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Cardiac remodeling and factors determining occurrence of atrial arrhythmia after surgical closure of atrial septal defect in adults

Leila Bazdah, Hédi Baccar, Wejdène Ouechtati, Slim Sidhom
Hôpital Charles Nicolle, cardiologie, Tunis, Tunisie

Objectives: the purpose of this study is to assess cardiac remodeling and to determine factors predicting the occurrence of atrial arrhythmia after surgical closure of atrial septal defect (ASD) in adults.

Methods: Retrospective study including 33 adult patients (>20 years old, mean age: 34±11 years, 26 women) who underwent surgical closure of secundum or sinus venosus ASD. Before operation, all patients had dyspnea and 15% were in NYHA III-IV. Sinus rythm was present in 85% of patients. The ratio of pulmonary to systemic blood flow was calculated, yielding a mean of 2,8±1 and pulmonary artery hypertension (PAH) was observed in 80% of patients with a mean value of 41,3±10 mmHg. The ASD were closed by pericardial or Dacron patch in 97% of cases.

Results: operative death was observed in 2 cases. In survival patient, with a follow-up of 97+/-57 months, regression of right ventricular dilatation and PAH occurred in the first post-operative month and was maintained at late follow-up. Atrial arrhythmia occurred in 4 patients and were determined by older operative age (p=0.003) and the absence of cardiac remodeling after surgery.

Conclusion: surgical correction of ASD in adults is safe and efficacious. Cardiac remodeling after ASD closure in the adult is a common and an early event and prevents late morbidity which is in most cases due to arrhythmias. The mode of closure does not seem to significantly impact remodeling.

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Perioperative assessment of patients with repaired tetralogy of Fallot undergoing pulmonary valve replacement?

Jean-Bernard Selly (1), Xavier Iriart (2), Philippe Mauriat (1), François Roubertie (1), Jean-Benoit Thambo (2)
(1) CHU de Bordeaux, Service des cardiopathies congénitales, Pessac, France – (2) CHU de Bordeaux hôpital cardiologique, service des cardiopathies congénitales, Pessac, France

Purpose: Pulmonary valve replacement (PVR) is commonly performed in aduts with repaired TOF to avoid late complications related to severe pulmonary regurgitation. However, few data are available concerning perioperative complications. The aim of this study was to evaluate the perioperative complications and to determine predictive factors of the Low Cardiac Output Syndrome (LCOS) in patients undergoing PVR.

Methods and Results: 30 patients with TOF who underwent PVR between 2008 and 2009 were retrospectivelly enrolled. Mean age at valve surgery was 29.5 (range: 6.5-56.5).PVR was conducted with beating heart using a normothermic CPB (77+/-25 min) in 16 pts. 14 pts underwent additional surgery requiring aortic cross clamp (CPB mean time was 113+/-21 min). