Bradyarrhythmias and Bundle brand blockages

Bradyarrhythmia

Bradycardic disorders are caused either by a malfunction in the sinus node or by a block in the conduction of the impulse from the atria to the ventricles. Sinus bradycardia is the most common.

Sinus bradycardia

This is a slowing of the heart rate < 60/min. Physiologically, it occurs in situations where vagal activity (sleep) predominates. The normal value is found in athletes, when it reaches even 40 beats per minute. **Possible causes** include: hypothyroidism, hypothermia, intracranial hypertension, AIM, inferior wall AIM, sick sinus syndrome. It is also often induced iatrogenically by beta blockers, verapamil, digitalis, amiodarone.

Therapy - in symptomatic individuals atropine 0.5-1 mg i.v. [1]

Sick sinus syndrome

It is a persistent or paroxysmal sinus bradycardia, up to sinus arrest. It may be interspersed with bouts of Atrial flutter/Atrial fibrillation. There is a disturbance of impulse generation, which may be functional/anatomical and transient/permanent.

Etiology:

- Idiopathic degenerative lesion in the SA node region SA node,
- IHD,
- cardiomyopathy,
- increased vagotonia,
- endocrinopathy,
- pharmacology,
- direct damage to the sinus node.

Clinical picture

Most patients are asymptomatic, otherwise palpitations, dizziness, confusion, presyncope, syncope, ↓ MCO (minute cardiac output).

Diagnostics:

On ECG we observe a slow or irregular sinus action, various alternate rhythms or, on the contrary, paroxysms of tachycardia. The negative influence of the autonomic nervous system is confirmed by the ability of the sinus node to increase heart rate during physical exercise (confirmed by ergometry). Holter ECG shows variability of heart rate by day and night, episodes of sinus pauses or the occurrence of other severe rhythm disturbances.

Therapy:

Asymptomatic patients do not require treatment. In case of bradycardia with haemodynamic disturbance we indicate pacemaker implantation.

Sinus arrest

This is a failure of varying length in the SA node. With a longer pause, a junctional or idioventricular contraction may occur. It may manifest clinically with syncope.

Therapy - in symptomatic individuals atropine 0.5-1 mg i.v. [1]

Sinoatrial blockade

It has three degrees, but only 3rd degree is of clinical significance, in which the impulse is not transmitted from the sinus node to the atrial myocardium → failure of one cardiac contraction. The entire P-QRS-T complex is absent from the ECG.

Therapy - in symptomatic individuals atropine 0.5-1 mg i.v.

Atrioventricular blockages

It is a defect in the transmission of the depolarizing wave to the ventricles. The blockage is most often in the AV node (suprahyseal), but can also occur in the His bundle (intrahyseal) or infrahyseal.

Etiology

- AIM (especially the lower AIM at the closure ACD),
- inflammation - viral myocarditis, borreliosis, Chagas disease,
- trauma,
- bradycardic drugs - digoxin, beta blockers,
- idiopathic fibrosis,
- cardiomyopathy.

Classification:

1st degree:

Prolonged AV conduction, therefore PQ > 0.2 s. Carditis in acute rheumatic fever, digoxin intoxication, β blockers. Clinically uncomplicated, it should be taken into account during medication with the above mentioned drugs.

2nd degree:

Intermittent interruptions of conduction from the atria to the ventricles (some impulses are not transmitted to the ventricles), P waves not followed by a QRS complex occur on the ECG.

A-V block II °, Mobitz type I (Wenckebach period):

With each transferred impulse, the disturbance intensifies and the time of conduction of the impulse from the atria to the ventricles increases. Eventually, the conduction disturbance escalates to the point that the atrial to ventricular excitation is not transferred. Temporary complete blockade of A-V conduction occurs. Failure to transfer the impulse allows the conduction system to "recover" and resume A-V transmission. This process repeats periodically. The interval from one complete A-V block to the next complete A-V block is called the Wenckebach period.

The more severe the disorder, the sooner the complete A-V block occurs and the shorter the Wenckebach period. The usual ratio of A-V transfer is 5:4, 4:3 and 3:2. In the extreme case, the Wenckebach period can be shortened so that a complete A-V block occurs after each successful A-V transfer. The transfer system can handle one atrial to ventricular transfer of excitation, but cannot handle another A-V transfer. This failure is called a fixed Wenckebach period. There is a 2:1 ratio of A-V transfer (every other P wave is transferred).

Fixed Wenckebach period, unlike Mobitz II A-V block, is not a clear indication for pacemaker implantation. An exception is symptomatic bradycardia in fixed Wenckebach period. For example, if the patient's S-A node rate drops to 60/min, then with a 2:1 A-V conversion, the QRS and ventricular systoles will be 30/min and this will usually already cause a significant drop in minute cardiac output. If S-A node function is good, the sympathetic system will resolve the 2:1 A-V block by stimulating the S-A node to a higher node frequency, e.g. to 120/min to maintain a QRS (ventricular systole) rate of 60/min.

ECG finding:

On ECG, each **QRS complex** is preceded by a **P wave**, the rhythm is sinus and the QRS complex lasts a **maximum of 0.12 s**. With each successive P-QRS complex, the **PQ interval** becomes progressively longer and longer as the A-V conduction disturbance increases. When complete A-V block occurs, the ECG shows a P wave (evidence of atrial depolarization) that is not followed by a QRS complex (evidence of complete A-V block). After a pause, a **P-QRS complex** appears on the ECG, usually with a normal P-Q interval (evidence of restored A-V conduction).

Lesion localization

The lesion in the transmission system is localized predominantly in the A-V node, less frequently in the His bundle. The localization of the disorder cannot be determined from the surface ECG, but only during invasive electrophysiological examination.

A-V block II °, Mobitz type II:

At a normal heart rate, the damaged site is unable to transmit all the control impulses from the A-V node to the ventricles. Each converted impulse induces a complete blockade of the A-V transmission (both bundle branches do not conduct). The fault site needs some recovery time to allow another A-V transfer to occur. At the mildest degree of failure, control impulses from the supraventricular area are transferred to the ventricles in a 2:1 ratio (P-P-QRS). At higher degrees of failure, the A-V transfer ratio increases to 3:1, 4:1, etc. With further progression, bundle branch completely stops conducting impulses and complete A-V block III° (there is conduction block in both bundle branches) occurs.

This type of A-V block is unstable and usually progresses to complete A-V block III° with ventricular bradycardia, or in the worst case with asystole and syncope (Adams-Stokes syndrome). Therefore, the **Mobitz II finding on ECG is very important** and is an indication for placing the patient on a monitored bed and for early pacemaker implantation. For the above reasons, the name Mobitz II should not be used for A-V blockade with a 2:1 conversion and a QRS complex shorter than 0.12 s. Reversible causes of A-V conduction disturbance should be excluded before indicating pacemaker implantation: unstable IHD, bradycardic drugs (beta-blockers, verapamil or diltiazem, digoxin), hyperkalemia and hypothyroidism. About 20% of patients with 2:1 A-V block have an intermittent lesion in the His bundle and a preexisting permanent conduction block in one of the Tavar arms (P-P-QRS...; and QRS wider than 0.12 s). In terms of lesion location, the lesion is a fixed Wenkebach period with an A-V conversion of 2:1, and this disorder does not usually carry the risk of significant bradycardia as Mobitz II° A-V block. However, these disorders cannot be distinguished from the normal ECG recording. In both cases, the QRS complex will be wider than 0.12 s, each QRS complex will be preceded by 2 P waves, and the P-Q interval will be constant.

ECG finding

On the ECG, there is a greater number of P waves per QRS complex (P-P-QRS ... P-P-P-QRS ... P-P-P-P-QRS). The PQ interval is constant (the ventricles are driven from the supraventricular area and if it is the S-A node it remains sinus rhythm. The QRS is wider than 0.12 s (there is a ventricular conduction disturbance - bundle branch block).

