Cardiac rhythm disturbances
Hypertensive emergencies
Syncope-Sudden cardiac death
Metabolic disorders

Dr. Szabó Zoltán
The aim of arrhythmia management

- To resolve symptoms
- To decrease the risk of SCD
- To prevent hemodynamic unstability
- To avoid consequences (e.g. stroke prevention)
Questions to answer

- Does the arrhythmia cause any symptom?
- Does it cause an increased risk of SCD?
- Which type of arrhythmia is present?
- Is cardioversion indicated?
- Is admission to hospital indicated?
- Is cardiologist needed to solve the problem?
- Specific management (e.g. anticoagulation?)
Anatomical background

- CAD
- Cardiomyopathies - dilated, hypertrophic
- ARVD
- Valvulopathy
- Congenital
- Primary electrophysiol.
- Neurohormonal
- Myocarditis/pericarditis
- Neoplastic
- Toxic

Transitoric factors

- Neuroendocrine
- Drugs
- Electrolyte
- pH, pO2
- Hemodynamic
- Wall stress
- Sleep
- Alcohol

Arrhythmia mechanism

- Reentry
- Automacy
- Triggered activity
- Block
- Uncoupling

Zipes and Wellens Circ 1998; 98:2334
I. Non invasive
1. Family history
2. complaints
3. Physical examination
4. ECG
5. Echocardiography
6. Laboratory examinations
7. Drug tests
8. Stress test
9. Holter-ECG
10. Transoesophageal stimulation
11. Autonomic nerve effects
12. Evaluation of arrhythmia substrate
Occurrence of arrhythmias

- AFib: 34%
- Non identified: 18%
- PSVT: 6%
- VPB: 6%
- A flutter: 4%
- SSS: 9%
- Pulse conduction disorder: 8%
- SCD: 3%
- VT: 10%
- VF: 2%

AF-mortality

Framingham

Age 55-74 years

Age 75-94 years

Percent of Subjects Dead in Follow-up

Classification of atrial fibrillation

First diagnosed episode of atrial fibrillation

- Paroxysmal (usually ≤48 h)
- Persistent (>7 days or requires CV)
- Long-standing Persistent (>1 year)
- Permanent (accepted)
Electrophysiologic background

Remodelling – multicircuit reentry

AF-consequences

Decreased atrial function → atrial thrombi → Embol, Stroke

Atrial pressure ↑ → Congestion → Dyspnea

Stroke volume ↓ → Cerebral perfusion → Vertigo

Irregular rapid ventricular response → O2-need ↑ → Angina pectoris

Coronary perfusion → Palpitation
AF-management

Medical
- Underlying disease
- Antiarrhythmic drugs
- Anticoagulant therapy

Non medical
- RFCA
- Pacemaker
- DC cardioversion

Hybrid treatment
Tachycardiomyopathy and atrial volume

Cardioversion not indicated

- Permanent AF (>2 years)
- Dilated LA
- SSS
- Evere mitral valvulopathy
- Recurrent AF, if the patient is older than 75 years
DC cardioversion indicated

- Unstable hemodynamic status
- Syncope
- Unstable angina pectoris syndrome
- Within 48 hours
Medical cardioversion

Pro:

– easy
– Less relapse

Con:

– Proarrhythmia
– <48 óra: 35-90% success
– >1 week: 20-65% success

ACC/AHA/ESC guidelines for the management of patients with atrial fibrillation. Europace 2006, 8, 651-745.
## Medical treatment

<table>
<thead>
<tr>
<th>First choice</th>
<th>No structural heart disease</th>
<th>Hypertension</th>
<th>CAD</th>
<th>Heart failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Propafenone</td>
<td>IC</td>
<td>IC</td>
<td>III</td>
<td>III</td>
</tr>
<tr>
<td>Flecainide</td>
<td>IC</td>
<td>Propafenone</td>
<td>Dofetilide</td>
<td>Dofetilide</td>
</tr>
<tr>
<td>IC</td>
<td>IC</td>
<td>Flecainide</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Second choice</th>
<th>No structural heart disease</th>
<th>Hypertension</th>
<th>CAD</th>
<th>Heart failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dofetilide</td>
<td>III</td>
<td>III</td>
<td>III</td>
<td>III</td>
</tr>
<tr>
<td>Ibutilidee</td>
<td></td>
<td>Dofetilide</td>
<td>Amiodarone</td>
<td>Amiodarone</td>
</tr>
<tr>
<td>III</td>
<td></td>
<td>Ibutilide</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| IA            | IA                          | IA           | IA  | IA           |
| Procaainamid  | IA                          | Procaainamid| Amiodarone | Amiodarone |
| Kinidin       |                             | Procaainamid|     |             |
| IA            | IA                          | IA           |     |             |
| Procaainamid  |                             | Procaainamid|     |             |
| III           |                             | Amiodarone   |     |             |
| Amiodarone    |                             | Amiodarone   |     |             |

Alkalmazott gyógyszerek: Vaughan-Williams szerint, Kardiológiai Szakmai Kollégium 2011. évi ajánlása
WPW and AF
Anticoagulant treatment

Intermittent, persistent and permanent AF

INR 2-3 között

Prosthetic valve: INR 2.5-3.5 (I/A)

stroke: INR 3.0-3.5

Lone AG, <60 years, Aspirin

Kardiológiai Szakmai Kollégium 2011. évi ajánlása
DC cardioversion-anticoagulants

- **AF>48 hrs**, 3 weeks before, 4 weeks after (I/C)
- **More than 4 weeks**: recurrent AF
- **AF<48 hrs** heparin, LMWH before (II/C)
- **Increased cardiovascular risk** (e.g. mitral valvulopathy)-TOE
Protocol-risk assessment

1. AF
2. 12 lead ECG
3. History
   - EHRA score
   - Concomitant diseases
4. Anticoagulant th?
5. Thromboembolic risk
   - OAC
   - PAI
   - None
6. Rhythm or rate control
    - AF type
    - Symptoms
8. Management of underlying disease
   - Upstream therapy
   - RAAS inhibitor, statin
9. Medical treatment
   - Ablation

Atrial flutter

- 240-440/min regular atrial movement
- atrial macroreentry
- **Type I**: II, III, aVF: negative F waves (right atrial)
- **Type II**: II, III, aVF: poz F waves (left atrial)

**Causes**
- Heart surgery
- Obstructive pulmonary diseases
- Valvulopathies
- Hyperthyreodism
Diagnosis

**Drugs**
I/A, I/C, III (Ibutilid!)

**DC shock**
Overdrive stimulation

**RFCA**

![Image of carotid sinus and related structures]

![Carotid message graph]
Atrial premature beats

- Asymptomatic - no treatment needed
- Symptomatic
  - Underlying disease
  - BB, I/A, I/C
Non Complex Ventricular Premature Beats

Treat the underlying disease

No structural heart disease

Asymptomatic: No treatment

Symptomatic: BB, propafenone

Structural heart disease

Asymptomatic: BB

Symptomatic: BB, sotalol, amiodarone
Complex VPBs and/or monomorphic non sustained VT
Treat the underlying disease

No structural heart disease

Structural heart disease

Asymptomatic

Symptomatic

No treatment

BB

Asymptomatic

Symptomatic

BB

BB, sotalol, amiodarone, RF ablation, ICD
AVNRT

- Vagus maneuvers
- Adenosin
  NDCCB
  BB

- DC cardioversion
- Ablation
SCD- Epidemiology

- Incidence: 1-2/1000
- Risk factors
  - Diabetes mellitus
  - Obesity (BMI!)
  - Dyslipidemia
  - Age (45-75 yrs)
  - Gender (male 3,8 X)
  - Left ventricular dysfunction (combined with CAD)
VT VF caused by:
- Idiopathic CM
- Hypertrophic CM
- Long QT syndrome
- ARVD
- WPW syndrome

Arrhythmias resulting in SCD:

- VT 62%
- Primary VF 8%
- Torsades de Pointes polymorphic VT 13%
- Bradycardia 17%

Stable VT treatment

**Monomorphic VT**

- **Norm systolic function**
  - Procainamide
  - Sotalol
  - Amiodarone
  - Lidocain

- **Decreased systolic function**
  - Amiodarone
  - Lidocain

**Polimorphic VT**

- **Norm. QT**
  - Treatment of ischemia
  - Electrolyte
  - BB
  - Amiodarone
  - Procainamide

- **Long QT**
  - Electrolyte
  - Magnesium
  - Overdrive pacing
  - Isoproterenol
  - Lidocain
  - Sotalol
**Ventricular fibrillation** There is a complete absence of properly formed QRS complexes and no obvious P waves. A recent onset (e.g., within minutes) of the arrhythmia is suggested by the coarse morphology of the fibrillatory waves.
Success of defibrillation

The success is reduced by 7-10% in every minute


* Non-linear
Hypertensive emergencies
Hypertensive emergency

A critical increase in blood pressure which may result in an abnormal arteriolar autoregulation that can lead to organ failure and death.
Types of hypertensive emergencies

„URGENCY”: a clinical precrisis which may worsen to a definitive organ failure

„EMERGENCY”: crisis, life threatening condition
Mechanism of hypertensive crisis

Hypertension
REMODELING
Abnormal autoregulation
Humoral factors

Increasing blood pressure

Further vasoconstriction

Tissue hypoperfusion

Hypertensive crisis
The severity of hypertensive emergency depends on the followings:

1. The severity of the actual hypertension
2. The increase of blood pressure in time
3. The condition of the vasculature
The clinical forms of hypertensive emergencies

Crisis

**Acute hypertensive encephalopathy**

**Hypertension and stroke**
- Sudden left ventricular failure
- Unstable angina pectoris, myocardial infarction
- Acute aortic dissection
- Acute renal failure
- Eclampsy
- Phaeochromocytoma crisis
- Retinal bleeding, exudate, papillary-oedema

Precrisis

- Non complicated malignant hypertension
- Perioperative hypertension
- Clonidin-withdrawal syndrome
Diagnosis of hypertensive emergencies

- Has the patient been suffering from hypertension before?
- Treatment?
- How rapid is the elevation of blood pressure?

- Repeated blood pressure measurements !!!
HYPERTENSIVE ENCEPHALOPATHY
PATHOMECHANISM

• Intracerebral vasoconstriction
• Arteriolar dysregulation-dilation
• Oedema formation
Treatment

- The patient should be admitted to a hospital
- Emergency - parenteral medication
- Urgency – oral medication
- **To reduce blood pressure by 25% or less in the first two hours**
- **To reduce blood pressure by 25% in the next 24 hours**
- **Do not decrease blood pressure immediately (cerebral oedema)**
- In the case of blood pressure above 180/120 Hgmm repeated measurements are advised in every 15 minutes.
Therapy-emergency

- uradipil iv.
- labetalol, verapamil, nicardipine
- Special circumstances
  - subarachnoidal haemorrhage: nimodipin
  - unstable angina: nitroglycerin
  - CABG: isradipin
  - pheochromocytoma: fentolamin
  - eclampsy: MgSO₄, isradipin
Treatment - urgency

- Oral medication
- Nifedipin
- Captopril – first choice
- urapidil or verapamil iv.

The use of oral nifedipin is strongly discouraged.
Management of hypertension in acute stroke
Rapid decrease in blood pressure.

ISCHEMIC PENUMBRA

Survival or death?

Blood pressure decrease in the case of stroke may worsen the clinical outcome.
Management of hypertension in acute stroke
(European Stroke Initiative, 2003)

- Only blood pressure above 220/110-120 mmHg should be treated in the first 24 hours
- Target level: 180/100-105 mmHg
- Between 180-220/105-120 mmHg: no treatment
- Exclusion criteria: AMI, HF, ARF, aortic dissection, thrombolysis

- urapidil 10-50 mg (i.v.)
- captopril 6-12,5-25 mg (p.o.)
Secondary hypertension

- The pathomechanism can be determined
- 10 %

- **Renal origin** (the most frequent)
  - Renovascular
  - Renoparenchymal
- **Coarctation of the aorta and aortitis**
- **Endocrine origin**
  - Hyperaldosteronism
  - Phaeochromocytoma
  - Cushing syndrome
  - Thyroid dysfunction
  - Primary hyperparathyreoidism
- **Neurological diseases**
  - Sleep apnea syndrome
- **Drugs**
Renovascularis hypertension

- 75% stenosis
- < 1%
- Atherosclerotic stenosis
  - >50 yrs, diabetes mellitus, smoking
- Fibromuscular dysplasia – young women, non inflammatory, non atherosclerotic
Renal artery Doppler

- anatomic/functional evaluation of the renal arteries
- Systolic and diastolic blood flow
CT-angiography

- Sensitivity: 98 %
- Specificity: 94 %
MR-angiography

- **Proximal stenosis**
  - sensitivity 100 %
  - specificity 70 %
Isotopic renography

- Captopril sensitizing
  - Lengthened isotope uptake
  - Decreased secretion (>10 mins)
Angiography

Right Renal Artery Stenosis

Aorta
Treatment

- ACE I (!):
  - Renal function
- CCB
- BB
- Diuretics
- Alpha receptor blockers
- Direct vasodilators
Follow up

- After angioplasty
- 6 months
- 12 months
- Yearly
- Worsening status—immediately
Pheochromocytoma

- <0.2%
- Most common between the age of 40-50
- Epinephrine, norepinephrine, dopamine (rare)
- Other: IL-6, EPO, VIP, ACTH, calcitonine, substance-P, adrenomedulline, endotheline
- 90% intraadrenal, 10% extradrenal (paraganglioma)
- MEN 2A, 2B, neurofibromatosis, von Hippel Lindau
- 10% malignant
Symptoms

- Paroxysmal hypertension
- Headache
- Arrhythmias (AF, VT, VF)
- Sweating
- Fatigue
- Tremor
- Panic
- 50% fixed hypertension
- Angina pectoris syndrome
- Acute heart failure
- Pale
- Nausea
- Blurred vision
- Rarely hypotension
- Polycythaemia (EPO)
- No symptoms - incidentaloma
Laboratory tests

– Urine collection (24 hrs)
  • Epinephrine, norepinephrine, dopamine
  • Metanefrines
  • Together: Sensitivity 98 %, specificity 98 %

– Serum catecholamines – low clinical relevance

– Chromogranin A
  • Produced by neuroendocrine cells
  • Not specific
Chromogranin A

- Hyperparathyreoidism
- Hyperthyreoidism
- Chr. hepatitis, cirrhosis
- IBD
- Irritable bowelsy.
- Heart failure
- PPI / H2 blocker
- RA
- Chr. bronchitis
- Chr. Renal failure
- Acute coronary syndrome
- Carcinoid tumor
- Thyroid medullar carcinoma
- Pituiter tumors
- Colon carcinoma
- Pancreas adenocarcinoma
- Colon carcinoma
- Pulmonary carcinoma
- Ovarial carcinoma
- Prostate carcinoma
Diagnostics

- CT
- MR
- I131 MIBG
- FDG-PET
- In-111 octreoscan
Sleep apnea syndrome

- Central
- Obstructive
- Mixed type

- smoking
- COPD,
- obesity,
- snoring
- hypertension
- arrhythmia

- Treatment
  - Weight loss
  - Stop smoking
  - Surgery
  - PEEP respiration
Metabolic emergencies
Hypoglycemia
Hyperglycemia
Hypoglycemia

- 3,0 mmol/l or under
- Incidence
  - Patient on insulin 10%/year
  - Intensified insulin treatment: 25%/year
  - sulfanyllurea 6-10%/month

- Mortality: 4% in type I diabetes
Symptoms

1. **Adrenergic neurogenic symptoms**: palpitation, tremor, (epinephrin, norepinephrin), sweating, hunger, nausea (Ach).

2. **Neuroglycopenic symptoms**: headache, sleepiness, fatigue, blurred vision, vertigo, confusion.

