular heartbeat classically triggered by excessive alcohol consumption, but also sometimes by moderate consumption, stress, or lack of sleep •Chronic kidney disease

Mechanisms include:
Volume overload, hemodynamic stress
→ atrial hypertrophy and/or dilatation → atrial fibrosis
Atrial ischemia
Inflammation of the atrial myocardium
Altered ion conduction by the atrial myocardium

In approx. 15% of cases, AF occurs in the absence of any of the above risk factors (idiopathic / lone AF).

Classification

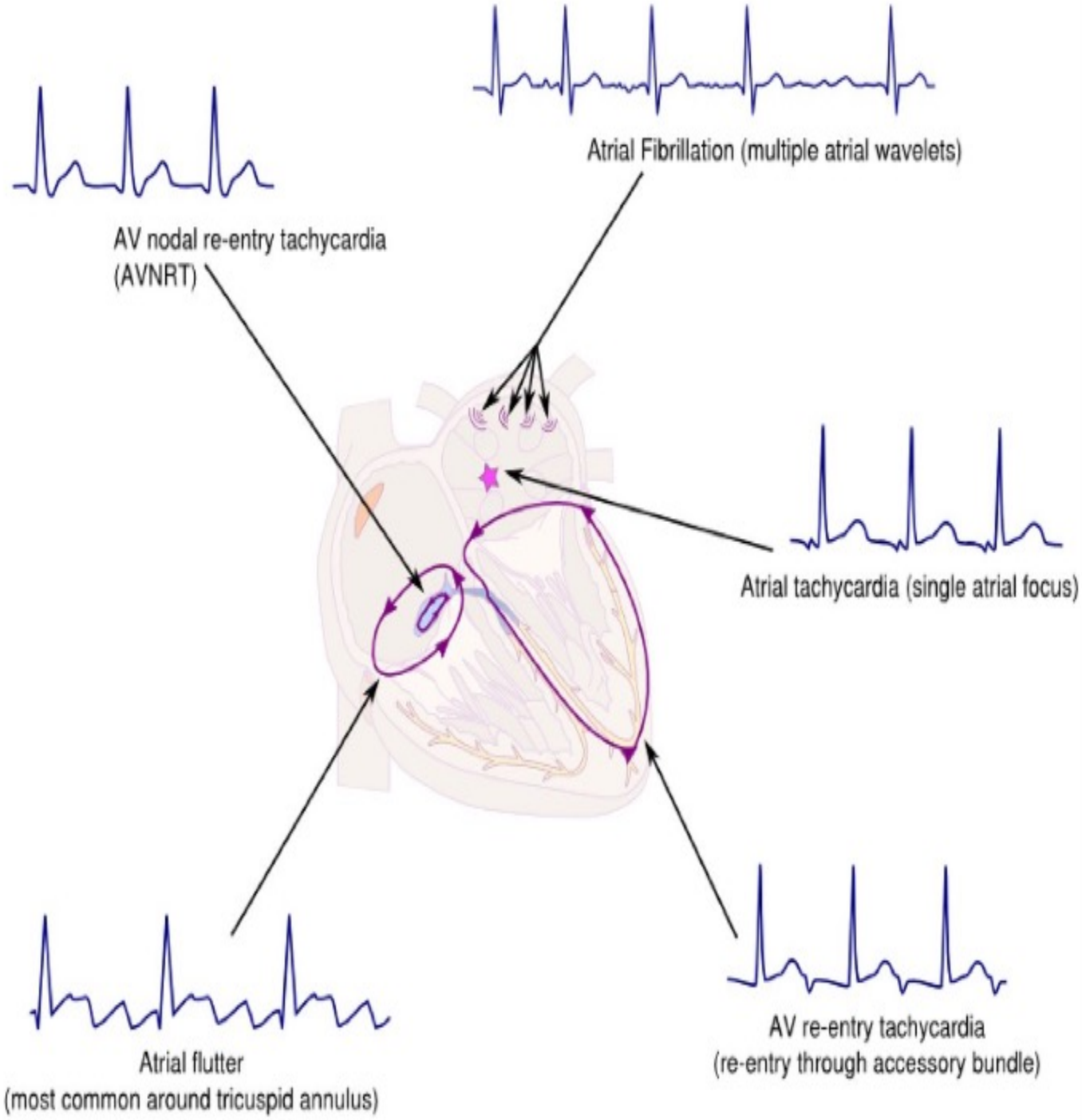
Classification criteria		Definition
Hemodynamic stability	Unstable AF	AF patients who present with chest pain, acute pulmonary edema, hypotension and/or other signs of shock
	Stable AF	AF patients who are hemodynamically stable
Heart rate	AF with rapid ventricular response	AF with a ventricular rate > 100 bpm (tachycardic AF)
	Slow AF	AF with a ventricular rate < 60 bpm (bradycardic AF)
Onset and duration of AF	New-onset AF	AF less than 48 hours in duration
	Paroxysmal AF	AF that terminates within 7 days of onset either following treatment or spontaneously
	Persistent AF	Continuous AF for > 7 days and less than permanent
	Long-standing persistent AF	Continuous AF for > 1 year (may be termed chronic)
	Permanent AF	Long-standing persistent AF that is not treated following a joint decision by the patient and the physician (chronic AF)
Mitral valve involvement	Valvular atrial fibrillation	AF in patients with mitral valve stenosis, artificial heart valves, and/or repaired mitral valves
	Non-valvular atrial fibrillation	AF in patients without mitral valve involvement → moderately elevated risk of thromboembolic events

