Impact of Atrial Extrasystoles on Conduction in Pediatric Patients

with Congenital Heart Disease

Nawin L. Ramdat Misier¹, BSc; Yannick J.H.J. Taverne², MD, PhD; Mathijs S. van Schie¹, MSc;

Rohit K. Kharbanda^{1, 2}, MD; Pieter C. van de Woestijne², MD; Wouter J. van Leeuwen², MD;

Beatrijs Bartelds³, MD, PhD; Ad J.J.C. Bogers², MD, PhD; Natasja M.S. de Groot¹, MD, PhD

¹Department of Cardiology, Erasmus Medical Center, Rotterdam

²Department of Cardiothoracic Surgery, Erasmus Medical Center, Rotterdam

³ Department of Pediatrics, Division of Pediatric Cardiology, Erasmus Medical Center, Sophia

Children's Hospital, Rotterdam, The Netherlands

Short title: AES in pediatric CHD

Word count: 354

Disclosures: None

Presenter author's contact details:

Nawin L. Ramdat Misier

N.ramdatmisier@erasmusmc.nl

Dr. Molewaterplein 40

3015GD Rotterdam, the Netherlands

Phone: +31-651271136

Abstract

Objectives:

Despite improvement in (timing of) intervention or surgery, atrial fibrillation still occurs more often and at a younger age in patients with congenital heart disease (CHD) than in the general population. This suggests that in this population atrial conduction is already affected in an early stage. We therefore investigated to what extent spontaneous aberrant atrial extrasystoles (AES) affect the occurrence of conduction disorders and morphology of extracellular potentials in pediatric patients, as an indicator of early, enhanced non-uniform anisotropy.

Methods:

Twenty-one pediatric patients with various CHD (median age=1.8 years [0.2-7.3]) undergoing primary cardiac surgery were included for an intra-operative epicardial mapping of both atria, including Bachmann's bundle. Conduction velocity (CV), localized areas of conduction delay and/or block (CD, CB and continuousCDCB) and unipolar electrogram (EGM) characteristics (voltages and amount of fractionated potentials) were quantified during sinus rhythm (SR) and compared with corresponding AES beats.

Results:

During 41 AES, median unipolar voltage and CV decreased compared to corresponding SR beats (5.7 [4.0-7.5] mV vs. 7.1 [5.5-9.1] mV, p<0.001); 84.5 [73.0-93.6] cm/s vs. 87.9 [76.7-101.6] cm/s, p=0.013; respectively), whereas the amount of fractionated potentials increased (19.5 [10.5-30.0] % vs. 12.7 [3.5-18.3], p<0.001). Conduction disorders, consisting of CD, CB and cCDCB, were more prominent during AES <math>(4.5 [2.3-6.3] % vs. 3.1 [0.8-4.5] %; 1.5 [0.0-4.3] % vs. 0.6 [0.0-2.8] %; 6.3 [2.6-11.2] % vs. 4.2 [1.0-7.3] %, respectively) (all p<0.006). Differences in EGM morphology

and conduction heterogeneity were not influenced by the degree of the prematurity of AES (p>0.05).

Conclusions:

AES in pediatric patients with CHD cause localized areas of conduction slowing and block, potentials with lower unipolar voltages and a higher degree of fractionation compared to SR potentials, independent of its prematurity. Our findings indicate that in pediatric patients with CHD, early, enhanced non-uniform anisotropic atrial conduction is already present and can be unmasked by AES. These insights are the first step in further understanding the time course of atrial fibrillation in CHD patients.

Image:

