ECG-Quiz

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Unstable angina

86-year-old patient with unstable angina.

Personal history
Paroxysmal supraventricular tachycardia.

Drugs before admission
Aspirin 100 mg/d. Verapamil 120 mg 2x/d.

Current admission
Recurrent episodes of typical chest pain lasting for up to 15 min at rest within the last 24 hours.

Clinical findings
68 kg, 168 cm, BMI 26.56 kg/m2, BP 146/90 mmHg, HR 62 bpm. RR 16. No signs of congestive heart failure.
Q1: Does this patient have an acute coronary syndrome (myocardial ischemia)?
Q1: Does this patient have an acute coronary syndrome (myocardial ischemia)?

A: Yes

B: No, ventricular preexcitation with pathological repolarization

C: No, T wave memory after an antidromic circus movement tachycardia (= AVRT)

D: No, myocarditis

E: No, hypertrophic cardiomyopathy
**Unstable angina**

**ECG:** SR, HR 59 bpm. PQ 160ms, QRS 90ms, QT 500ms. Left axis deviation. Partial RBBB. Deep symmetric negative T waves in V3-V6 (arrows). **Diagnosis:** Ischemia due to an occlusion of the proximal LAD.

Cardiology, STZ, 2014.
Unstable angina

Coronary angiography: Critical stenosis of the LAD (arrows).

Cardiology, STZ, 2014.
Unstable angina

Wellens’ syndrome
Dynamic symmetric T-wave inversion in the precordial leads and QTc prolongation in the setting of acute coronary syndrome caused by a critical stenosis of the left anterior descending coronary artery (LAD).

Pathophysiology
Myocardial edema in the anterior and apical segments of the left ventricle.

Differential diagnosis
• Critical proximal LAD stenosis
• LAD vasospam
• Tako tsubo syndrome
• Intracranial bleeding

T2-weighted MRI for edema during acute phase.

T2-weighted CE-MRI at 6-8 weeks follow-up showing disappearance of the edema.

Acute chest pain

57-year-old lady with new onset chest pain.

**Personal history**
Risk factors: nicotine (40 py), family history (SCD in a 60 year-old brother).

**Drugs before admission**
None.

**Current admission**
First episode of massive chest pain.
Direct admission to the cath lab by the ambulance after on site ECG recording.

**Clinical findings**
49 kg, 154 cm, BMI 20.7 kg/m2, BP 90/60 mmHg, HR 74 bpm. RR 24.
Q2: Does this patient have an acute coronary syndrome (myocardial ischemia)?
Acute chest pain

Q2: Does this patient have an acute coronary syndrome (myocardial ischemia)?

A: Yes
B: No, acute pulmonary embolism
C: No, acute bronchial asthma
D: No, myocarditis with RBBB due to cardiac sarcoidosis
E: Not diagnostic ECG due to the (possibly preexisting) RBBB

Cardiology, STZ, 2014.
Acute chest pain

ECG: SR, HR 85 pm. RBBB. ST segment elevation in V1-3, aVL. ST segment depression in V4-6, aVF.
Diagnosis: Anterior myocardial infarction with periinfarction RBBB.
Coronary angiography: 90% stenosis of the left main (arrow head) and occlusion of the proximal LAD (open arrows).
Blood supply of the conduction system

**Sinus node**
55% RCA, 45% RCX

**AV-node and proximal His**
RCA

**Distal His**
Right bundle branch
Left anterior fascicle
Septal perforators of the LAD

**Left posterior fascicle**
Septal perforators of the LAD
RCA

---

Signs of myocardial infarction

1. ST segment elevation in 2 contiguous leads and reciprocate ST segment depression

2. Wide symmetric T waves

3. Negative symmetric T waves in the precordial leads

4. Loss of the R wave in the precordial leads

5. New onset right bundle branch block (RBBB)

Cardiogenic shock

68-year-old lady with new onset chest pain and syncope.

**Personal history**
Mastectomy because of breast cancer, late recurrence with bone metastasis.

**Drugs before admission**
None.

**Current admission**
New onset chest pain followed by syncope.

**Clinical findings**
GCS 9-10, BP 60/40 mmHg, HR 45 bpm.
Q3: Why is this patient in cardiogenic shock?
Q3: Why is this patient in cardiogenic shock?

A: Complete AV block and insufficient escape rhythm
B: Inferior myocardial infarction with mechanical complication
C: Inferior myocardial infarction with complete AV block
D: Cardiac metastasis with pericardial effusion
E: Not diagnostic ECG because of the missing lead V6
**Diagnosis:** Infero-posterior STEMI with complete AV block. aCL 600ms, aHR 100 bpm, vCL 1480ms, vHR 40 bpm. Complete AV block. Lead V6 is missing.
Diagnosis: Inferior STEMI with complete AV block.

Cardiology, STZ, 2012.
Cardiogenic shock

ECG after successful revascularisation of the RCA.

Cardiology, STZ, 2012.
AV conduction disturbances in acute MI

<table>
<thead>
<tr>
<th>Infarct location</th>
<th>Inferoposterior</th>
<th>Anterior</th>
</tr>
</thead>
<tbody>
<tr>
<td>Culprit coronary artery</td>
<td>Proximal RCA</td>
<td>Proximal LAD</td>
</tr>
<tr>
<td>Escape rhythm</td>
<td>Narrow or wide QRS</td>
<td>Wide QRS</td>
</tr>
<tr>
<td></td>
<td>HR 40-60 bpm</td>
<td>HR &lt;40 bpm</td>
</tr>
<tr>
<td>Incidence</td>
<td>12-20%</td>
<td>5%</td>
</tr>
<tr>
<td>Duration</td>
<td>Usually transient</td>
<td>Usually transient</td>
</tr>
<tr>
<td>Excess of inhospital mortality rate compared with no conduction disturbance</td>
<td>2-3 times</td>
<td>4 times</td>
</tr>
</tbody>
</table>

AV conduction disturbances in acute MI

<table>
<thead>
<tr>
<th>ECG findings</th>
<th>Location of the conduction disturbance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>AV node</td>
</tr>
<tr>
<td>PR interval</td>
<td>&gt;200ms</td>
</tr>
<tr>
<td>2nd degree AV block</td>
<td>Wenckebach</td>
</tr>
<tr>
<td></td>
<td>2:1 block</td>
</tr>
<tr>
<td>Conducted PR interval</td>
<td>&gt;200ms</td>
</tr>
<tr>
<td>PR increment</td>
<td>Big</td>
</tr>
<tr>
<td>HPS disturbances</td>
<td>Rare</td>
</tr>
<tr>
<td>Complete AV block</td>
<td>Progressively occurring</td>
</tr>
<tr>
<td></td>
<td>Infra nodal</td>
</tr>
<tr>
<td></td>
<td>Normal or &gt;200ms</td>
</tr>
<tr>
<td>2nd degree AV block</td>
<td>Mobitz II</td>
</tr>
<tr>
<td></td>
<td>2:1 block</td>
</tr>
<tr>
<td>Conducted PR interval</td>
<td>Normal or &gt;200ms</td>
</tr>
<tr>
<td>PR increment</td>
<td>Small</td>
</tr>
<tr>
<td>HPS disturbances</td>
<td>RBBB ± LAHB or LPHB</td>
</tr>
<tr>
<td>Complete AV block</td>
<td>Sudden onset</td>
</tr>
</tbody>
</table>

**Note:** LBBB in acute MI is **rare** and if it occurs it is due to a pause dependent phase 4 block in the conduction system and associated with a high rate of complete AV block.

Pacing after myocardial infarction

**Permanent pacing**
Conduction disturbances lasting >14 days related to myocardial infarction.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Class</th>
<th>Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Persistent AV block III</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>Persistent AV block II, Mobitz II associated with BBB</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>Transient AV block II, Mobitz II or AV block III associated with new onset BBB</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>Transient AV block II or III without BBB</td>
<td>III</td>
<td>B</td>
</tr>
<tr>
<td>LAHB newly developed or present on admission</td>
<td>III</td>
<td>B</td>
</tr>
<tr>
<td>Persistent AV block I</td>
<td>III</td>
<td>B</td>
</tr>
</tbody>
</table>

BBB denotes bundle branch block, LAHB left anterior hemiblock.

Dyspnea

81-year-old man with new onset dyspnea.

**Personal history**
Coronary artery disease, PCI RIVA 2001, RCX 2002, RCA 2009, LVEF 75%.
Arterial hypertension. Benign prostatic hyperplasia.

**Drugs before admission**
Aspirin 100 mg 1x/d, Losaratan 50 mg/d, Amlodipin 10 mg/d,
Pravastatin 40 mg/d, Alfuzosin 10 mg/d (α-blocker)

**Current admission**
Progressive dyspnea NYHA III since about 2 weeks.

**Clinical findings**
BP 160/90 mmHg, HR 64 bpm, irregular, RR 16. Bilateral pulmonary rales.
Q4: Why has this patient dyspnea?
Q4: Why has this patient dyspnea?

A: Supraventricular bigeminus, slow effective heart rate
B: 2\textsuperscript{nd} degree AV-block, type Wenckebach
C: 2\textsuperscript{nd} degree AV-block, type Mobitz
D: Complete AV-block
E: Atrial flutter with slow ventricular response
Dyspnea

**Diagnosis:** 2\(^{nd}\) degree AV-block with 3:2 conduction. aCL 640 ms, aHR 94 bpm (P waves indicated by arrows), vHR around 67 bpm. No significant PR prolongation (PR 200 ms). QRS 160 ms, complete RBBB and LAFB.

Cardiology, STZ, 2016.
2nd degree AV block

Mobitz type I, Wenckebach
Stable PP interval, progressive increase in the PR interval until a P wave fails to conduct. PR increment usually decreases with each beat → the RR intervals shorten. After the blocked P, the PQ interval returns to the initial value.

Mobitz type II, Mobitz
Stable PP interval, no measurable prolongation of the PQ interval before an abrupt conduction failure.

Laddergrams drawn by Woldemar Mobitz 1924.

## Localization of the AV block

<table>
<thead>
<tr>
<th>ECG findings</th>
<th>Localization of the block</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. QRS width</td>
<td>BBB pattern</td>
</tr>
<tr>
<td></td>
<td>Narrow QRS</td>
</tr>
<tr>
<td></td>
<td>AV node, His bundle</td>
</tr>
<tr>
<td>2. PQ interval of conducted P waves</td>
<td>PQ &gt;300ms</td>
</tr>
<tr>
<td></td>
<td>AV node</td>
</tr>
<tr>
<td></td>
<td>PQ ≤160ms</td>
</tr>
<tr>
<td></td>
<td>His bundle, His-Purkinje-system</td>
</tr>
<tr>
<td>3. Response to atropine or effort</td>
<td>Conduction↑</td>
</tr>
<tr>
<td></td>
<td>AV node</td>
</tr>
<tr>
<td></td>
<td>Conduction↓</td>
</tr>
<tr>
<td></td>
<td>His bundle, His-Purkinje-system</td>
</tr>
<tr>
<td>4. Carotid sinus massage</td>
<td>Conduction↓</td>
</tr>
<tr>
<td></td>
<td>AV node</td>
</tr>
<tr>
<td></td>
<td>Conduction↑</td>
</tr>
<tr>
<td></td>
<td>His bundle, His-Purkinje-system</td>
</tr>
<tr>
<td>5. Retrograde conduction</td>
<td>Present</td>
</tr>
<tr>
<td></td>
<td>His bundle, His-Purkinje-system</td>
</tr>
<tr>
<td></td>
<td>Absent</td>
</tr>
<tr>
<td></td>
<td>Not possible</td>
</tr>
</tbody>
</table>

BBB denotes bundle branch block.
Dyspnea

Therapy

**Chest X ray:** After the implantation of a dual chamber pacemaker.

Cardiology, STZ, 2016.
Recurrent syncope

59-year-old very sportive man with recurrent syncope

**Personal history**
No disease.

**Drugs before admission**
None.

**Current admission**
Daily episodes of dizziness and dyspnea and recurrent syncope after cycling.

**Clinical findings**
69 kg, 186 cm, BMI 19.9 kg/m2; BP 135/80 mmHg, HR 42 bpm. No signs of congestive heart failure.
Q5: What is the rhythm documented in this ECG?
Recurrent syncope

Q5: What is the rhythm documented in this ECG?

A: Sinus bradycardia
B: Sinus bradycardia and a faster atrial ectopic rhythm
C: Sinus arrest and junctional escape rhythm
D: Atrial fibrillation and complete AV-block
E: Atrial fibrillation with slow ventricular response and intermittent aberrant conduction
Recurrent syncope

ECG: Sinus arrest and slow wide escape rhythm (HR 40 bpm) with retrograde atrial activation (negative P waves in II, III, aVF). Diagnosis: Sick Sinus Syndrome and junctional escape rhythm.

Cardiology, STZ, 2011
ECG: Sinus arrest and slow wide escape rhythm (HR 40 bpm) with retrograde atrial activation (negative P waves in II, III, aVF). **Diagnosis:** Sick Sinus Syndrome and junctional escape rhythm.
Recurrent syncope

Echocardiography
No structural heart disease.

Physical stress test
Performance: 263W (163%), HR 50-150 bpm,
Maximal BP 192/88 mmHg.
Recurrent syncope

Q6: Why does the patient have pauses during the recovery phase?

Cardiology, STZ, 2011
Recurrent syncope

Q6: Why does the patient have pauses during the recovery phase?

A: Sinus arrhythmia
B: Sinus arrest followed by atrial ectopic beats
C: Sinus arrest followed by junctional ectopic beats
D: Atrial ectopies, not conducted to the ventricle
E: Sinus arrest and blocked atrial ectopies (B and D)
Recurrent syncope

ECG during recovery after a physical stress test: A: Intermittent sinus pauses up to 1720 ms followed by an atrial escape beat (green asterix). B: PAC (red asterixes) without conduction to the ventricle.

Cardiology, STZ, 2011
Chronotropic incompetence

Definitions
(1) Failure to reach a HR that is 70-85% of the age-predicted maximum at peak exercise
(2) Failure to achieve a HR of 100 bpm
(3) Maximal HR > 2 SD below that in a control population

Age predicted maximum heart rate

Old formula:
$HR_{\text{max}} = 220 - \text{age [yr]}$

New data (2001):
$HR_{\text{max}} = 206 - 0.88 \times \text{age [yr]}$

# Chronotropic incompetence

## Incidence: 5.6-58%

**Table 1**  Definition and incidence of chronotropic incompetence

<table>
<thead>
<tr>
<th>Authors (publishing year)</th>
<th>Definition of CI</th>
<th>Incidence of CI (%)</th>
<th>Sample size</th>
<th>Patient population</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bruce et al. (1980)²⁹</td>
<td>&lt;90% age predicted HR_{max}</td>
<td>5.6</td>
<td>2,365</td>
<td>Healthy men</td>
</tr>
<tr>
<td>Chin et al. (1979)¹⁶</td>
<td>&lt;mean−2SD</td>
<td>28.57</td>
<td>98</td>
<td>Suspected CAD</td>
</tr>
<tr>
<td>Corbelli et al. (1990)²</td>
<td>&lt;85% age predicted HR_{max}</td>
<td>47.37</td>
<td>19</td>
<td>AF</td>
</tr>
<tr>
<td>Crook et al. (1995)¹⁷</td>
<td>&lt;2SD of normal intercept</td>
<td>9</td>
<td>46</td>
<td>Paced for SSS</td>
</tr>
<tr>
<td>Ellestad et al. (1975)²⁸</td>
<td>&lt;95% C-I for normals</td>
<td>7.37</td>
<td>1,152</td>
<td>Suspected and confirmed CAD</td>
</tr>
<tr>
<td>Gwinn et al. (1992)²⁵</td>
<td>&lt;80% age predicted HR_{max}</td>
<td>58</td>
<td>38</td>
<td>27 AVB+11 SSS</td>
</tr>
<tr>
<td>Hammond et al. (1983)¹⁵</td>
<td>≤mean−1SE</td>
<td>13.59</td>
<td>103</td>
<td>CAD</td>
</tr>
<tr>
<td>Fei et al. (1994)²²</td>
<td>&lt;80% age predicted HR_{max}</td>
<td>24.39</td>
<td>41</td>
<td>DCM</td>
</tr>
<tr>
<td>McNeer et al. (1978)³⁰</td>
<td>&lt;120 beats/min</td>
<td>9.47</td>
<td>169</td>
<td>CAD</td>
</tr>
<tr>
<td>Wiens et al. (1984)²⁷</td>
<td>&lt;mean−2SD for normals</td>
<td>5.77</td>
<td>312</td>
<td>Suspected CAD or chest pain</td>
</tr>
</tbody>
</table>

**Abbreviations:** CI = chronotropic incompetence, C-I = confidence interval, HR\textsubscript{max} = maximal heart rate, SD = standard deviation, SE = standard error, CAD = coronary artery disease, AF = atrial fibrillation, DCM = dilated cardiomyopathy, AVB = atrioventricular block.

## Clinical pattern

A  Failure to achieve the HR\textsubscript{max}
B  Delay to achieve the HR\textsubscript{max}
C  Inadequate submaximal and recovering HR
D  Rate instability during exercise

Recurrent syncope

Therapy

**Chest X ray:** After the implantation of a dual chamber pacemaker.

Cardiology, STZ, 2011.
55-year-old patient with symptomatic paroxysmal atrial fibrillation

**Personal history**
Arterial hypertension

**Cardiac history**
2012  First episodes of symptomatic atrial fibrillation
  **Therapy:** Aspirin. No antiarrhythmic drugs.

3/13  Recurrent episodes of palpitations

**Work-up**
- **ECG:** Documentation of paroxysmal atrial fibrillation
- **TTE:** No structural heart disease, LVEF 56%, mild dilatation of the left atrium (PLAX 40mm, 35.2ml/m^2)
Diagnosis
1. Symptomatic paroxysmal atrial fibrillation
   First diagnosis 2012, EHRA II, CHA$_2$DS$_2$-VASc = 1
2. Arterial Hypertension

Therapy
• No antithrombotic therapy
• Flecainide

Follow-up, current admission
6/13 Recurrent episodes of palpitations associated with dizziness and lightheadedness.

ECG: next slide.
7d-Holter-ECG: next slides
ECG: SR, HR 56 bpm. PQ 160ms, QRS 80ms, QT 380ms.

Cardiology, STZ, 2013
ECG: Paroxysmal fast atrial fibrillation, HR 126 bpm.
Palpitations

Holter-ECG

ECG: Narrow QRS tachycardia, vHR 150 bpm.

ECG: Irregular narrow QRS tachycardia, vHR around 180-200 bpm. 3 consecutive beats with wide QRS complexes.

Cardiology, STZ, 2013
ECG: Narrow QRS tachycardia, vHR 270 bpm. Non-sustained wide QRS tachycardia (HR 270 bpm) for 28 beats.

ECG: Sustained wide QRS tachycardia (HR 270 bpm).
ECG: Transition from the wide QRS tachycardia to the narrow QRS tachycardia finally slowing down to a normal HR.

Q7: Why does this patient have palpitations with dizziness and lightheadedness?

A: Paroxysmal atrial fibrillation with fast ventricular response
B: Paroxysmal atrial fibrillation with fast aberrant conduction to the ventricle
C: Paroxysmal atrial fibrillation with recurrent non-/sustained ventricular tachycardias
D: Paroxysmal atrial flutter with 2:1 conduction to the ventricle
E: Paroxysmal atrial flutter with intermittent 1:1 conduction to the ventricle
Palpitations

ECG: Atrial flutter with variable conduction, up to 1:1 with wide QRS complexes (aberrant conduction).

Diagnosis
1. Paroxysmal atrial fibrillation, EHRA II, CHA$_2$DS$_2$-VASc = 1
   - Antiarrhythmic treatment with flecainide
2. Typical atrial flutter (IC flutter)
   - Intermittent 1:1 conduction and QRS widening

Why did this life-threatening arrhythmia occur?
Flecainide, Class IC

Drug
Flecainide / Tambocor®

Mechanism
(1) Use-dependent inhibition of the open and inactive Na-channel ($I_{Na}$)
   Slow dissociation from the Na-channel
   → Delayed depolarization
   → HR dependent slowing of the conduction velocity

(2) Weak inhibition of $I_{Kr}$ in Phase 1
   → Repolarization(↑) and QT↑

Flecainide – side effects

Non-cardiovascular side effects
Neurological side effects (e.g. paresthesia, paresis, blurred vision, dizziness)

Cardiovascular side effects
• Worsening heart failure

Proarrhythmic side effects
• Bradycardia
• Atrial and ventricular arrhythmias: 7-27%

• Conversion of atrial fibrillation to atrial flutter (IC flutter): 13% 
  54% with typical atrial flutter (based on surface ECG) 
  → Successful therapy by RF ablation of the cavotricuspid isthmus in 85%

• No effect on the conduction velocity in the AV node 
  → Combination with Class II or IV drugs recommended

Flecainide – side effects

Tachycardia induced widening of the QRS complex
Sinus tachycardia enhances the use-dependent Na-channel blockade → facilitating the occurrence of ventricular reentry.

6% of the population develop marked QRS widening at higher HR → routine exercise testing 1 week after initiation of flecainide therapy is recommended.

