DISTURBANCES OF CARDIAC RATE & RHYTHM

Clinical significance

Consequences
- Lethal (sudden cardiac death)
- Symptomatic (syncope, near syncope, dizziness, palpitations, Asymptomatic.

Danger
- Reduction of cardiac output.
- Tendency to deteriorate into more serious arrhythmias.

Mechanisms of arrhythmias
- Disorders of impulse formation or automaticity
- Abnormalities of impulse conduction
- Reentry
- Triggered activity

Techniques for evaluating rhythm disturbances
- ECG monitoring, (24 h Holter)
- Electrophysiologic testing
- Autonomic testing
  - Table tilting: investigation of vasovagal syncope
  - Carotid sinus massage: should not be done in patients with carotid bruits or cerebrovascular disease!

THE CLASSIFICATION OF ARRHYTHMIAS

Supraventricular arrhythmias
1. Sinus arrhythmia, bradycardia, tachycardia
2. Atrial premature beats (atrial extrasystoles)
3. Paroxysmal supraventricular tachycardia (PSVT, Atrial or junctional tachycardia)
4. Atrial fibrillation, atrial flutter
5. Multifocal (chaotic) atrial tachycardia
6. AV junctional tachycardia
7. Supraventricular tachycardias due to accessory AV pathways (Preexcitation syndromes)

Ventricular arrhythmias
1. Ventricular premature beats (Ventricular extrasystoles, VES-s)
2. Ventricular tachycardia (VT)
3. Ventricular fibrillation (VF)
4. Accelerated idioventricular rhythm
5. Long QT syndrome
SUPRAVENTRICULAR ARRHYTHMIAS

Sinus arrhythmia, bradycardia & tachycardia

Sinus arrhythmia
- Caused by change in vagal tone. No clinical significance.

Sinus bradycardia
HR < 50/min, Causes: increased vagal influence (incl. hypertension, increased intracranial pressure), sinus node disease, hypothyroidism, uremia, biliary peritonitis, drugs: ß-blockers, digitalis.

Sinus tachycardia
HR > 100/min. Causes: fever, exercise, emotion, pain, anemia, heart failure, shock, thyrotoxicosis, drug effect (e.g. sympathicomimetics, atropine), pulmonary embolism, etc. Onset and termination are gradual. Rate infrequently exceeds 160/min but may reach 180/min in young persons.

Atrial premature beats (atrial extrasystoles)
- The contour of the P wave usually differs from the normal. Ventricular systole occurs prematurely, the compensatory pause is only slightly longer than the normal RR period.
- Early atrial ES can be conducted aberrantly or not conducted.
- Speeding up the heart rate usually abolishes atrial premature beats.
- Occur frequently in normal hearts and are never a sufficient basis for diagnosis of heart disease.

Paroxysmal supraventricular tachycardia (PSVT, atrial and junctional tachycardia, PAT)
- The most common paroxysmal tachycardia. Occurs most often in young patients with normal hearts.
- Stress, emotion, alcohol ingestion or smoking can precipitate attacks.
- HR: 160-220(140-240)/min, perfectly regular. P wave usually differs from sinus beats. PAT with AV block (1:2, 1:4) may result from digitalis toxicity. PSVT is started and terminated by a fortuitously timed SVES. Most attacks break spontaneously, but PSVT can seriously deteriorate heart failure, or coronary heart disease.

Mechanisms:
- Reentry (80%) perfectly regular rhythm in the higher frequency range.
- Ttrigger (20%): slightly irregular on ECG. HR is in lower range.

Treatment of PSVT
Treatment of the acute attack:

Vagal stimulation: Valsalva maneuver, coughing, carotid sinus pressure (cautiously, see above!). Pressure should not be exerted on both carotids at the same time. Eyeball pressure should be avoided because of retinal detachment.

Drug therapy*: Verapamil, adenosine, esmolol are the best. Edrophonium (Tensilon) 5-10 mg IV, metaraminol, phenylephrin, digoxin. These drugs may be contraindicated in WPW syndrome!

Cardioversion: DC shock 50-100 J. Not to be used when digitalis toxicity is present!

Prevention of attacks:
Drugs: digoxin, verapamil, ß-blockers, chinidine+digoxin, procainamide, dysopyramide, propafenone, amiodarone.
AV node modification and His bundle ablation.

Antitachycardia pacemaker.

*In each disease, drugs are listed in order of choice.