3. **Coma**: the skin is pale and wet, RR norm or ↑, Babinski-poz, seizures.
Causes of hypoglycemia

Diet
- Problem with insulin administration

Weather (warm)
- Problem with the time of eating
Causes of hypoglycemia

- Increased physical activity
- Alcohol
- Decreased insulin secretion (renal disease)
Treatment of hypoglycemia

- Glucose per os or iv
  - if the underlying use is not recognized hospitalization advised
- Long term consequence
  - Altered mental status
### Incidence of diabetes ketoacidosis

#### 4.6-8 episodes/ 1000 patients

<table>
<thead>
<tr>
<th></th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Till 1922</td>
<td>100%</td>
</tr>
<tr>
<td>1932</td>
<td>30%</td>
</tr>
<tr>
<td>Present</td>
<td>2-10%</td>
</tr>
<tr>
<td>&gt;65 years</td>
<td>20%</td>
</tr>
<tr>
<td>Youngsters</td>
<td>2-4%</td>
</tr>
</tbody>
</table>

Sagarin M., McAfee A.: Hyperosmolar hyperglycemic nonketotic coma
## Underlying diseases

<table>
<thead>
<tr>
<th>Infection</th>
<th>Renal, GI</th>
<th>Drug induced</th>
<th>Endocrine</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pneumonia</td>
<td>Ileus, Renal failure</td>
<td>Diuretics BB</td>
<td>Thyreotoxicosis</td>
<td>AMI</td>
</tr>
<tr>
<td>Uro-infection</td>
<td>Uro-infection</td>
<td>Cimetidin CCB</td>
<td>Cushing-syndrome Acromegaly Diabetes</td>
<td>Hypothermia</td>
</tr>
<tr>
<td>Sepsis</td>
<td>Sepsis</td>
<td>Phenytoin Steroid</td>
<td></td>
<td>Burn</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Parenteral feeding</td>
<td></td>
<td>Surgery</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Hyperglycaemia

extracellular space hyperosmolarity

intracellular space

Volume loss

PO$_3$, K

ketoacidosis

Electrolyte imbalance

Na, K, Ca, Mg loss

References:
Hyperglycemia

- Dry skin
- Blood pressure decreased
- Compressible pulse
- Kussmaul-breathing
Symptoms

- nausea
- polyuria
- vomitus
- abdominal pain
- lethargy
- somnolence
- Kussmaul breathing
- symptoms of hypovolemia
- RR ↓
- Pulse is frequent
- Dry skin
- Leucocytosis – hemoconcentration
- acetonuria

Abraham M. R.: Diabetes Care: 22, 1380-1381; (1999)
Treatment

- Iv volume
- Insulin
- Electrolyte
- PH
Indication for bicarbonate

- pH < 6.9
- Severe hyperkalemia
- Shock

Volume therapy in ketoacidosis

RR, Hemodynamic status

Hypovolemic shock
Shock index > 1

Hypotension

Serum Na ↑

0,9% NaCl 1 l/h, or colloid

Cardiogenic shock: pulmonary oedema

Serum Na ↓

0,45% NaCl 4-14 ml/tskg/h

Hemodynamic monitoring

0,9% NaCl 4-14 ml/tskg/h

Insulin therapy in ketoacidosis

- Insulin treatment
  - Rapidly acting insulin (Actrapid, Humulin R)
    - 0.15 E/kg iv.
  - 0.1 E/kg/h perfusor (Actrapid, Humulin R) infúzió iv.
  - If blood glucose not decreased by 3-4.2 mmol/l/hour
  - If blood glucose decreased by 3-4.2 mmol/l/hour
  - Duplicate the dose until glucose reacheis 15 mmol/l
  - Continue infusion until glucose reaches 15 mmol/l
  - 5%-os dextrose, 0.45% NaCl 150-250 ml/h, insulin 0.05-0.1 E/kg/h

Correction of acidosis

Acidosis

pH: < 6.9

pH: 6.9 - 7.0

100 mmol Na HCO₃ in 400 ml water, 200 ml/h

pH: > 7.0

100 mmol Na HCO₃ in 400 ml water, 200 ml/h

No bicarbonate indicated

Potassium and ketoacidosis

- **SeK:< 3.3 mmol/l**
  - Wait with insulin 40 mmol/l/h K+ iv.

- **SeK: 3.3-5.5 mmol/l**
  - 20-30 mmol/l K+ until se K reaches 4-5 mmol/l

- **SeK: >5.5 mmol/l**
  - No potassium administration needed

Thank you for your attention!