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Clinical Pharmacological Approach To The Use Of Antiarrhythmic Drugs In Children

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Abstract. The clinical pharmacological approach to the use of antiarrhythmic drugs in children is crucial for ensuring effective and safe treatment of cardiac arrhythmias. This article explores the classification, mechanisms of action, indications, contraindications, and potential adverse effects of commonly used antiarrhythmic agents in pediatric patients. Special attention is given to the pharmacokinetics and pharmacodynamics of these drugs in children, considering age-related differences in drug metabolism and response. The importance of individualized therapy, monitoring strategies, and recent advancements in pediatric cardiology are also discussed.
Keywords: Antiarrhythmic drugs, pediatric cardiology, arrhythmia treatment, clinical pharmacology, drug safety, pharmacokinetics, pharmacodynamics, individualized therapy.

Introduction

Treatment of cardiac arrhythmia (CRA) in children is one of the most serious problems in the practice of both a pediatrician and an "adult" cardiologist. Despite the commonality of many therapeutic approaches, the peculiarities of the physiological development of the child, the absence of typical adult causes of arrhythmia development in children often determine slightly different pathogenetic mechanisms of CRA development and approaches to treatment [1].

Materials And Methods

In the treatment of atrial fibrillation, there are drug and non-drug methods. Non-drug methods include surgical, minimally invasive methods of electrotherapy (radiofrequency catheter ablation) and the use of implantable antiarrhythmic devices. Drug methods can be divided into emergency relief of the most dangerous forms of atrial fibrillation and chronic pharmacotherapy.

Emergency relief is required for atrial fibrillation with a high risk of developing heart failure, circulatory arrest and sudden death - primarily ventricular tachycardia (VT) turning into fibrillation and bradyarrhythmia. To a much lesser extent, supraventricular paroxysmal tachycardia (SPT) can be the direct cause of circulatory arrest in children over one year old. However, in infants, SPT poses a much greater threat as a possible cause of sudden death.

When SPT develops, treatment begins with vagal tests. In children, their effectiveness is maximum in the first 20-30 minutes after the onset of the attack. If the attack cannot be interrupted during this time, they proceed to the administration of antiarrhythmic drugs - AAD. The drug of choice in this case is adenosine (ATP). The clinical effectiveness of ATP is due to the rapid onset of action (up to 10 s), minimal possible side effects (cough, feeling of heat, hyperemia, bradycardia), which quickly pass. The initial dose is administered as a 1% solution intravenously, quickly (3-4 s), without dilution, at a dose of 0.1 mg / kg. For emergency relief of an attack, you can also focus on the age-related dosages of the drug: up to 6 months - 0.5 ml; 6 months - 1 year up to 0.7 ml; 1-3 years - 0.8 ml; 4-7 years - 1.0 ml; 8-10 years - 1.5 ml; 11-14 years - 2.0 ml. If the rhythm is not restored within 1-2 minutes, a double dose is administered again and, if necessary, repeated once more [2].

Results And Discussion

In the development of SPT in patients with Wolff-Parkinson-White syndrome (WPW), the introduction of giluritmal (ajmaline) at a dose of 1 mg/kg, intravenously, but not more than 50 mg, is effective. The relief of SPT with isoptin also remains relevant. Although the drug can cause bradycardia and arterial hypotension, it is effective in the treatment of children with polytopic atrial tachycardia. The following doses of isoptin are used: up to 1 month - 0.2-0.3 ml; up to 1 year 0.3-0.4 ml; 1-5 years - 0.4-0.5 ml; 5-10 years - 1.0-1.5 ml, over 10 years - 1.5-2.0 ml. Isoptin is absolutely contraindicated in tachycardia of

