REVIEW ARTICLE

A practical approach to rhythm disorders in pediatrics

Abordagem prática dos distúrbios de ritmo em pediatria

Roberta da Silva D'Alessandro Tonello[1], Helen de Menezes Monteiro[1], Henrique de Assis Tonelli[2], Alexandre Rodrigues Ferreira[3]

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ABSTRACT

Arrhythmias may be caused by changes in the generation of cardiac electrical stimulus, in stimulus conduction through the heart, or by a combination of both. The general pediatrician it is not usually expected to be able to identify and treat all cardiac arrhythmias, referring to the cardiologist for this task. However, pediatrician are expected to be able to identify and treat arrhythmias manifested in urgent or emergency care, i.e. those that lead to hemodynamic instability or to shock, given that, in such cases, the patient's life depends on how fast the therapeutic measures are taken. In this article we aim to demonstrate some basic pointers that facilitate the assessment and primary approach to the main pediatric cardiac arrhythmias and to guide in the first and main steps of treatment.

Key words: Arrhythmias, Tachycardia, Bradycardia; Emergency Care; Children.

RESUMO

As arritmias podem ser causadas por alterações na geração do estímulo elétrico cardíaco, na propagação deste estímulo através do coração ou pela combinação de ambas. Normalmente, não se espera que o pediatra geral seja capaz de identificar e tratar todas as arritmias cardíacas, ficando essa tarefa para o cardiologista. Entretanto, é função do pediatra saber identificar e tratar as arritmias que se manifestam como urgência ou emergência, ou seja, as que levam à instabilidade hemodinâmica ou choque, pois, nesses casos, a vida do paciente depende da rapidez com que as medidas terapêuticas são tomadas. Visa-se, neste artigo, demonstrar pontos básicos para facilitar a avaliação e abordagem primária das principais arritmias cardíacas pediátricas e orientar os primeiros e principais passos do tratamento.

Palavras-chave: *Arritmias; Taquicardia, Bradicardia; Atendimento de urgência; Criança.*

[1] Horizontal Care Physician of Pediatrics ICU at the Hospital das Clínicas, Universidade Federal de Minas Gerais – UFMG. Belo Horizonte, MG – Brazil.
[2] Pediatric Cardiologist. Pediatric Cardiology, Hospital das Clínicas, UFMG. Belo Horizonte, MG – Brazil.
[3] Associate Professor of the Department of Pediatrics at the UFMG School of Medicine. Belo Horizonte, MG – Brazil.
[4] Medical Coordinator of the Functional Pediatrics Unit of the Hospital das Clínicas, UFMG. Belo Horizonte, MG – Brazil.

INTRODUCTION

Heart rhythm disorders occur as a result of abnormalities or lesions in the intracardiac formation and conduction system or the contractile myocardium. The identification of arrhythmias presented as pediatric urgency or emergency in does not require any significant knowledge of electrocardiography. To define the initial therapeutic conduct all that is required is the ability to differentiate, through quick clinical evaluation, between bradyarrhythmias, tachyarrhythmias and pulseless rhythms or heart failure.[1,2] In case of tachycardia, important extra information must be obtained from an electrocardiogram (ECG), so it is essential to be able to assess whether QRS is widened.[1] In cardiorespiratory arrest (CRA) visual identification of the electrocardiographic pattern reveals the arrest rhythm

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Institution:
Hospital das Clínicas, UFMG – Functional Pediatrics Unit. Belo Horizonte, MG – Brazil

Corresponding Author:
Alexandre Rodrigues Ferreira
E-mail: alexfer1403@gmail.com

and refine options for cardiorespiratory resuscitation procedure.

