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Review

Surgical ablation of atrial arrhythmias: The electrophysiologist's point of view

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ABSTRACT

The most significant long-term sequelae of repaired congenital heart disease and the most important reasons for hospitalization in this population of patients are arrhythmias.
The high prevalence of postoperative atrial tachycardias among patients who have undergone specific surgical procedures involving extensive atrial dissection and repair indicates a particular dependence on certain types of surgical manipulation of the atrium. Drug therapy is likely a disappointing approach in these patients. Most antiarrhythmic agents carry the risk of proarrhythmia, and many agents aggravate sinus node dysfunction and compromise ventricular function, diminishing their utility in these patients, particularly in the absence of pacemaker therapy.
Application of innovative ablative approaches to modify the arrhythmia substrate at present appear to be considerably more promising. Catheter ablation requires specific experience and skill; results are generally worse in adult congenital heart disease as recurrence of tachycardia continues in a substantial number of patients, particularly in patients with Fontan procedures.
Surgical intervention for arrhythmias at the time of reoperation for congenital heart disease has been shown to be highly effective therapy for atrial re-entry tachycardia and atrial fibrillation already present or to prevent the occurrence of future arrhythmias in lesions known to have a significant risk of developing supraventricular tachycardia. Thus, arrhythmia surgery can be thought of as both therapeutic and prophylactic in nature.
Arrhythmia surgery is reserved for patients failing both medical and catheter ablation techniques and the procedures can be easily and successfully incorporated into the repair of congenital heart disease, after a careful preoperative evaluation of the arrhythmic circuit by mapping, as a tailored approach related to electrophysiology mapping is needed.
In conclusion, every reoperation in congenital heart disease is an opportunity to assess the arrhythmic condition, to intervene to improve the hemodynamic condition and to modify the electrical substrate.

1. Introduction

The number of patients surviving to adulthood following repair of congenital heart disease now exceeds the number of children with congenital heart disease in the United States, Canada and Europe. Over 85% of children with congenital heart defects can now be expected to reach adulthood.

The most significant long-term sequelae of repaired complex heart disease and the most important reasons for hospitalization in this population of patients are arrhythmias [1–6].

Postoperative atrial and ventricular tachycardias occurring in patients who have undergone palliation or repair of congenital heart disease remain one of the most challenging problems facing the field of modern electrophysiology.

In designing a rational, safe, and effective approach to those patients suffering from atrial tachycardias, a number of important questions remain to be addressed, some of which are quite fundamental to our understanding of the arrhythmia and its relation to the overall evolution of congenital heart disease in the affected patient. Suggestive and worrisome associations have been demonstrated between the occurrence of atrial tachycardia and of thromboembolism and sudden cardiac death [6–7]. However, it is not clear whether atrial tachycardias promote such events, or are merely a concomitant problem occurring in patients with sick, prematurely aging hearts.

Furthermore is not clear the role of atrial tachycardia in the gradual deterioration of older survivors of congenital heart disease [8].

2. Atrial arrhythmias in adults with congenital heart disease

The high prevalence of postoperative atrial tachycardias among patients who have undergone specific surgical procedures involving

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extensive atrial dissection and repair indicates a particular dependence on certain types of surgical manipulation of the atrium [1,4,6,9–10].

The etiology of arrhythmias in adult patients with congenital heart disease is multifactorial, including electrical disturbances that are inherent components of certain malformations or that develop as a consequence of previous operations or are the result of haemodynamic abnormalities during follow-up. However, it has been shown that factors other than gross surgical scarring, such as diffuse fibrosis, cellular hypertrophy, and/or changes in the characteristics of cellular action potentials, may also contribute significantly to the clinical arrhythmia substrate [10].

Adult patients with congenital heart disease may have particular problems, such as abnormal anatomy, increased atrial wall thickness, sequelae of previous surgical procedures, and arrhythmogenic foci that may be both congenital and secondary to the structural defects and prior operations [11].

Direct epicardial mapping techniques have been used to study the activation sequences of these tachycardias, and have tended to confirm the role played by lines of conduction block caused by surgical incision and/or extended suturing.

The arrhythmic problems in these patients are strongly dependent on the type of congenital heart disease and the anatomic variants and must, therefore, be evaluated particularly carefully in order to provide the correct management.