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unknown etiology with a wide QRS complex and WPW syndrome, since it is possible for SPT to transform into ventricular fibrillation due to acceleration of anterograde conduction along additional conduction pathways. The drugs of choice for stopping SPT in children are also cordarone (IV with 5% glucose solution at a dose of 5 mg/kg), digoxin (IV, slowly, in saline, at a dose of 0.1-0.3 ml), novocainamide (IV, slowly, in saline, at a dose of 0.15-0.2 ml/kg, up to a maximum of 17 mg/kg). Possible arterial hypotension is prevented by administering a 1% solution of mesaton at a dose of 0.1 ml per year of life, but not more than 1.0 ml. The effect of AAP in SVT is enhanced by the combined use of tranquilizers with a sympatholytic effect (relanium, tazepam, radedorm). Tachycardia with a wide QRS complex detected on the ECG during an attack does not always allow determining whether it is VT or SVT with conduction aberration or bundle branch block. If the form of arrhythmia is not precisely determined, treatment is carried out as for VT. Calcium antagonists are contraindicated in all cases. The first-line drug for stopping VT is lidocaine, which is administered intravenously, slowly, in a 5% glucose solution, at an initial dose of 1.0 mg / kg as a 1-2% solution. If the rhythm is not restored, the drug can be additionally administered every 5-10 minutes in half the dosage (up to a total dose of no more than 3 mg / kg). As second-line drugs for VT, one can use novocainamide, giluritmal, cordarone or β-blockers (dosages are indicated above). The drug of choice for stopping an attack of pirouette tachycardia, typical for patients with prolongation of the QT interval, is magnesium sulfate (administration of a 10% solution of 25-50 mg/kg, maximum 2 g over 1-2 minutes, if ineffective - repeat after 5-10 minutes). Of bradyarrhythmias, symptomatic bradycardia, asystole or electromechanical dissociation (the presence of sinus bradycardia on the ECG in the absence of a pulse wave) require emergency treatment. The main measures for the development of asystole in children are the administration of adrenaline and atropine. If small doses of adrenaline (0.01 mg/kg/ IV) are ineffective and cardiac arrest develops, higher doses are administered (0.1-0.2 mg/kg IV), which can be repeated every 3-5 minutes as long as there is a risk of arrhythmia recurrence. As a rule, atropine is used to treat bradyarrhythmias after adrenaline. The IV dose is 0.02 mg/kg (maximum single dose is 0.5 mg in young children and 1.0 mg in adolescents), which can be repeated every 5 minutes (up to a total dose of 1.0 mg in young children and 2.0 mg in adolescents). Long-term pharmacotherapy of ARS in children is based on the correction of intra- and extracardiac mechanisms of their development [3]. In the first case, we are talking about the use of traditional cardiology AAPs that directly affect the electrophysiological mechanism of triggering and maintaining arrhythmia (the so-called symptomatic therapy). Their use is quite effective, however, with long-term treatment, almost all of them give side effects and have a proarrhythmogenic effect. Taking into account the proven importance of the autonomic and central nervous system in the pathogenesis of the development of NRS in children, in the treatment regimen for idiopathic arrhythmias in pediatrics, a significant role belongs to drugs that normalize the level of cardiocerebral interactions, which is the basis of the so-called basic antiarrhythmic therapy. It includes nootropic, membrane-stabilizing and metabolic drugs. Nootropics (piracetam at a dose of 0.2 ml three times a day; pyriditol 0.05-0.1 ml three times a day; aminalon 0.5-1.0 ml three times a day; glutamic acid from 0.1 to 1.0 ml three times a day; phenibut 0.05 - 0.3 3 times a day) stimulate ATP synthesis and have a vagolytic effect. The recommended course of treatment is 4-6 weeks of taking each drug, while no more than two drugs are prescribed at a time. Among other drugs, carnitine chloride, coenzyme Q, mildronate should be noted. Xidifon is actively used as an antioxidant drug in the treatment of children. In recent years, magnesium preparations, in particular magnerot, have found wide application in the complex therapy of NRS. The compound of magnesium with orotic acid summarizes their positive metabolic and antiarrhythmic effects. Recommended dosages: Children under 6 months 40 mg per day, 6 months-1 year - 60-75 mg per day, 1-3 years - 80 mg per day, 4-6 years - 120 mg per day, 7-10 years - 170 mg per day, 11-14 years 270 mg per day, over 15 years 280-400 mg per day.