Hemodynamic evaluation, used to define whether the patient is stable or if s/he presents signs of being in shock, must be fast and performed routinely, as with any patient suspected of instability, regardless of cause.[2] Pulse analysis has its own peculiarities, especially in tachyarrhythmias, when amplitude may be reduced, even in the absence of low cardiac output, due to restriction to diastolic filling (diastole time reduction) and consequent reduction in ventricular systolic volume. It is, then, risky to consider low amplitude of pulse as an isolated indication of hemodynamic instability in tachycardias.[2]

In severe status patients or those who present risk factors for arrhythmias, continuous electrocardiographic monitoring is mandatory. Included in that group are all the patients in intensive treatment for shock, respiratory failure or other vital dysfunctions, post-resuscitation, depression of sensorial system, or during general anesthesia and deep sedation.[1] It is important to highlight that in those cases monitoring with a pulse oxymeter should not replace the use of electrocardiographic monitoring and that the latter does not eliminate the need of intermittent and frequent clinical evaluation.[1,2]

Unlike adults, whose primary arrhythmias are more frequent, arrhythmias in children are often secondary to other conditions, especially when associated with acidosis, shock, hypoxemia, or metabolic disorders. Primary arrhythmias are less common, although not too rare. Arrhythmias that are manifested as emergencies are bradyarrhythmias and tachyarrhythmias (supraventricular tachycardia and ventricular tachycardia).[3,4]

The goal of this paper it to approach the main arrhythmias potentially present on severe status children and that characterize urgency or emergency situations pediatricians may face. We will not address cardiac arrest rhythms in this paper. The following classification will be used for prescription of treatment resources, according to evidence of success:

- **class I –** definitely recommended;
- **class II –** acceptable indication
  - a = probably beneficial;
  - b = possibly beneficial;
- **indeterminate class –** no conclusive evidence of utility or risks;
- **class III –** unacceptable indications (no proven benefit or potentially harmful).

BRADYARRHYTHMIAS

Bradyarrhythmias are the most commonly observed rhythms in children. There are many possible causes, among which the most frequent are hypoxia, acidosis, and shock. Other causes that must be remembered are vagal reflex, hypoglycemia, hypothermia, intracranial hypertension, congenital or acquired atrioventricular block, and intoxication caused by digitalis or beta-blockers. [2,4]

Bradycardia in children is defined as HR below 60 bpm or values above 60 bpm progressing to a rapid fall and with associated symptoms. General diagnosis of slow pulse is enough to guide initial therapeutics. Additional information to the ECG test on the arrhythmia mechanism are usually unnecessary, since different causes may share the same mechanism and identifying the type of disorder does not, in principle, modify the therapeutic approach proposed. Among the most commonly found mechanisms we find sinus bradycardia and atrioventricular block, with or without escape rhythms (junctional or idioventricular slow rhythms).[2,4]

Emergency treatment is only indicated for symptomatic patients with signs of instability. Asymptomatic or oligosymptomatic stable patients should be monitored, and wait for a cardiologist to be present. Emergency treatment is based on the reversion of the most important causes (hypoxia, hypercapnia, acidosis, or shock) by checking for adequately patent airways, ventilation and oxygenation, and by addressing tissue perfusion. In the absence of response, thoracic compressions are to be initiated.[3] If bradycardia persists, drug therapy is the next step, adrenalin being the first choice for children (class Ia indication), followed by atropine (class IIb). Atropine is the first choice in bradycardia mediated by vagal reflex, if it is the outcome of atrioventricular blocking (class I) and in poisoning by organophosphorus.[2,5]

The persistence of bradycardia despite those measures suggests more severe cases or cases with more complex etiologies, structural changes in the electrical stimulus formation, or conduction system. In these cases, use of chronotropics in continuous infusion (dopamine, adrenalin, isoproterenol) is a good choice for attempting to stabilize the patient and buy time for discussion and definition of more precise therapeutic alternatives.[5] A temporary pacemaker (transcutaneous, transesophage-

al, transvenous) is a feasible stabilization option (class IIb) for cases in which drug therapy fails. Later use of permanent pacemaker is very likely in these cases. Experience in Pediatrics is, however, limited, and the use of temporary pacemakers is not considered useful in the cases of bradycardia associated to cardiac stress by hypoxia, acidosis, or shock. (Figure 1).

TACHYARRHYTHMIAS

After identifying that rhythm is too fast for the age (tachyarrhythmias), it is be necessary to verify by ECG the type of QRS found. QRS with duration < 0.09s are classified as narrow, while QRS with duration > 0.09s are considered widened.[2]

Tachyarrhythmias with narrow QRS complex: sinus tachycardia

It is a physiological response to various situations, normal or pathological, experienced by the body. Among the possible causes we can highlight: anxiety, irritability, pain, crying, physical effort, fever, hyperthermia, anemia, hypervolemia, infection, congestive heart failure (CHF), hypoxia, acidosis, shock, use of adrenergic drugs, hyperthyroidism, and pheochromocytoma, among others.[2]

Its electrocardiographic characteristics include a variability from heartbeat to heartbeat, with activity or stress level alterations, heartbeat rate lower than 220 bpm in infants and lower than 180 bpm in children, P waves present in the normal axis (positive in D1 and in aVF), PR interval constant and with normal duration, R-R interval variable and narrow QRS complex.[2,4]

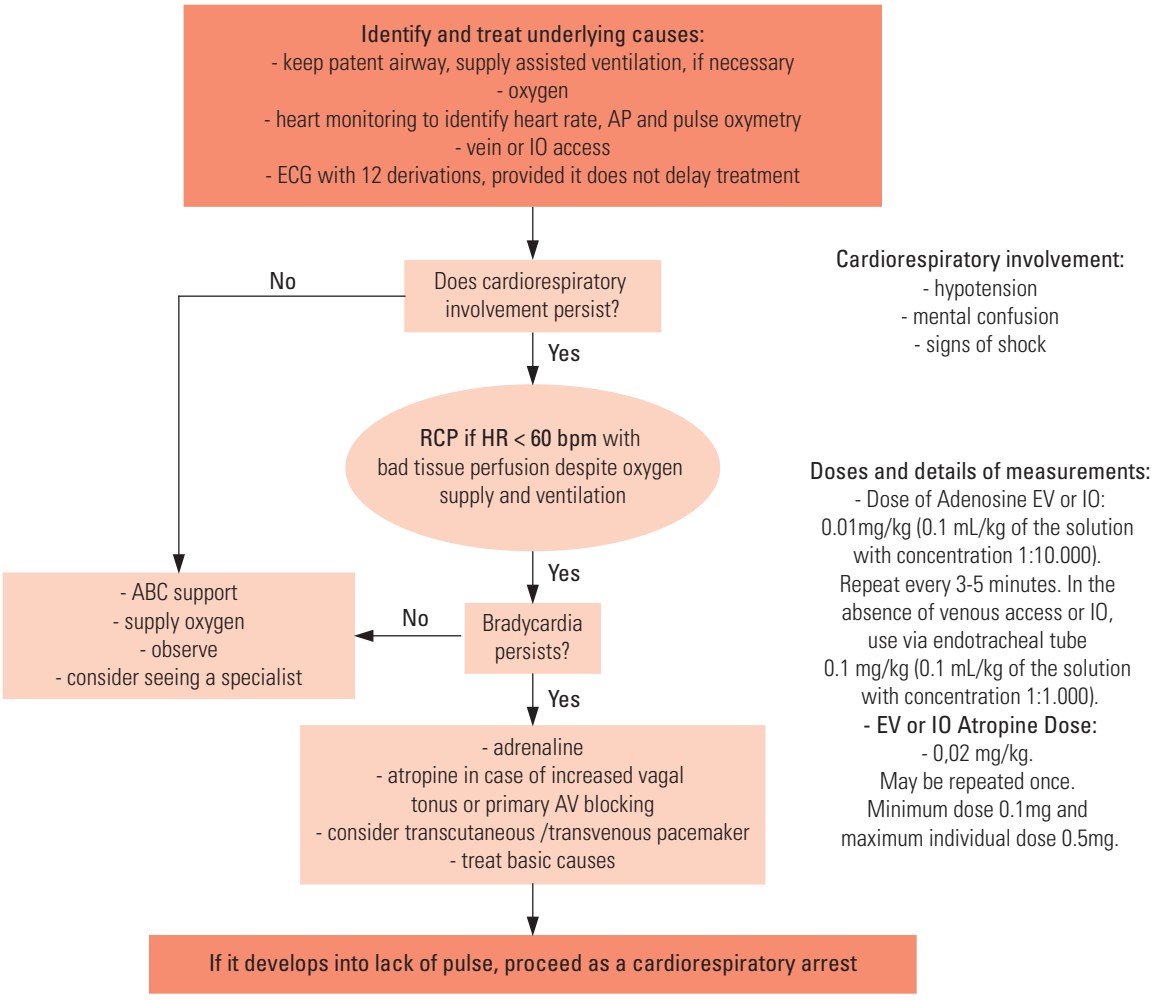

Figure 1 - Algorithm flowchart of pediatric care to bradycardia with pulse and with hemodynamic involvement.

Cases of sinus tachycardia situations in severe status patients under hypoxia, acidosis, heart failure or in shock should be highlighted. In such cases, existing instability and high heart rate values may make differential diagnosis more difficult with supraventricular tachycardia (more difficult identification of P on tracing).[1] Treatment is aimed to reverse the cause, and there is n point in using of drugs to reduce.[2]

Tachyarrhythmias with narrow QRS complex: supraventricular tachycardia (SVT)

A large variety of tachyarrhythmias can be classified under this denomination, with different electro pathological mechanisms. However, we are interested in the SVT by reentry involving the A-V node, for this is the most relevant kind in Pediatrics, both in frequency and potential severity. This form of tachycardia represents around 95% of the SVTs in any pediatric age group.[3,4] In most cases, there are no structural changes of the conditions associated to SVT by reentry in A-V node. It can, however, be associated with Ebstein's anomaly, L--transposition of the great vessels of the base, IVC, IAC, aortic stenosis, endocardial fibroelastosis, coarctation of aorta, Tetralogy of Fallot, tricuspid atresia, etc.[4]

