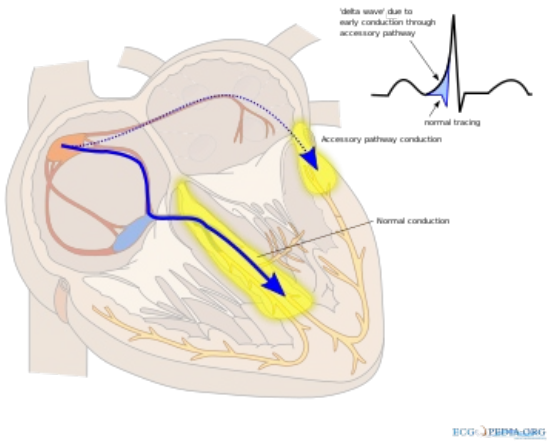
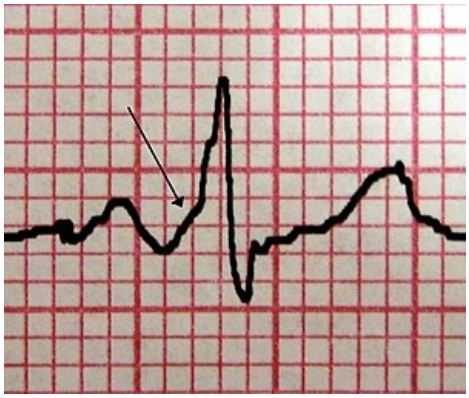
Wolff-Parkinson-White syndrome

Ventricular preexcitation syndrome Under physiological conditions, the atrial myocardium is electrically connected to the ventricular myocardium **only** via the **bundle of His**, which penetrates through the fibrous skeleton into the interventricular septum. The impulse from the SA node spreads as follows: SA node → internodal atrial junctions → **AV node** → **bundle of His** → bundles of Tawar → Purkinje fibers → ventricular myocardium.



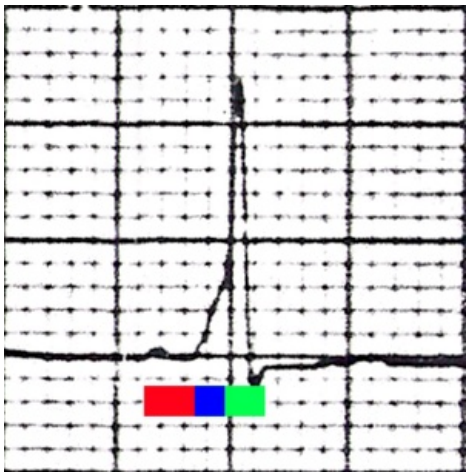
In the case of preexcitation syndromes, in addition to the bundle of His, there is an **another connection between the atria and the ventricles**. These so-called ``accessory pathways *(accessory conduction bundles) do not connect to the AV node, therefore there is ``no* slowing down of conduction from the atria to the ventricles, which results in ``premature activation *(excitation) of the myocardium of the ventricles (→ ventricular preexcitation syndrome).*



Accessory bundles directly connect the atria to the ventricles. In most patients with ventricular preexcitation syndrome, they are located in the left heart.

The 3 most well-known ventricular preexcitation syndromes include:

1. **WPW syndrome** (Wolf-Parkinson-White syndrome);
2. **LGL syndrome** (Lown-Ganong-Levine syndrome);
3. **Mahaim type of preexcitation**.



Wolff-Parkinson-White syndrome (WPW) is the most common ventricular *preexcitation* syndrome. This is a cardiac arrhythmia in which the impulse (wave of depolarization) spreads from the atria to the ventricles **outside** of the AV node. The accessory path here consists of the so-called **Kent's bundle** (= pathological connection between atria and ventricles).

Etiology

- Most often it is a *congenital* disease, which is associated with Ebstein's anomaly, tuberous sclerosis, etc.
- Of the **acquired** causes, WPW syndrome can cause e.g. endocarditis or myocarditis.

In a small group of patients, the cause of WPW syndrome is a mutation of the ``PRKAG2 *gene. This gene encodes a protein that is part of an enzyme called* ***AMP-activated protein kinase*** (AMPK) [1].

There is also a familial form of WPW syndrome (AD inheritance), but it is extremely rare [2].

Symptoms

WPW syndrome can manifest itself with the following symptoms:

- dizziness, syncope;
- palpitations (feelings of a pounding heart);
- a feeling of "shortness of breath", the need to "breathe".

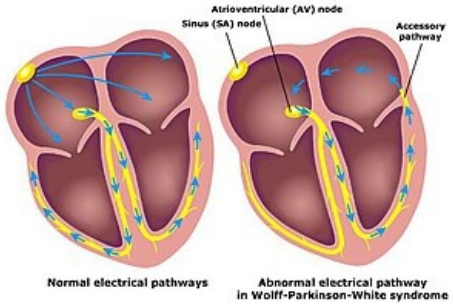
Diagnostics

The diagnosis is based on the **ECG** picture. In WPW syndrome, the following changes are observed on the ECG:

- **shortening of the PQ (PR) interval** → PQ ≤ 0.12 s (there is no delay of the depolarization wave in the AV node);
- **delta wave** (the beginning of the QRS complex is deformed by a *delta wave*, the myocardium of the ventricles is overexcited);
- the second half of the QRS complex is normal (the wave of depolarization that reached the ventricles via the bundle of Kent meets the wave of depolarization that reached the ventricles normally via the bundle of His);
- **QRS complex widening** above 0.10 s (QRS complex widening may occur due to delta wave).

In symptomatic patients (WPW + tachyarrhythmia) an electrophysiological examination is also necessary.

Complications ==



Abnormal junction of the ventricles with the atria (bundle of Kent) may be associated with other arrhythmias. The most common is **paroxysmal supraventricular tachycardia'**, less often permanent supraventricular tachycardia, rarely ventricular tachycardia. Very rarely, WPW syndrome can result in ventricular fibrillation and sudden cardiac death.

The danger lies in the formation of the so-called *reentry circuit* and the so-called *AV reentry tachycardia* (AVRT, atrioventricular reentry tachycardia) [3].

- In the case of *orthodromic AVRT*, the wave of depolarization propagates from the atria to the ventricles normally via the bundle of His and back from the ventricles to the atria via the bundle of Kent (95%).
- In the case of *antidromic AVRT*, the wave of depolarization spreads from the atria to the ventricles via the bundle of Kent and from the ventricles to the atria via the bundle of His (5%).

→ In both cases, the atria are reactivated and a pathological circuit is formed, which results in tachycardia.

Treatment

Asymptomatic patients' - treatment is not necessary (catheter ablation is indicated in patients with risky occupations: pilots, drivers, ...) [4].

Symptomatic patients [5]

1. Cancellation of an acute paroxysm of tachyarrhythmia: antiarrhythmics I. or III. classes.
2. Pharmacological treatment: single administration of an antiarrhythmic drug at the beginning of a tachycardia paroxysm or long-term regular antiarrhythmic therapy.
3. Catheter ablation (as part of an electrophysiological examination): radiofrequency disruption of Kent's bundle.

Links

Related Articles

- Lown-Ganong-Levine syndrome
- Conduction system of the heart

External links

- Wolff Parkinson White (WPW) syndrome (TECHmED) (https://www.techmed.sk/wolff-parkinson-white-wpw-syndrom/)
- ECG localization of Kent's bundle (TECHmED) (https://www.techmed.sk/kentov-zvazok-lokalizacia-pri-wpw-syndrome/)
- Podcast about WPW on kardioblog.cz (https://kardioblog.cz/wpw/)

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