No more than three representatives of different groups are prescribed at the same time.

In the presence of organic myocardial damage (cardiomyopathy, heart defects, carditis, etc.), idiopathic arrhythmia persisting for more than 6 months against the background of basic therapy, the development of hemodynamic disorders or arrhythmogenic symptoms, classical AAPs of class I-IV are added to the treatment. The first class includes AAPs that block fast cardiomyocyte channels (potassium - class IA and sodium IB and IC), the second - β-blockers, the third - agents that increase the duration of action, and the fourth - calcium ion antagonists. This division is quite arbitrary, since it does not take into

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account many electrophysiological properties of AAPs and does not include a number of effective AAPs that are additionally identified. Below, we will focus on the main AAPs used in children.

Quinidine is the longest used of the AAPs of class IA. Per os it is used in the form of sulfate or gluconate. When taking gluconate, the peak concentration occurs after 3-4 hours (taken three times a day), sulfate - after 1-2 hours (taken four times a day at a dose of 30-60 mg / kg). Of the side cardiac effects, the most significant is the possibility of developing bradyarrhythmia and prolongation of the QT interval. Gastroenterological or neurological disorders are possible. The practical use of quinidine in pediatrics is currently small, although it can be the drug of choice in the treatment of atrial fibrillation. Novocainamide is used primarily for emergency termination of tachyarrhythmia (see above). Oral administration at a dose of 40-100 mg / kg / day. The daily dose is divided into six doses for infants, four for children 1-2 years of age, and three doses at an older age. In addition to bradycardia and hypotension, side effects may include prolongation of the QT interval, lupus syndrome, arthralgia, and rash.

Unlike quinidine and novocainamide, neogilurytmal does not have a pronounced hypotensive effect. It has a negative inotropic and moderate adrenolytic effect. The drug is highly effective in WPW syndrome. The saturation dose is 20 mg every 6-8 hours (3 days), then 20-40 mg per day. For the prevention of attacks, a single dose of 10-20 mg in the morning is possible. Complications are AV blocks, bradyarrhythmia, prolongation of the QT interval. Of the class IB drugs (local anesthetics), mexiletine (mexitil) is promising when taken per os. There is evidence of its effectiveness in patients with long QT syndrome with sodium channel defect. The starting dose of the drug is 2 mg / kg, divided into 4 doses per day. A single dose can be increased to 5 mg / kg in older children and up to 7-8 mg / kg in infants.

The class IC drug flecainide reaches its peak concentration in 1-2 hours. The body surface area dose in children correlates more closely with the plasma drug level than the body weight dose. The starting dose in infants is 80-90 mg/m2 per day, divided into two doses. At an older age, the dose is increased to 100-110 mg/m2 per day (maximum up to 200). There is data on the effective use of the drug in pediatrics in combination with cordarone and mexiletine in the treatment of resistant forms of VT. Ritmonorm (propafenone) has been widely used in pediatrics in recent years. The drug reaches its peak concentration when taken per os in 2-3 hours. The dose of the drug is 10-20 mg/kg in four doses or, based on the body surface area, 150-200 mg/m2 per day with a maximum dose of up to 600 mg/m2 per day. Side effects of flecainide and rhythmonorm are common (prolongation of the QT interval, AV blocks, intraventricular blocks, paresthesia and transient disturbances

Conclusion

The effectiveness and safety of therapy for arrhythmia is significantly increased by using chronotherapeutic regimens based on determining the daily type of arrhythmia using Holter monitoring data and taking 2/3 of the daily dose of AAP with calculation of the maximum of its effect before the expected time of intensification or occurrence of arrhythmia.

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