SVT is the type of arrhythmia that most frequently requires emergency intervention in Pediatrics. Around 50 to 60% of the cases present their initial episode during the first year of life, particularly in the first three months. The cause is a reentry mechanism involing the A-V node, with or without an accessory conduction pathway (anomalous part out of the A-V node). Accessory pathways constitute the most common situation in any pediatric age group.[4]

The most marked characteristics of SVT by reentry are: typically abrupt beginning and ending (paroxysmal), relatively fixed heart rate (little variability from heartbeat to heartbeat), heart rate above 220 bpm in infants and above 180 bpm in children, absent P waves (or with abnormal axis, usually deforming the end portion of QRS), constant R-R interval and often narrow QRS complex. It may revert spontaneously and reoccur at very variable intervals. It should be remembered that in less than 10% of the cases SVT can be found with widened QRS. Clinical status varies from asymptomatic tachycardiac patients to those with ICC manifestations or in shock. Newborns and infants, due to their lower functional reserve, are the biggest candidates for clinical manifestations, particularly when arrhythmia persists for several hours or reaches frequencies above 280 bpm. Patients with cardiomyopathy may develop early symptoms. Clinical manifestations of ICC or low heart output in patients over 5 years old are rare. Besides the clinical manifestation of ICC and shock, we can highlight other more specific findings: palpitation, precordial heartbeat or cervical vessels with evidently accelerated heartbeat, thoracic discomfort, dizziness or syncope.[3]

Treatment varies according to the patient's clinical condition. In addition to routine patient monitoring measures, electrocardiogram tracing (with, at least, 12 derivations) and contacting the cardiologist, some therapeutic options are at the pediatrician's reach.[2]

STABLE PATIENTS

Vagal maneuvers may be easily tried in any stable patient before cardioversion with drugs, with variable success rates. The vagal reflex creates a transitory blocking of the A-V node that breaks the reentry circuit. In infants, the vagal maneuver may be performed with an ice bag on the patient's face for about 20 seconds (diving reflex), as long as the airways remain unobstructed (class IIa). In older patients, alternated carotid sinus massage (on the carotid and on the highest spot possible), on each side of the neck, and the Valsalva maneuver are the most indicated actions (class IIb). Compression of the eyeball is contraindicated to induce vagal reflex due to the risk of retina displacement.[3,4]