3. Therapeutical approach to atrial arrhythmias

In some patients the need to treat atrial tachycardias aggressively is very clearly mandated by their inability to tolerate their clinical symptoms, the risk of thrombosis and/or thromboembolism and the risk of worsening congestive heart failure.

In other situations, the clinical need to treat tachycardias aggressively may not be as clear, and in some patients symptoms of tachycardia may be so subtle and/or intermittent that the amount of the day spent in tachycardia is not easily quantified.

Arrhythmias that might be considered benign in patients with a normal heart, may lead to catastrophic haemodynamic deterioration and be life-threatening in patients with surgical sequelae.

The underlying congenital lesions and the surgical procedures, which create the substrate for the development of arrhythmias in adult patients, are often associated with a marginal hemodynamic status, which not only exacerbates the effects of the tachycardia, but also may limit the options for arrhythmia management.

Drug therapy is likely a disappointing approach in these patients. In the absence of specific evidence-based recommendations, pharmacological therapy is often guided by principles established in other forms of heart disease. These include considerations regarding systemic ventricular dysfunction, sinus node disease, impaired AV node conduction, negative inotropic effects, and proarrhythmia. The comparative efficacy of antiarrhythmic agents remains poorly studied, with little data regarding dosing and toxicity for the various age groups adults with congenital heart disease.

In addition, most antiarrhythmic agents carry the risk of proarrhythmia, and many agents aggravate sinus node dysfunction and compromise ventricular function, diminishing their utility in these patients, particularly in the absence of pacemaker therapy.

Amiodarone-associated thyroid dysfunction is common in adults with congenital heart disease, especially in women and those with complex cyanotic heart disease or univentricular hearts with Fontan palliation [12]. There is much interest in the new generation of antiarrhythmic agents that can have fewer multisystemic side-effects without increased mortality in the setting of left ventricular dysfunction.

Application of innovative ablative approaches to modify the arrhythmia substrate at present appear to be considerably more promising. Anatomical complexities and vascular access issues may complicate catheter-based interventions and implantation of pacemakers or implantable cardioverter-defibrillators [13–15]. Although the success rate is modest, some patients may benefit from pacemakers with automated overdrive pacing algorithms to terminate atrial tachyarrhythmias [16], but effective use of this modality requires accurate sensing of atrial activity, a sometimes challenging task, due to the abnormal anatomy and electrophysiology [17], and the multiple prosthetic patches placed at the time of surgery. In addition, anti-tachycardia pacing does not prevent and may even enhance the possibility of sudden arrhythmic death. Challenges in this device therapy include circumventing obstructed vessels, conduits, or baffles; minimizing thromboembolic risk in the presence of intracardiac shunts.

With the advent of three-dimensional electroanatomic mapping and advances in catheter technology permitting larger and deeper lesions, transcatheter ablation has emerged as a promising alternative for many patients with tachyarrhythmias [18–21]. While acute success rates in dedicated centers are high, recurrences and the onset of new arrhythmias remain problematic, particularly in patients with Fontan procedures.

4. Limits of catheter ablation therapy

The recent evolution of several commercially available techniques for high-density mapping of postoperative atrial tachycardia has led to a considerable improvement in our ability to define specific atrial activation sequences unambiguously and target radiofrequency ablation lesions to specific sites.

However, catheter ablation requires specific experience and skill; results are generally worse in adult congenital heart disease but nevertheless preferred treatment where feasible [1].

However, despite increased acute success rates and measurable clinical benefit associated with targeted ablation, recurrence of tachycardia continues in a substantial number of patients [18].

There are a great number of limits of radiofrequency catheter ablation of atrial arrhythmias in adult congenital heart disease (Table 1). First of all, there are limits due to the access to cardiac chambers. Moreover, it is mandatory to have more precise and unambiguous mapping of the underlying atrial anatomy and electrophysiological substrate due to circuit complexity and relationship with congenital heart disease and normal conduction system [22].

It is clear from multiple studies that, although many atrial reentrant circuits can be acutely interrupted with a single radiofrequency application, it is common that the isthmuses that need to be transected are of substantial width, and would require a confluent linear lesion to provide assurance of complete conduction block.

Table 1
Atrial arrhythmias in adult congenital heart disease: limits of radiofrequency ablation therapy.