Irregular palpitations

71-year-old lady with paroxysmal irregular palpitations.

**Personal history**
Slightly elevated blood pressure, no medical treatment.

**Drugs before admission**
None.

**Current admission**
10/15  Recurrent episodes of paroxysmal, irregular palpitations lasting for a few minutes up to 2 hours.

**Clinical findings**
75 kg, 165 cm, BMI 27.5 kg/m2, BP 145/100 mmHg,  HR 136 bpm, irregular. RR 16. No signs of congestive heart failure.
Q8: Why does this patient have palpitations?
Q8: Why does this patient have palpitations?

A: Sinus tachycardia
B: AV nodal reentry tachycardia (AVNRT)
C: Orthodromic circus movement tachycardia (= AVRT)
D: Focal atrial tachycardia
E: Atrial fibrillation

Cardiology, STZ, 2016
Irregular palpitations

ECG: SR (P waves indicated by arrow heads), HR 100 bpm. Non-sustained focal atrial tachycardia (aCL 180-210 ms, P waves indicated by arrows) with irregular (~ 2:1) conduction to the ventricle (vHR around 170 bpm).

Cardiology, STZ, 2016
Irregular palpitations

**ECG:** SR (P waves indicated by arrow heads), HR 100 bpm. Non-sustained focal atrial tachycardia (aCL 180-210 ms, P waves indicated by arrows) with irregular conduction to the ventricle (vHR around 100-170 bpm). Aberrant conduction to the ventricle with RBBB morphology due to phase 3 block (Ashman phenomenon) (asterisk).
The length of R1R2 interval determines the duration of the effective refractory period (ERP) of the His-Purkinje system (HPS). The longer the RR interval, the longer the next ERP of the bundle branches (LBB < RBB). An early PAC may encounter a partially refractory HPS with an open LBB and a still blocked RBB.
Localization of the atrial focus

Algorithm to detect the localization of the atrial focus

V1  neg  pos/neg  neg/pos iso/pos  iso  pos

V2 – 4

pos  CT

neg  no  aVL

CT

neg in all inf. leads

yes  no  SMA

neg  pos  R. Septum Perinodal

neg in all inf. leads  sinus rhythm

pos  P wave

yes  no

TA  TA RAA

yes  no  LPV LAA

CS body

pos  CT RPV  RPV

Localization of the atrial focus

Algorithm to detect the localization of the atrial focus

Atrial tachycardia, localization of the focus

Anatomic distribution of focal atrial tachycardias.

Irregular palpitations

Holter-ECG

Diagnosis
1. Symptomatic paroxysmal atrial fibrillation
   - EHRA II, CHA$_2$DS$_2$-VASc 2
2. Arterial hypertension
76-year-old lady living in a nursing home with fast atrial fibrillation.

**Personal history**
Arterial hypertension.
Diabetes mellitus type 2.
Carotid and peripheral atherosclerosis with vascular bypass surgery.
Chronic kidney disease, Creatinine 315 umol/l, CrCl 13 ml/min

**Drugs before admission**
Rivaroxaban 20 mg/d, nebivolol 5 mg/d, amlodipin 20 mg/d, olmesartan 40 mg/d, torasemid 40 mg/d, statin, oral antidiabetic drugs, insulin.

**Current admission**
Admission because of low blood pressure (100 mmHg instead of 140 mmHg).
Q9: What rhythm is documented by this ECG?
Q9: What rhythm is documented by this ECG?

A: Sinus tachycardia
B: Focal atrial tachycardia
C: Atrial flutter with fast ventricular response
D: Atrial fibrillation with fast ventricular response
E: Atrial fibrillation with ventricular preexcitation
**ECG:** Atrial fibrillation, vHR 100-130 bpm. QRS 100 ms, QT 320 ms. ST segment depression and negative T waves in I-III, aVF, and V4-V6.

Cardiology, STZ, 2013.
Follow-up
Adjustment of rate control and heart failure therapy:
• Rivaroxaban 20 mg/d
• Ramipril 5 mg/d
• Torasemid 20 mg/d
• Bisoprolol 10 mg/d
• Diltiazem 180 mg/d
• Digoxin 0.25 mg/d (added after hospital discharge in the nursing home)

Readmission after 5 weeks
Admission because abdominal pain, nausea and vomiting.