Regarding drug treatment, adenosine is the drug of choice for stable patients (class IIa) in any age group. It presents ultrashort half-life (< 10 seconds) as well as duration of action (< 2 minutes), and is very effective for quickly and safely converting arrhythmia. Adenosine is not used for maintenance treatment due to its ultrashort action. Response to adenosine is of important semiological value, indicating that the arrhythmia at hand must, actually, be an arrhythmia caused by a SVT by reentry involving the A-V node (other forms of SVT are not often convertible with adenosine). Administration of the drug must be made intravenously (IV) or by intraosseous infusion (IO), by the rapid *bolus technique*: two syringes interconnected by *three--way* with the drug *bolus* in the first syringe (closer to the patient) immediately followed by a *flush* of saline solution (at least 5 mL) in the second syringe. During the *bolus*, it may be interesting to try to register the conversion by electrocardiogram.[2-4]

Amiodarone and procainamide may be used for cardioversion in stable patients or even for maintenance after drug-based cardioversion, especially in reoccurring SVTs, at short intervals, constitute important existing alternatives (class IIb). Due to the potentially severe, though not frequent, side effects, use of both drugs drugs must be limited to the hospital, ministration being performed always under electrocardiographic monitoring. A good response to the loading dose is followed by continuous infusion of the drug; the time of use of the infusion must be discussed with the cardiologist (see the tachycardias algorithm).[4]

Synchronized electrical cardioversion (SEC) may be necessary for cases that respond neither to vagal maneuvers nor to medication. It is better for a cardiologist to indicate the procedure and it should be done quietly, under sedation, analgesia, and assisted ventilation (by mask or endotracheal tube).[2,4] See below the detailed description of the SEC procedure.

UNSTABLE PATIENTS

Treatment for unstable patients must be performed by SEC or by adenosine bolus. The alternative that allows for the fastest treatment should be preferred. SEC procedure should not be delayed for more than 30 seconds for the venipuncture attempt, sedation, intubation or preparation of boluses of adenosine.[6,7]

SEC consists of the synchronized depolarization of a critical myocardium mass, interrupting the mechanisms of tachyarrhythmias. It allows the sinus node to then resume control over heart rhythms. The higher the current through the myocardium, the better the chances of success are for cardioversion. Therefore, all effort to reduce impedance of the thoracic shovel-wall system is important to optimize the procedure. Thus, choice of adequate-sized shovels, use of surface conductor medium, and firm application of shovels on the thoracic surface bring more chances of success. The myocardium "environment" also interferes in success of the intervention, and its effectiveness is reduced under hypoxia, shock, hypoglycemia, hypothermia, and acidosis. Synchronism means that the power or current is being released during the ventricular activation (QRS), purposely escaping the myocardium's refractory period (descending t-wave). In that period, external electrical stimulation may cause tachycardia or even ventricular fibrillation. Special care must thus be taken when selecting the synchronized mode during device preparation. Vagal maneuvers can be performed until the treatment of choice is ready to be used, thus not postponing its beginning.[2]

Amiodarone and procainamide are indicated for cases refractory to SEC and/or to adenosine and cases that reoccur at short intervals. Even without a precise diagnosis for the kind of tachyarrhythmia, it is important to highlight that SEC will always constitute the first choice of treatment for unstable patients, and should be performed the usual way (Table 1).