Lesion localization

The lesion in the transmission system is localized in one of the bundle branches in 80% of these disorders. The lesion is distal to the bifurcation of the bundle of His in a situation where the other bundle branch does not conduct at all (concomitant complete conduction block in the contralateral bundle branch).

3rd degree

Permanent complete A-V blockage. None of the supraventricularly generated impulses reach the ventricles.

It arises either as a progression of Mobitz II° blockade or by sudden damage to the conduction system, for example in acute myocardial infarction.

A-V block III° with ventricular rhythm is a clear indication for pacemaker implantation. Implantation should be waited in reversible causes of A-V conduction block (acute diaphragmatic myocardial infarction, bradycardic drugs, hyperkalaemia, hypothyroidism, infective endocarditis). In contrast, when A-V block III° occurs in acute anteroseptal myocardial infarction, pacemaker implantation is not delayed. A-V block III° with a QRS complex shorter than 0.12 s (40% of A-V blockades) usually does not result in severe bradycardia (the alternate ventricular control centre is supraventricular). If the surrogate centre has a rate greater than 40/min, this complete A-V block is not an indication for pacemaker implantation.

ECG finding:

On the ECG recording, **P waves appear independently of QRS complexes wider than 0.12 s** (there is a junctional or idioventricular rhythm, usually with SF < 40/min). There is A-V dissociation, in which P waves have a higher frequency than QRS complexes. A-V dissociation and A-V blockade can be reliably demonstrated by scribbling R-R intervals from the ECG on a free piece of paper. Comparison of the plotted R-R intervals will demonstrate mismatch with P-P intervals and shorter P-P intervals than R-R intervals.

Lesion localization

In 61% of all A-V III° blocks, the lesion is localized in one of the bundle branches with a concomitant previous conduction block in the contralateral bundle branch.

Therapy

1st degree

- not being treated.

2nd degree

- elimination of the potential cause - antiarrhythmic therapy, digoxin, hyperkalemia, myocardial ischemia, hypotension
- type I:
  - atropine: 0.02 mg / kg i.v., i.o., e.t., ie 0.1 mg / 5 kg (0.2 ml) in symptomatic individuals;
- type II:
  - alternatively isoprenaline 0.02 mg / kg;
  - permanent pacemaker implantation
- temporary cardiac pacing in symptomatic and asymptomatic patients with AIM (mainly anterior wall) with 2nd degree AV block, RBBB or LBBB;

3rd degree

- elimination of the potential cause - antiarrhythmic therapy, digoxin, hyperkalemia, myocardial ischemia, hypotension
- atropine or temporary pacemaker during AIM with AV blockage
- permanent pacing in chronic symptomatic AV blockages with signs of heart failure.

Principles of pacemaker implantation

Cardiostimulators			
Diagnosis	**Stimulation mode**		
	Optimal	Possible	Inappropriate
Sick sinus syndrome	DDD(R)	AAI(R)	VVI, VDD
AV blockade	DDD	DD	AAI, DDI, VVI + VA conduction
SSS + AV block or branch block	DDDR, DDIR	DDD, DDI	AAI, VVI
Permanent atrial fibrillation or atypical atrial flutter	VVI(R)	VVI	AAI, DDD
SSS or AV block + paroxysmal atrial arrhythmias	DDDR + AMS	DDIR, DDDRP + AMS	VDD, AAI
Heart Failure	DDD BiV	VVIR BiV	AAI, VDD
Hypertrophic cardiomyopathy	DDD + optimalized AV delay	VDD, DDDR + optimalized AV delay	AAI, VVI
BiV: biventricular stimulation, DDDRP: dual-cavity rate-responsive pacing using preventive atrial pacing algorithms in patients with paroxysmal atrial fibrillation			
taken from [2]			

Bundle branch blockages

Bundle Branch Blocks:

Right bundle branch block (RBBB)

Right bundle branch block (RBBB) is a disorder of myocardial impulse conduction due to impairment of the cardiac conduction system resulting in delayed depolarization (and therefore activity) of the right ventricle.

Types

According to the width of the QRS complex, there are 2 types of RBBB:

1. complete RBBB (QRS longer than 0.12 s, blockage of the proximal part of the right bundle branch);
2. Incomplete RBBB (QRS shorter than 0.12 s, blockade of the distal part of the right bundle branch).

The normal width of the QRS complex is 0.06-0.11 s.

Etiology

RBBB alone is haemodynamically insignificant. However, it may signal damage to the right heart. RBBB often occurs in:

- pulmonary chronicum (right heart pressure overload);
- cor pulmonale acutum (embolism a. pulmonalis, right heart pressure overload);
- atrial septal defect (right heart overload);
- ischemic cardiomyopathy;
- cardiomyopathy due to a valvular defect;
- congenital or idiopathic cardiomyopathy.

The ECG picture of right branch bundle blockage can also occur in healthy people. It is mainly an incomplete RBBB with normal QRS complex width in young endurance athletes (during endurance sports there is a volume load on the right ventricle).

Diagnostics

Diagnosis of RBBB is based on **ECG**. In case of **complete RBBB**:

- **QRS complex is dilated above 0.11 s** (3 small squares);
- in leads **V1-V2** (right-sided leads, above the right ventricle) we observe the image of **rSR´**, **descending ST segment depression** and **negative T wave** (for complete BPRT it is typical that **R´** is higher than **r**);
- in leads **V4-V6**, **I and aVL** (left-sided leads, above the left ventricle) we find a **deep and wide S oscillation** and a **positive T wave**.

In the case of incomplete RBBB, the QRS complex lasts less than 0.12 s.

Differential diagnostics

- Right ventricular hypertrophy,
- intraventricular block,
- non-specific conversion failure,
- Brugado syndrome,
- preexcitation syndrome,
- posterior myocardial infarction,
- ventricular rhythm.

Left bundle branch block (LBBB):

Left bundle branch block (LBBB) is a myocardial impulse conduction disturbance resulting from impairment of the cardiac conduction system, resulting in **delayed depolarization** (and therefore activity) of the left ventricle. The entire left ventricle is depolarized from the right side of the right Tawar arm, causing widening and morphological change of the QRS complex.

Types

We distinguish according to the width of the QRS complex 2 BLRT types:

1. complete LBBB (QRS longer than 0.11 s);
2. incomplete LBBB (QRS in the range of 0.06-0.11 s).

The normal width of the QRS complex is 0.06 – 0.11 s.

Etiology:

LBBB alone is haemodynamically insignificant. However, it signals damage and/or increased left ventricular workload, which may be due to the following conditions:

- cardiomyopathy,
- left heart valvular defects,
- hypertension (hypertensive cardiomyopathy),
- IHD (ischemical cardiomyopathy).

Complications

LBBB increases the risk heart failure, IM, sudden heart death, 2nd degree AV block, 3rd degree AV block.

Diagnostics

LBBB diagnostics is based on ECG. In case **complete LBBB**:

- The QRS complex is dilated over 0.11 s (3 small squares) and notched, resembling the letter "M" (RsR´) in V6;
- in V1 we observe a QS or qRS image (qRS resembles the letter "W");
- in the lateral leads (V5, V6, I, aVL) there is T-wave inversion and descending depression of ST segments (=secondary repolarization changes);

- the axis is normal or deviated to the left.

In case of incomplete LBBB, the QRS complex lasts 0.06-0.11 s.

Attention! CAVE! LBBB makes IM diagnosis impossible. If we suspect an IM (pain of coronary origin) in a patient with left bundle branch block, it is always necessary to hospitalize this patient!!

Differential diagnostics

- Left ventricular hypertrophy,
- Lateral IM,
- Preexcitation syndrome.

Bundle branch block therapy

It is governed by the presence and severity of the underlying organic disease. Chronic, incidentally diagnosed brachial plexus blockade is not an indication for any treatment; in acute myocardial infarction, the introduction of temporary pacing is considered. [3]

Links

Related articles

- Manifestation of faults in the formation and conduction of excitement on the elctrocardiogram
- Antiarrhythmics
- Radiofrequency catheter ablation
- Electrophysiological examination
- Cardiac transmission system

Reference

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