**Atrial fibrillation**
- The most common chronic arrhythmia.
- Occurs in rheumatic heart disease, dilatative cardiomyopathy, ASD, hypertension, mitral valve prolapse, hypertrophic cardiomyopathy, thyrotoxicosis, and without cardiac disease. Pericarditis, trauma, surgery, excessive alcohol intake may cause attacks. Often occurs paroxysmally before becoming an established rhythm. Atrial rate: 400-600/min, ventricular rate: 80-180/min (absolute arrhythmia: rapid, irregular ventricular rate). Pulse deficit may occur.
- Major morbidity: precipitation of cardiac failure or ischemia, and arterial embolization from the LA: cerebral, limb, renal, mesenterial arteries). Therefore anticoagulant therapy is required.
- "Lone" atrial fibrillation: near-normal HR, no underlying cardiac disease, there is a small risk for embolization under 60, so no anticoagulation therapy is needed.

**ECG criteria**
- No P wave in any leads, not even in V1 or V2.
- Ventricular rate is variable (low, normal, high)

**Treatment of atrial fibrillation**

**Acute**
- Ventricular rate control Can be urgent when ischemia, hypotension, or heart failure is present. For outpatients: digoxin, verapamil, diltiazem, esmolol, digoxin. These drugs are contraindicated in WPW syndrome.
- Cardioversion. Immediate DC shock 100 J, followed by amiodarone, sotalol, propafenone, chinidine.pharmacologic: quinidine, propafenone, sotalol, amiodarone.

**Chronic**
- Digoxin, alone, or in combination with verapamil or a β-blocker.
- Amiodarone, sotalol.

**Atrial flutter**
- Occurs in COPD, rheumatic or coronary heart disease, congestive heart failure, or ASD.
- Ectopic impulse (f wave) formation occurs at atrial rates of 250-380/min, with a transmission rate (block) of 2:1, 3:1, or 4:1, resulting in ventricular rates of 150, 100, or 75/min, respectively. Standing or exercise can decrease the block rate from 4:1 to 2:1, HR: 75 to 150/min. The risk of embolization is lower than in atrial fibrillation.

**Therapy**
- DC shock (often < 50 J)
- Class Ia or lc agents should be avoided unless given with delayers of the AV conduction (digoxin, verapamil, or β-blocker)!
- digoxin+chinidine

**Prevention**
as with atrial fibrillation.
**Multifocal (chaotic) atrial tachycardia**
- Varying (at least 3 different) P wave morphology, with markedly irregular PP intervals.
- Ventricular rate: 100-140/min, no block occurs.
- Cause: chronic respiratory failure, COPD.
- Therapy: treatment of the underlying lung disease; verapamil.

**Supraventricular tachycardias due to accessory AV pathways (preexcitation syndromes)**

**Pathophysiology & clinical findings**
- Accessory pathways between the atria and the ventricles avoid the conduction delay in the AV node, and predispose to reentry tachycardias. Accessory fibers, which occur in 0.1-0.3% of the population, may be:
  1. totally or in part in the AV node (Mahaim fibers) → Long-Ganong-Levine (LGL) syndrome: short PR, normal QRS morphology.
  2. more commonly, a direct connection between the atria and the ventricles through the Palavino-Kent fibers: Wolf-Parkinson-White (WPW) syndrome.

**The Wolf-Parkinson-White (WPW) syndrome**
- Short PR and early δ wave at the onset of the wide, slurred QRS complex owing to the early ventricular depolarization of the region adjacent to the pathway.
- A→V conduction through AV node → narrow QRS
- A→V conduction through bypass tract → wide QRS. This direction results in very fast rates.
- Digoxin, verapamil, and β-blockers may decrease accessory pathway refractoriness and increase ventricular response rate and should be avoided in atrial fibrillation with accessory pathways!

**Treatment of excitation syndromes**

**Pharmacologic therapy**
- narrow QRS tachycardias: adenosine.
- wide QRS tachycardias: class Ia, newer class Ic and class III antiarythmics.

**Electric cardioversion**

Long-term therapy combination of Ia of Ic agents (bypass refractoriness is increased), provided atrial fibrillation with short PR cycle lengths is not present. In resistant cases: propafenone, amiodarone.

**Electrophysiologic evaluation & specialized treatment**
- Ablation with radiofrequency catheters.
- Surgical ablation.
- Antiarrhythmia pacemakers.

**VENTRICULAR ARHYTHMIAS**

**Ventricular premature beats (ventricular extrasystoles, VES-s)**
- Are more common than SVES-s.
- Wide QRS with morphology differing from normal beats. Are usually not preceded by a P wave, although retrograde VA conduction may occur. There is a fully compensatory pause. Bigeminy or trigeminy may be found.
Exercise generally abolishes VES-s in normal hearts. The patient may or may not sense the irregular beat, usually as a skipped beat.

