The relationship between sinus node dysfunction, bradycardia-mediated atrial remodelling, and post-operative atrial flutter in patients with congenital heart defects

Natasja M.S. de Groot* and Martin J. Schalij

Department of Cardiology, Leiden University Medical Center, P.O. Box 9600, 2300 RC Leiden, The Netherlands

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This editorial refers to ‘Chronotropic incompetence in young patients with late postoperative atrial flutter: a case–control study’† by N. Anand et al., on page 2069

Atrial flutter (AFL) frequently develops in patients with congenital heart defects (CHD) after palliative or corrective cardiac surgery.1 Post-operative AFL is an important clinical problem as the presence of AFL is associated with both increased morbidity and mortality.2 Strategies to prevent the occurrence of post-operative AFL are therefore necessary. However, in order to prevent post-operative AFL, knowledge of the underlying mechanism resulting in the development of these tachyarrhythmias is mandatory.

In line with the observation that chronotropic incompetence predisposes to recurrent episodes of atrial fibrillation (AF), Anand et al. tested the hypothesis that sinoatrial node dysfunction and associated chronic bradycardia may also predispose to AFL after cardiac surgery for CHD. If a relationship could be established between chronotropic incompetence and post-operative AFL, pacing manoeuvres aimed at restoring chronotropic competence may serve a valuable prevention strategy.

Sinus node function after cardiac surgery

Sinus node dysfunction in CHD patients is caused by direct damage to the sinus node or the sinus node blood supply during cardiac surgery. Furthermore, sinus node dysfunction may be the result of the underlying congenital disorder. Damage to the sinus node and its artery can be the result of cannulation of the superior caval vein, the atriotomy, or the placement of the suture line as for example for the interatrial baffle around the superior caval vein in case of a Mustard procedure. Post-mortem examinations of the sinus node (artery) obtained from patients who were palliated with a Mustard procedure demonstrated inflammation and intimal sclerosis within the sinus node artery and necrosis of the sinus node and paranodal tissue.3 Over the years, surgical procedures have been refined by modifying cannulation techniques and atriotomy incisions to prevent sinus node damage.

Sinus node function after cardiac surgery for CHD was first evaluated in 1987 by Vetter et al.4 They performed an electrophysiological study in 64 patients, 5.8 years after the Mustard procedure. A prolonged corrected sinus node recovery time was present in 32 out of 33 patients who were in sinus rhythm at the time of the electrophysiological study. Later, they demonstrated that the degree of sinus node dysfunction was related with the incidence of AFL.5 Kurer et al.6 also reported sinus node dysfunction in patients after a modified Fontan procedure for functional monoventricles. During electrophysiological studies, prolonged sinoatrial conduction times were found in 50% of the patients with normal sinus rhythm. Furthermore, these electrophysiological studies not only revealed sinus node dysfunction in most patients, but also prolonged atrial refractoriness and intra-atrial conduction delays most likely the consequence of extensive scar tissue formation after surgery.7

Sinus node dysfunction and atrial remodelling

The relationship between sinus node dysfunction and conduction abnormalities and prolonged refractoriness was analysed by Sanders et al.8 Electrophysiological properties of the atria in patients with sinus node dysfunction were compared with age-matched controls.

They demonstrated that sinus node dysfunction in patients without atrial arrhythmias was associated with enlarged atria, the presence of low voltage areas, slowing of atrial conduction, and increased atrial refractoriness. Though it is most likely that the development of late postoperative AFL involves multiple factors, the results of the abovementioned studies indicate that in most patients with sinus node dysfunction an arrhythmogenic substrate will be present. The causal relationship, however, is not known and warrants further study.
Anand et al. non-invasively studied the degree of sinus node dysfunction in young CHD patients (mean age 9 years) with and without AFL. Patients with AFL had frequently episodes more than 6 months after cardiac surgery. Comparing the two groups, patients with AFL in general had a lower average heart rate (during sinus rhythm), reach a lower maximum heart rates during exercise, and had a lower chronotropic index; there was no difference in the minimum heart rates. Though, as pointed out by the authors, the value of the chronotropic index needs to be prospectively evaluated during long-term follow-up, the results of this study are promising and may help to identify patients who will benefit from a pacemaker. The key finding that chronotropic incompetence, as a manifestation of sinus node dysfunction, is related with late post-operative AFL, suggests that, in line with the studies discussed earlier, bradycardia-mediated remodelling enhances the vulnerability for AFL.

In this study population, there was a wide variety in the complexity of underlying CHDs and hence surgical repairs. It can therefore be expected that the surgical procedure performed determines the degree of sinus node dysfunction. To increase the insight into the mechanism of development of post-operative AFL, future studies comparing sinus node function after different types of surgical repair in larger populations are necessary.

Though surface electrocardiogram criteria have been developed to distinguish typical AFL from intra-atrial re-entrant tachycardia, the authors did not attempt to discriminate between these two different atrial tachyarrhythmias. It could be hypothesized that the atria of patients with intra-atrial re-entrant tachycardias are more diseased than the atria of patients with typical AFL. Thus, it could be of significance to relate the chronotropic index to different types of atrial arrhythmias.

Future therapy: preventive pacing?

Theoretically, anti-bradycardia pacing diminishes dispersion of atrial refractoriness and prevents the occurrence of premature atrial beats thereby reducing the likelihood of atrial tachyarrhythmias to develop. Comparable to the observation that chronotropic incompetence predisposes to recurrent episodes of AF and that preventive pacing can reduce these episodes, Anand et al. suggested that rate-adaptive pacing strategies may be beneficial in patients with post-operative AFL. So far, there are no data available to support this interesting hypothesis and further studies are needed. However, as most patients with complex CHD have extensive atrial scar tissue formation, lead placement will be crucial. In other words, if the lead is implanted in a region surrounded by scar tissue pacing will result in an increased conduction delay and may even promote the initiation of atrial tachyarrhythmias.

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References