Mitral valve involvement should always be assessed in patients with AF!

Pathophysiology

- Atrial fibrillation is a **supraventricular arrhythmia**.
- AF is initiated by
    - Bursts of electrical activity from automatic foci, or in diseased, **fibrosed atrial tissue**
    - Pre-excitation of the atria as a result of aberrant pathways (e.g., Wolff-Parkinson-White syndrome)
    - Re-entry rhythms are more likely to occur with enlarged atria, diseased heart tissue, and/or aberrant pathways (e.g., Wolff-Parkinson-White syndrome).

- Effects of AF
  - The **atria contract rapidly but ineffectively** and in an uncoordinated fashion → stasis of blood within the atria → risk of **thromboembolism**
  - **Irregular activation of the ventricles** by conduction through the AV node → tachycardia



Pathophysiology of supraventricular tachycardias

Overview of common types of supraventricular tachycardia, including sites of stimulus origin, conduction pathways and resulting ECG patterns.

Clinical features

Most patients are asymptomatic

Less commonly, symptoms of arrhythmias such as palpitations, dizziness, syncope, fatigue, and or dyspnea

Symptoms of the underlying disease (e.g., murmurs of mitral stenosis)

Tachycardia with an **irregularly irregular** pulse

Complications of long-standing AF

- **Acute left heart failure → pulmonary edema**
- **Thromboembolic events**: **stroke/TIA**, renal infarct, splenic infarct , intestinal ischemia , acute limb ischemia
- **Life-threatening ventricular tachycardia**

Patients with atrial fibrillation may be asymptomatic for long periods of time!
The brain, kidney, and spleen are the three organs most likely to be damaged by emboli!

Diagnostics

ECG (initial investigation)

- **Irregularly irregular RR intervals**
- **P-waves are indiscernible**
- **Tachycardia**
- **Narrow QRS complex (< 0.12 seconds)**

In a patient, who presents with risk factors for AF and symptoms suggestive of arrhythmias,
Holter ECG monitoring may be used to
detect paroxysmal AF.

Echocardiography

- **Transthoracic echocardiogram (TTE)**
  - Indications: all patients with new-onset AF
  - Assesses cardiac function and rule out underlying structural cardiac disease, e.g., mitral valve stenosis
- **Transesophageal echocardiogram (TEE)**
  - Indications: patients with AF or atrial flutter > 48 hours or of unknown duration for whom electrical or pharmacological cardioversion is planned, but who have not received anticoagulation therapy for at least 3 preceding weeks

Visualizes the atria and the left atrial appendage (hotspots for thrombogenesis) to identify thrombi before attempting cardioversion
Further assesses heart function and rules out underlying structural disease

Laboratory tests: to identify underlying risk factors for AF

Troponin levels: to rule out myocardial infarction

D-dimer levels: if risk factors (e.g., DVT) or clinical features of pulmonary embolism are present

Brain-natriuretic peptide (BNP): to identify heart failure

CBC: to identify anemia, infection

TSH, fT_4: to screen for hyperthyroidism

Serum electrolytes (Na^+, K^+, Mg^{2+}, and Ca^{2+}): to identify electrolyte imbalances

BUN, serum creatinine: to identify chronic kidney disease

Ethanol levels, digoxin levels and/or urine toxicology (e.g., cocaine, amphetamines)

Differential diagnoses

The differential diagnosis of AF include other tachyarrhythmias. See specifically "supraventricular tachyarrhythmias", which typically have a **narrow QRS complex**.

Treatment

The general principles of treating atrial fibrillation include:

1. **Correcting reversible causes and/or treatable conditions** (e.g., hyperthyroidism, electrolyte imbalances)
2. **Controlling heart rate and/or rhythm**
3. **Providing anticoagulation**

Controlling heart rate and/or rhythm

- **Unstable AF**: **emergent electrical cardioversion**
- **Stable AF**: rate control or rhythm control strategies to control AF and prevent long-term recurrence .