Further assessment can be done by Holter monitoring and exercise ECG. VES-s have a questionable significance in the absence of heart disease.

VES-s induced by a low level of exercise may have a worse prognosis than those which occur spontaneously. Sudden death occurs more frequently (presumably as a result of VF) when VES-s occur in the presence of organic heart disease.

**Therapy**

- If no associated cardiac disease is present and if the ectopic beats are asymptomatic, no specific therapy is indicated (Electrolyte disturbances, hyperthyroidism and occult heart disease should be excluded).
- When mitral prolapse, hypertrophic cardiomyopathy, LV hypertrophy, or coronary disease, long QT interval is present, β-blocking agents can be given. Class Ia and Ib agents are effective but are also arrhythmogenic.

**Ventricular tachycardia (VT)**

**Definition**

3 or more consecutive ventricular premature beats. The usual HR is 160-240/min, moderately regular but less so than in PAT. Carotid sinus pressure has no effect.

**Mechanism**

Mostly reentry

- non-sustained VT: lasting less than 30 s
- sustained VT: lasting longer than 30 s, respectively. VT is a frequent complication of acute myocardial infarction, COCM, but may occur in hypertrophic cardiomyopathy, mitral valve prolapse, myocarditis, etc.
- "Torsade de pointes VT": varying QRS morphology. This VT is caused by drugs that prolong the QT interval (e.g. class I, Ic, III drugs, quinidine) and has a poor prognosis. Intravenous β-blockers may be effective, so are temporary ventricular or atrial pacing).

**Treatment of ventricular tachycardia**

**In acute VT**

- DC shock (100-200 J)
- lidocaine IV (also prophylactically)
- procainamide (100 mg IV every 5 min up to 750-1000 mg, followed by infusion at 20-80 µg/kg/min)
- β-blockade, phenytoin, bretylium, amiodarone
- ventricular overdrive pacing
- Diphedane given IV is good in VT induced by digitalis intoxication.

Sustained VT requires therapy regardless of symptoms

- Medical: amiodarone
- Electrical: automatic implantable cardiac defibrillator (AICD)
- Surgical resection

**Ventricular fibrillation (VF)**

The most serious arrhythmia leading to death without acute and effective treatment
Symptoms
Clinical death

Therapy
DC shock (300-400J). If unresponsive: bretylium and repeated DC while cardiopulmonary resuscitation is administered.

Prophylaxis of recurrent VF
- amiodarone, β-blockers
- automatic implantable defibrillator
- ablation of foci.

Long QT syndrome
- Idiopathic (congenital) long QT syndrome is associated with deafness, ventricular arrhythmias and sudden death. QT is between 0.5-0.7s.
- Therapy: β-blockers, phenytoin, blockade of the ganglion stellatum. Class Ia, Ic, and III drugs are contraindicated.
- Acquired long QT interval: is due to antiarrhythmic agents, antidepressant drugs, electrolyte abnormalities, myocardial ischemia, significant bradycardia. These may result in VT ("torsade de pointes")

Antiarrhythmic drugs
Class I: block sodium channels. Subclasses are divided by their effect on Purkinje fiber action potential.
- Ia: slows the rate of rise of the action potential (Vmax) and prolongs its duration, slow conduction and increase refactoriness. Quinidine, procainamide, disopyramide, moricizine
- Ib: shortens action potential duration. DOES NOT affect conduction or refactoriness. Lidocaine, tocainide, mexiletine, phenytoin
- Ic: prolongs Vmax and slow repolarization, thus slowing conduction and prolonging refactoriness, but more so than class Ia drugs. Flecainide, encainide, propafenone

Class II: β-blockers, decrease automaticity, prolong AV conduction and refactoriness. Esmolol, propranolol, acebutolol

Class III: block potassium channels, prolong repolarization, widen QRS and prolong the QT interval. Decrease automaticity and conduction and prolong refactoriness. Amiodarone, sotalol, bretylium

Class IV: Slows calcium channel blockers, decrease automaticity and AV conduction. Verapamil, diltiazem

Class V: Adenosine, digoxin

CONDUCTION DISTURBANCES

Classification of conduction disturbances
Sinoatrial (SA) exit block

Atrioventricular (AV) block
1. 1st degree AV block
2. 2nd degree AV block:
- Mobitz type I (Wenkebach)
- Mobitz type II

3. High degree AV block
4. 3rd degree AV block

Intraventricular conduction defects

- bundle branch blocks, RBBB, LBBB, LAH, LPH

Associated diseases

- Sick sinus syndrome
- AV dissociation

Sinoatrial (SA) exit block

- Pause duration equal to a multiple of the underlying PP interval
- Often there is progressive shortening of the PP interval prior to the pause (SA Wenkebach)

Causes

- excessive vagal tone, ischemia, fibrosis, calcification of the conduction fibers, sick sinus syndrome, drug effect (digoxin, calcium channel blockers, antiarrythmic agents, sympatholytics).
- Usually asymptomatic.