Table 1 - Sequence and care in synchronized cardioversion (and in defribillation)

1. Monitor the rhythm using electrode system, connect monitor to defibrillator. Some devices detect rhythm using own paddles.
2. Apply paste or conductive substance on the paddles (use 4.5 cm paddles for children under one year of age and 8 or 13 cm paddles for children over one year of age). Do not allow the conductive medium to melt between the paddles (risk of short circuit with loss of electric current).
3. Turn on the equipment (monitor and defibrillator).
4. Turn to synchronized mode in synchronized cardioversion. Do not turn to synchronized mode in defibrillation.
5. Check LED lights (or equivalent) for blinking if QRS are being correctly detected by the equipment – important in synchronized cardioversion.
6. Select and load the energy to be applied: 0.5-1.0 J/kg for synchronized cardioversion and 2-4 J/kg for defibrillation. Dose must be doubled in the second attempt (up to 2 J/kg in synchronized cardioversion and 10 J/kg in defibrillation).
7. Place the paddles in the adequate position (right infraclavicular region; anterior axillary line, to the left from the left nipple). Do not allow contact between the paddles (risk of short circuit with loss of electric current); double check rhythm on the monitor before the next attempt.
8. Clear the area, tell all to make room and keep distance from the bed, reanimation materials and serum lines in contact with the patient and the patient him/herself (avoid accidents).
9. Firmly press the paddles against the patient's chest and shoot the load using the buttons on the device, keeping them pressed for some time (important in synchronized cardioversion).
10. Once again check heart rhythm and pulse.
11. If the arrhythmia persists, repeat the procedure doubling the dose, if necessary, after specific measurement.

Note:
. The interface medium between paddles and thoracic surface must be the conductive gel. Ultrasound gel and gauze soaked with alcohol are bad conductives; alcohol increases the risk of skin burning. Gauze soaked in saline may be used, as long as the saline solution does not cover the paddles, which causes a short circuit and reduces the efficiency of the cardioversion.
. In case pediatric paddles are not available, cardioversion is still possible for younger children using adult-size paddles positioned in an alternative way: anterior thorax (left mean sternum edge) and back (paravertebral left region).

TACHYARRHYTHMIAS WITH WIDENED QRS (DURATION > 0.09S)

A tachyarrhythmia with widened QRS is probably a ventricular tachycardia (VT), which, by definition, involves cardiac structures below the Bundle of His bifurcation. For practical purposes, all regular tachyarrhythmias with widened QRS are to be treated as if they were VT, as long as there are no other accidents on the tracing's baseline, such as the "saw tooth" waves of atrial *flutter*. Proceeding in this manner in an emergency context does not incur in any extra risk for the patient. It is important to remember that the higher limit of 0.09s for QRS in children is a practical simplification and that one will not always have a clear extrapolation of that value in the VT, especially not in infants; the most significant data to be considered is QRS widening in relation to the base sinus rhythm. VT is an uncommon disorder in the pediatric age group.[3,4]

Unlike SVT, the risk of structural cardiac modification is of concern in VT cases, especially in sustained forms of tachycardia and in symptomatic patients, who in most cases have a structural heart disease or some canalicular syndrome (such as long QT syndrome). Several mechanisms are potentially involved in its genesis, including change in automatism, intraventricular reentry, and the loading activity.[1,6,7] A variety of conditions may be related to VT (Table 2).

Diagnosis is reached essentially by finding tachyarrhythmia (HR ranging from close to normal up to above 250 bpm) with widened QRS and more commonly spacing at regular intervals. P waves are not usually visible and when present, a clear dissociation in relation to QRS can be noted. Irregularity in QRS spacing and other accidents on the tracing baseline, such as the "sawtooth" waves from atrial *flutter* eliminate VT as a diagnosis possibility. A specific morphology of VT deserves attention, one that characterizes the "torsades de pointes" – typical of long QT syndrome: bizarre QRS with variable amplitude and polarity, as if the waves were twisting themselves around the baseline. In ECG baseline a widening of the QTc interval can be noted (above 0.44-0.46s).[4,6,7]

The VT can have variable duration; it can be called sustained when exceeding 30 seconds in duration. It may revert spontaneously and reoccur some time later. Its character is frequently paroxysmal, and the clinical status ranges within a spectrum of severity similarly to what has already been described in SVT. The form of treatment depends on the patient's clinical condition .[2]