Laboratory values
Hb 9.8 g/dl, Lc 8.2 G/l, Tc 193 G/l. CRP 56 mg/l. Na 133 mmol/l, K 3.99 mmol/l, Crea 233 umol/l.

Cardiology, STZ, 2013.
Q10: What rhythm is documented by this ECG?
Q10: What rhythm is documented by this ECG?

A: Sinus bradycardia
B: Sinus rhythm with 3rd degree AV block
C: Atrial flutter with slow ventricular response
D: Atrial fibrillation with slow ventricular response
E: Atrial fibrillation and 3rd degree AV block

Cardiology, STZ, 2013.
**ECG:** Atrial fibrillation, 3\textsuperscript{rd} degree AV block, and junctional escape rhythm at a vHR of 50 bpm. Ongoing atrial fibrillation waves are visible in lead V1.
Why did the complete AV block occur?
(1) Rate control with 3 drugs:
   Bisoprolol 10 mg/d, diltiazem 180 mg/d, and digoxin 0.25 mg/d
(2) Digoxin intoxication due to renal insufficiency
   Crea 233 umol/l, CrCl 18 ml/min
   Digoxin 7.8 nmol/l (no 1.0-2.6 nmol/l)

Therapy
Rhythm monitoring on the ICU.
Administration of digoxin antibodies.

Follow-up
Rate control: Bisoprolol 10 mg/d and diltiazem 180 mg/d. AF, vHR 70 bpm.
Change anticoagulation: Vitamin K antagonist, INR 2-3.
Digoxin toxicity

Actions of digoxin
• Inhibition of the Na\(^+\)/K\(^+\)-ATPase → intracellular [Na\(^+\)]↑ → intracellular [Ca\(^{2+}\)]↑ → Inotropy↑
• Increase in vagal tone → slowing of the vHR in AF

Mechanism of digitalis toxicity

Digoxin induced intracellular Ca\(^{2+}\) overload promotes the increased activity of the Na\(^+\)/Ca\(^{2+}\) exchanger in Phase 4 → delayed after depolarization (DAD).

Digoxin toxicity

**Actions of digoxin**
- Inhibition of the $\text{Na}^+/\text{K}^+$-ATPase $\rightarrow$ intracellular $[\text{Na}^+]$ $\uparrow$ $\rightarrow$ intracellular $[\text{Ca}^{2+}]$ $\uparrow$ $\rightarrow$ Inotropy $\uparrow$
- Increase in vagal tone $\rightarrow$ slowing of the vHR in AF

**Mechanism of digitalis toxicity**

Digoxin toxicity

Plasma levels
Digoxin \( \geq 1.7-2.0 \text{ nmol/l} \) (no correlation with toxicity)

Risk factors for clinical manifestation
- Hypokalemia, especially as a trigger for cardiac arrhythmias
- Hypomagnesemia
- Hypercalcemia
- Myocardial ischemia

Symptoms
**Gastrointestinal:** anorexia, nausea, vomiting, and abdominal pain
**Neurological:** Changes in mental status as lethargy, fatigue, delirium, confusion, disorientation, and weakness.
**Visual changes:** chromatopsia, diplopia, photophobia, decreased visual acuity, photopsia, scotomas, or blindness

## Digoxin toxicity: cardiac arrhythmias

<table>
<thead>
<tr>
<th>Level</th>
<th>Bradyarrhythmias</th>
<th>Tachyarrhythmias</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinus node</td>
<td>• Sinus bradycardia&lt;br&gt;• SA block</td>
<td>• Sinus tachycardia</td>
</tr>
<tr>
<td>Atrium</td>
<td></td>
<td>• Ectopic atrial tachycardia (aHR usually &lt; 250 bpm) with (2:1) AV block</td>
</tr>
<tr>
<td>AV node</td>
<td>• PR prolongation &gt; 200 ms&lt;br&gt;• 2\textsuperscript{nd} degree AV block, type Wenckebach&lt;br&gt;• 3\textsuperscript{rd} degree AV block</td>
<td>• Junctional rhythm with narrow QRS at various HR (HR &lt; 40-60-120 bpm)</td>
</tr>
<tr>
<td>Ventricle</td>
<td></td>
<td>• PVC, bigeminus&lt;br&gt;• Ventricular tachycardias&lt;br&gt;• Bidirectional ventricular tachycardia&lt;br&gt;• Ventricular fibrillation</td>
</tr>
</tbody>
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