Sick sinus syndrome

Forms

- Sinus arrest, SA exit block, persistent sinus bradycardia, bradycardia-tachycardia syndrome:

Causes

- Degenerative fibrosis of the conduction system.
- Common in sarcoidosis, amyloidosis, Chagas' disease, cardiomyopathies. Coronary artery disease is an uncommon cause.

Symptoms

Most patients are asymptomatic, but may experience syncope, dizziness, confusion, palpitations, heart failure, or angina. Holter monitoring is essential.

Therapy

- Oral theophylline, ephedrine
- Symptomatic patients will require pacing (ventricular or dual chamber pacing).

1st degree AV block

- PQ > 0.20 s but each P is followed by a conducted QRS.

2nd degree AV block 1: Mobitz type I (Wenkebach)

- The HR progressively lengthens, with the RR interval shortening before the blocked beat. Abnormal conduction is in the AV node.
- Occurs in normal individuals with heightened vagal tone, drugs (digoxin, calcium channel blockers, β-blockers, other sympatholytic drugs), ischemia, infarction, inflammatory process, fibrosis, calcification, infiltration.
- Prognosis is good, since reliable alternative pacemakers arise from the AV junction below the level of block if higher degrees of block should occur.
2nd degree AV block 2: Mobitz type II
- Is abrupt and is not preceded by a lengthening AV conduction time.
- Block is within the His bundle system.
- Almost always due to organic disease, involving the infranodal conduction system. In the event of progression to complete heart block, alternative pacemakers are not reliable.
- Prophylactic ventricular pacing is required.

3rd degree, complete AV block
- Due to a lesion distal to the His bundle and is associated with bilateral bundle branch block.
- P and QRS have no relationship to each other (AV dissociation).
- QRS is wide and arises from a ventricular pacemaker with a rate of less than 45/min.
- Exercise does not increase the rate. The first heart sound varies in intensity, wide pulse pressure, changing in systolic blood pressure level, canon venous pulsation in the neck vein are prominent.

Symptoms
- Asymptomatic, or weakness, dyspnea, syncope
- Therapy: pacemaker implantation.

Right bundle branch block (RBBB)
- QRS duration > 0.12 s in limb leads.
- rSR' in leads of the RV (V1 and V2).
- Wide S in leads of the LV(I, aVL, V5, V6). The T wave is usually positive here.

Significance
Occurs sometimes in normal individuals, transiently in pulmonary embolism, or RV strain of other cause, coronary disease, ASD, diastolic overload of the RV, myocarditis.

Incomplete RBBB: rSR' in RV leads, but the QRS is not wider than 0.12 s.

Left bundle branch block (LBBB)
- QRS duration > 0.12 s in limb leads.
- Wide, spiked R waves or RsR' in LV leads (I, aVL, V5, V6).
- Lack of q wave in the LV leads.
- Wide, cleft QS complexes in RV leads (V1, V2, sometimes V3).
- Directions of the ST and T waves are opposite to those of the QRS: ST depression and T inversion on LV leads, and ST elevation and positive T wave in RV leads (secondary changes of repolarization).

Incomplete LBBB: LBBB Tawara pattern in the appropriate leads, but the duration of the QRS is not longer than 0.12 s.

Left anterior hemiblock
- Small initial q wave in I, aVL, and a small r wave in II, III, aVF, respectively.
- Frontal R axis deviation to the left is over -30°.
- Deep S in II, III, aVF. SII > RII, and SIII > SII.
- No pathological widening of the QRS in any leads.

Left posterior hemiblock
- Often associated with RBBB
- Deep S in I, aVL, and rqR' in II, III, aVF.
• Frontal R axis deviation to the right is between +90 and +120°.
• R3 is usually > R2.
• No pathological widening of the QRS in any leads.
• Bifascicular, trifascicular blocks can occur.
• Prognosis and treatment: is that of the underlying disease.
• Indications for permanent pacing: symptomatic bradyarrhythmias, asymptomatic Mobitz type II AV block, complete heart block.

References
• Current diagnosis and therapy, ed. Thierney et al.
• ECG: Rohla M.: EKG alapismeretek