Table 2 - Conditions associated to VT

Acute Conditions
- hypoxia, acidosis, hypercalemia, hypermagnesemia, hypocalcaemia, hypoglycemia; hypothermia
- medication: sympathomimetic amines, H1 antagonists (phenothiazines, terfenadine), tricyclic antidepressants, amphetamines, cocaine, heroin, inhalatory anesthetics, antiarrhythmics (quinidine, procainamide, amiodarone, etc.), macrolide-cisapride interaction. Several drugs induce VT by widening the QT interval (acquired long QT syndrome): H1 antagonists, tricyclic antidepressants, inhalatory anesthetics, antiarrhythmics and macrolide-cisapride interaction.
- scorpionism;
- infectious myocarditis and endocarditis → structural alteration;
Chronic Conditions
- normal heart;
- associated with congenital cardiopathies (Falot tetralogy, Ebstein's abnormality, mitral prolapse valve, aortic valve disease, Einsenmenger syndrome) → structural alteration;
- heart surgery postoperative (Falot tetralogy, CIV, truncus, aortic change, etc.) → structural alteration;
- arrhythmogenic dysplasia of VD → structural alteration;
- dilated, hypertrophic myocardiopathy → structural alteration;
- ventricular tumors → structural alteration;
- congenital long QT syndrome, short QT syndrome, Brugada syndrome, catecholaminergic polymorphous tachycardia.

APPROACH FOR STABLE SYMPTOMATIC PATIENTS

Considering that some varieties of VT operate as markers for severe structural heart diseases or may deteriorate into dangerously high frequencies or, even, into ventricular fibrillation, special care must be taken when approaching these patients, even if they seem hemodynamically stable. The same general measures described in SVT are to be adopted here: monitoring, conducting an ECG, and contacting the cardiologist. Vagal maneuvers are of no therapeutic use in this situation. In cases associated with reversible conditions, identification and specific treatment must be performed for the cause. Cardioversion should be attempted by administering drugs, among which, in addition to amiodarone and procainamide (class IIb), we include lidocaine as a therapeutic option, although it is less effective. Its major indication would be for VT associated to focal myocardial ischemia.[6,7]

In the specific case of "torsades de pointes"-type VT, treatment must be done using magnesium sulfate in bolus – 25 mg/kg (maximum 2 g), slowly, in 10 to 20 minutes.

Adenosine, at first, does not have any therapeutic utility in tachycardia with widened QRS. In the cases of tachycardia with monomorphic, widened QRS and regular RR interval that is refractory to conventional therapy, adenosine can be attempted for both therapeutic and diagnostic purposes; if conversion of arrhythmia occurs, it will characterizes supraventricular tachycardia with widened QRS.

After drug-based cardioversion, some drug must be maintained (amiodarone, procainamide or lidocaine) in continued infusion for prophylaxis and to avoid recurrence. Duration of infusion must be discussed with the cardiologist. Cases that do not respond to drugs may require SEC. As already seen in SVT, it is better that a cardiologist prescribes the procedure, which should take place under sedation, analgesia, and assisted ventilation.[7]

UNSTABLE PATIENT

Treatment for unstable patients must be performed by SEC. Whenever possible a speed bolus should be performed prior to the SEC (as long as it does not delay it), using either amiodarone, procainamide or lidocaine, so as to reduce the chances of recurrence of arrhythmia after cardioversion. SEC should not be delayed for venipuncture attempts, sedation, intubation or preparation of bolus for medications. In case the patient responds to cardioversion, continued infusion of any of the drugs mentioned must be maintained. Duration of infusion will depend on each case (the higher the severity and the chance of recurrence of arrhythmia, the longer it will be). In the case of "torsades de pointes"-type VT, treatment will be done as described above[2,4,6] (Figure 2).

FINAL REMARKS

In emergency care it is essential to be able to distinguish situations that require immediate intervention from situations that can wait for a specialist to be present. In addition to the arrhythmias herein addressed, many other rhythm disorders may manifest in children and not characterize urgency or emergency. In those cases patient monitoring, specialist referral or contact with cardiologist are usually the only initiative expected from the general pediatrician or general practitioner.