Treatment strategy	Rate control	Rhythm control
Goal and rationale	• Normalizing the ventricular heart rate	• Terminating atrial fibrillation and restoring it to sinus rhythm in order to prevent atrial remodeling
Indications	• Elderly patients	• Failure of rate-control strategy to control symptoms • Younger patients
Contraindications	• AF due to pre-excitation syndromes	• Long-standing persistent AF

Treatment strategy		Rate control	Rhythm control
Therapeutic measures	1st line	• 1st choice: beta blockers (esmolol, propanolol, metoprolol) OR nondihydropyridine calcium channel blockers (diltiazem, verapamil) • 2nd choice: digoxin • 3rd choice: dronedarone, amiodarone	• 1st choice: elective electrical cardioversion • 2nd choice: pharmacologic cardioversion with antiarrhythmic drugs such as flecainide, propafenone, ibutilide, dofetilide • See prerequisites for cardioversion of AF below
	2nd line (ablative procedure)	• AV nodal ablation and implantation of a permanent ventricular pacemaker	• Catheter-based radiofrequency ablation of atrial tissue around pulmonary vein openings (pulmonary vein isolation)

Patients with unstable AF should be treated with immediate cardioversion!

Anticoagulation

Prerequisites for cardioversion of AF

- **New onset AF (< 48 hours) in patients with:**
  - Low thromboembolic risk → consider anticoagulation directly before or after cardioversion
  - High thromboembolic risk → start anticoagulation immediately before or after cardioversion
  - Anticoagulation options: **IV heparin or LMWH**, direct thrombin inhibitors (e.g., **dabigatran**), or factor Xa inhibitors (e.g., **rivaroxaban, apixaban**)

- **AF ≥ 48 hours or of unknown duration in patients with:**
  - **Unstable AF (require urgent cardioversion)**: **IV heparin or LMWH** immediately before cardioversion followed by **warfarin for up to 4 weeks** after cardioversion
    - TEE to rule out atrial thrombi recommended if anticoagulation has not been administered at least 3 week prior to cardioversion
  - **Stable AF (do not require urgent cardioversion)**: **warfarin** with bridging therapy for 3 weeks before and up to 4 weeks after cardioversion
- Anticoagulation therapy should be considered in all patients who are about to undergo cardioversion.

Anticoagulation therapy should be considered in all patients who are about to undergo cardioversion.

Long-term anticoagulation

Indications for long-term anticoagulation

• Nonvalvular atrial fibrillation: The need for anticoagulation therapy is based on the CHA2DS2-VASc score
  • Score = 0: no anticoagulation
  • Score = 1: no anticoagulation OR treatment with oral anticoagulants
  • Score ≥ 2: oral anticoagulation with either warfarin or newer oral anticoagulants (dabigatran, rivaroxaban, apixaban)
• Valvular atrial fibrillation: anticoagulation with warfarin is required regardless of the CHA2DS2-VASc score

CHA2DS2-VASc score		
Acronym	Risk factor	Points
C	CHF or left-sided heart failure	1
H	Hypertension	1
A2	Age ≥ 75	2
D	Diabetes Mellitus	1
S2	Stroke or TIA or thromboembolism	2
V	Vascular disease (prior MI, peripheral artery disease, or aortic plaque)	1
A	Age 65–74	1
Sc	Sex category (female sex)	1
CHA2DS2-VASc scores of 1, 2, 3, 5, and ≥6 carry an annual stroke risk of approx. 1%, 2%, 3%, 7%, and >9% respectively.		

Long term anticoagulation for patients with AF in order to prevent thromboembolic complications is indicated if the patient has an underlying valvular disease and/or a $\underline{CHA_2DS_2\text{-}VASc}$ score ≥ 2

The risk of bleeding due to anticoagulation should always be taken into consideration when initiating anticoagulation therapy.

Atrial flutter

- Atrial flutter is a **supraventricular tachyarrhythmia** that is usually caused by a single macroreentrant rhythm within the atria.
- **Epidemiology**
  - Sex: ♂ > ♀ (5:2)
  - Peak incidence: risk of atrial flutter increases with age
- **Etiology**: similar to atrial fibrillation (see "Etiology" above)
- **Clinical features**
  - **Most patients are asymptomatic**
  - Less commonly, symptoms of arrhythmias such as palpitations, dizziness, syncope, fatigue, and or dyspnea
  - Symptoms of the underlying disease (e.g., murmurs of mitral stenosis)
  - Tachycardia with a **regular pulse**

- **Diagnostics** (similar to atrial fibrillation)
  - **Sawtooth appearance of P waves** (flutter waves or F waves), at a rate of ~ 300 bpm
  - Regular, narrow QRS complexes
- **Treatment**: similar to atrial fibrillation .
- **Complications**:
  - Frequently degenerates into **atrial fibrillation** .
  - 1:1 conduction leading to life-threatening **ventricular tachycardia**

Suggested approach for management of tachycardias :

Narrow complex tachycardias

- Regular (supraventricular tachycardia [SVT])
  - Sinus tachycardia
    - Physiological response to insult. Impulse originates from sino-atrial (SA) node.
  - Atrial tachycardia
    - Aberrant atrial focus producing impulse independent of SA node
  - Atrioventricular nodal re-entry tachycardia (AVNRT)
    - Re-entry circuit within or near AV node
  - AV re-entry tachycardia (AVRT)
    - Re-entry circuit conducted from atria to ventricles via abnormal accessory pathway; usually due to Wolff-Parkinson-White (WPW) syndrome
  - Atrial flutter with regular AV block (eg 2:1, 3:1)
    - Re-entry circuit within the atria
- Irregular
  - Atrial fibrillation (AF)

    Atria twitch instead of beating in a coordinated manner

- Narrow complex tachycardias (QRS duration <0.12 s)
  - Regular: likely SVT
    - Attempt vagal manoeuvres
      - Valsalva (e.g : ask patient to blow); carotid sinus massage.
        - If this fails then:
    - Adenosine 6 mg IV
      - Rapid bolus ideally into a large-bore cannula in the antecubital fossa
      - Warn patients of transient unpleasant side effects: flushing, nausea and chest tightness, ‘feeling of impending doom’
      - Avoid in patients with asthma, WPW syndrome.
      - Caution in taking theophylline, dipyridamole.
      - If 6mg unsuccessful:

- Adenosine 12 mg IV
  - If first 12mg unsuccessful:
- Further adenosine 12 mg IV
- If adenosine is contraindicated, consider verapamil 2.5-5.0 mg IV, or flecainide 2 mg/kg IVI over 20-30 min if no evidence of structural heart disease or amiodarone.

- Irregular: likely AF
  - Onset <48 hours
    - Aim for rhythm control
      - Flecainide 2 mg/kg IVI over 20-30 min if no evidence of structural heart disease or amiodarone 300 mg IV over 20-30 min and 900 mg over 24 hours if flecainide contraindicated
      - Anticoagulate with enoxaparin 1.5 mg/kg subcutaneous (SC) prior to this

- Onset >48 hours
  - Aim for rate control
    - Metoprolol 5 mg IV OR bisoprolol 5 mg orally (PO) OR verapamil 5 mg IV
      - If signs of heart failure try digoxin 0.5 mg IVI over 30-60 min
      - Digoxin can be added to the above if beta-blockade unsuccessful
    - Anticoagulate with enoxaparin 1.5 mg/kg subcutaneous (SC) .

Broad complex tachycardias :

- Regular
  - Ventricular tachycardia (VT)
    - Generated by a single ventricular focus
  - SVT with bundle branch block (BBB)
    - This is rare. Any broad complex tachycardia should be treated as VT unless there the patient has an old ECG with clear previous bundle branch block of unchanged morphology.
- Irregular
  - Polymorphic VT (Torsades de pointes)
    - Sinusoidal morphology usually due to abnormal ventricular repolarisation (long QT)
  - AF with bundle branch block

- In broad complex tachycardias (QRS duration <0.12 s):
  - If regular:

- If likely monomorphic VT
  - Give amiodarone 300 mg IVI over 20-30 min followed by amiodarone 900 mg IVI over 24 hours
  - Any broad complex tachycardia should be treated as VT unless there the patient has an old ECG with clear previous bundle branch block of unchanged morphology.
- If definitely SVT with BBB
  - Try adenosine as for regular narrow complex tachycardias
- Irregular
  - If likely AF with BBB
    - Treat as for irregular narrow complex tachycardias
  - If likely polymorphic VT (Torsades de pointes)
    - Magnesium 2 g IV over 10 min
    - Stop any medications which prolong the QT interval
    - Correct any electrolyte abnormalities if not already done so, and give

- **Further management of tachycardia**
- Request 12 lead ECG once back in sinus rhythm
  - Look specifically for ischaemic changes (ST elevation, ST depression and T wave inversion), prolonged QT interval (QT_c >440 ms) and signs of WPW syndrome (shortened PR interval, delta wave and broad QRS complex) and identify and correct any underlying cause .

Cardioversion in tachyarrythmias :

- **If adverse features are present [shock, syncope, myocardial ischaemia, heart failure], prepare for emergency synchronised DC cardioversion .**
- **Under general anaesthesia or conscious sedation**
  - Once ready, warn all those nearby to stand clear and remove any oxygen delivery device whilst the defibrillator is set to synchronised mode and charged to 120 J
  - Once the defibrillator is charged and all are clear, deliver the shock
  - Should this fail, two subsequent shocks at increasing increments may be tried
  - Should this fail, give a loading dose of amiodarone 300 mg IV over 10-20 minutes and repeat DC cardioversion followed by amiodarone 900 mg IV over 24 hours