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Treatment of supraventricular tachycardias in the new millennium –
drugs or radiofrequency catheter ablation?

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Abstract Supraventricular tachycardias are a cause of significant morbidity in children. Newer anti-arrhythmic drugs and the invention of radiofrequency catheter ablation have revolutionised anti-arrhythmic therapy in children.

Conclusion With the availability of these very effective therapeutic options, an improved knowledge of these methods and their indications is required.

Key words Anti-arrhythmia agents · Catheter ablation · Supraventricular tachycardia

Abbreviations AET atrial ectopic tachycardia · AV atrioventricular · JET junctional ectopic tachycardia · RFA radiofrequency catheter ablation · SVT supraventricular tachycardia · WPW Wolff-Parkinson-White

Introduction

Supraventricular tachycardias (SVTs) are a recognised cause of significant morbidity and even death in children. This has dramatically changed over the last two decades since radiofrequency catheter ablation (RFA) and newer anti-arrhythmic drugs have emerged in the management of paediatric arrhythmias. During the same time a growing understanding of arrhythmia mechanisms facilitated the application of such treatments. Nevertheless, a thorough knowledge of the natural history of the different types of SVTs and of efficacy and side-effects of the various treatment options is essential for a safe and successful management of paediatric arrhythmias. This review focuses on diagnostic and therapeutic aspects of SVT in children and is based on the authors experience and practice.

Scope of the problem

SVTs represent the most common form of paediatric arrhythmias with an estimated incidence of 0.1–0.4%. As shown in Fig. 1, almost 66% of initial manifestations of SVT occur in the 1st year of life [36]. A substantial number of infants have a history of fetal SVT [32]. Spontaneous resolutions of SVT are common in young children, although the arrhythmia may recur later on. By contrast, children older than 5 years are likely to continue with SVT episodes. Most of the affected children have a structurally normal heart. However, associated structural cardiac anomalies have been reported in 22–27% of children with SVT [36, 51]. Thus, congenital cardiac defects have to be excluded.
Mechanisms of supraventricular tachycardia

Basically there are two well-known mechanisms that cause most SVT in children and which are schematically represented in Fig. 2. The vast majority of SVTs are due to a reentrant circuit whereas automatic tachycardias account only for approximately 5%–20% of SVT [42]. Table 1 shows the clinical presentation and underlying pathophysiological mechanism for different types of SVT.

Physiologically, electrical impulse propagation from atrial to ventricular tissue occurs exclusively through the compact atrioventricular (AV) node, while the fibrous AV valvar rings represent electrical conduction barriers. In the presence of an electrically conducting accessory pathway somewhere around the tricuspid or mitral valve annulus, the AV node may be bypassed. If the accessory pathway is conducting during sinus rhythm from atrium to ventricle, a pre-excited ventricular area called delta-wave can be seen on the ECG which is typical for Wolff-Parkinson-White (WPW) syndrome. If the accessory pathway only conducts in a retrograde manner, i.e. from ventricle to atrium, this may not be perceived on a standard ECG recording; in this situation the accessory pathway is called “concealed”. Importantly, accessory pathways may form a circus movement tachycardia by a reentrant circuit with the AV node and cause a narrow QRS complex “orthodromic AV reentrant tachycardia” (antegrade conduction through the AV node, retrograde via accessory pathway) or “antidromic” broad QRS complex AV reentrant tachycardia (antegrade conduction via accessory pathway, retrograde via AV node). Orthodromic AV reentrant tachycardias are much more common. Rapidly conducting accessory pathways may, in the presence of atrial flutter or atrial fibrillation, precipitate ventricular fibrillation and result in syncope or sudden death [4, 34]. Although atrial flutter and fibrillation are rare in the healthy paediatric population, each child with WPW syndrome requires a careful evaluation because of this potential risk [47].

AV node reentrant tachycardia is another form of reentrant SVT. In this case a so-called dual AV node conduction forms two functionally distinct and separate conduction pathways in the region of the AV node. These two pathways can create a reentrant circuit and cause AV node reentrant tachycardia. This mechanism is rare in infancy but becomes increasingly more common in adolescents and adults. Reentrant circuits may also occur in the atria and form the base for intra-atrial reentrant tachycardia or atrial flutter. Both arrhythmias are rarely seen in children with structurally normal hearts.

The second main mechanism underlying SVT is enhanced automaticity because of abnormal impulse generation from one or rarely several foci. Most of these foci are located in the atria and consist of pacemaker cells with abnormal automaticity or with abnormal response to adrenergic stimulation. Rarely automatic tachycardias originate in the area of the AV node. Depending from the location of the focus these tachycardias are either called atrial ectopic (AET) or junctional ectopic tachycardia (JET). These ectopic arrhythmias mainly occur in children, are usually incessant and notoriously difficult to treat with most anti-arrhythmic drugs [2, 42, 50].

Clinical presentation

The clinical significance of a SVT episode is related to the age of the patient, rate and duration of the tachycardia, and the presence of associated cardiovascular disease. Thus, SVT episodes may cause no symptoms or, at the other end of the spectrum, can provoke cardiovascular collapse. Already younger children complain of palpitations, chest discomfort and nausea due to the fast