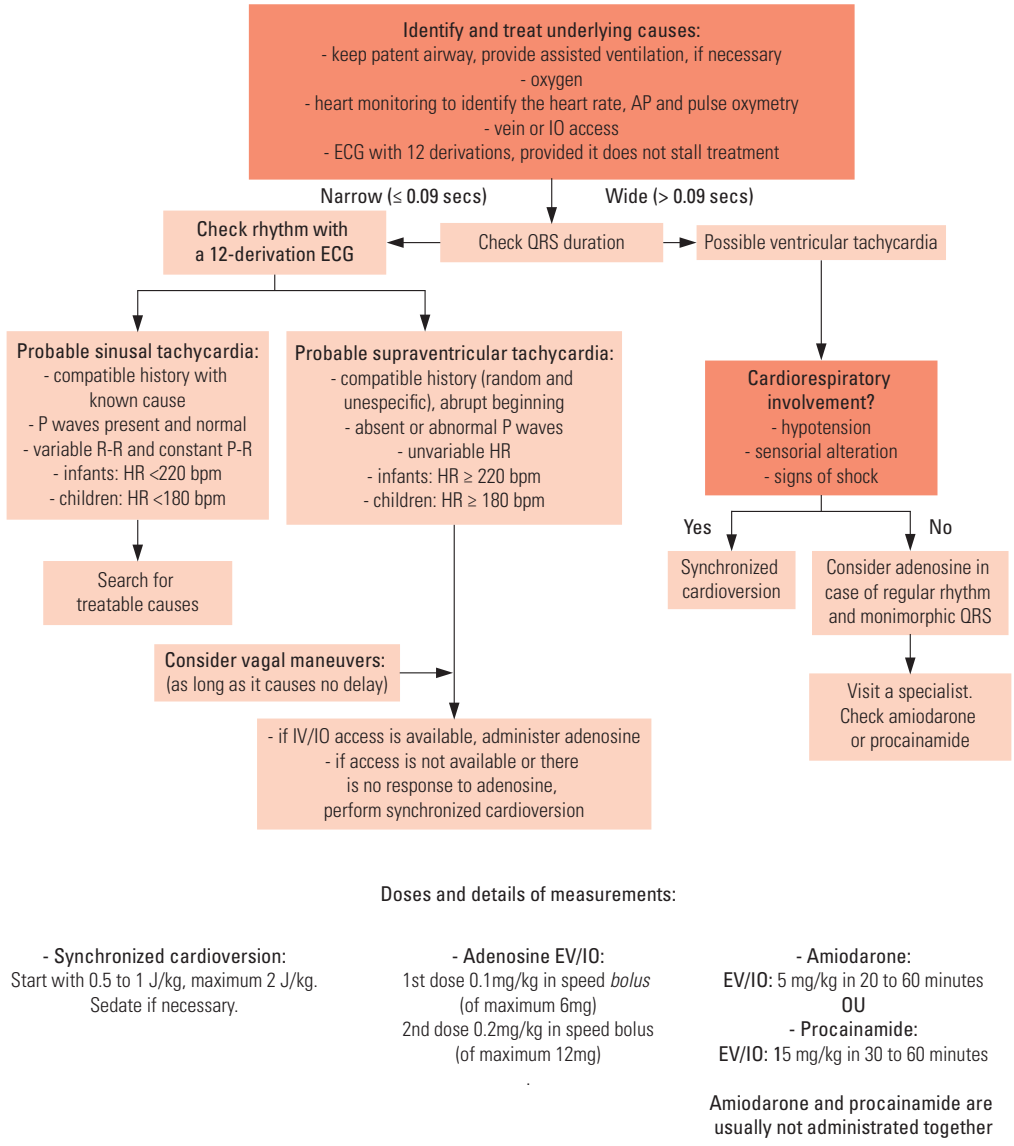

Figure 2 - Algorithm flowchart of pediatric care to tachycardia with pulse and with hemodynamic involvement.

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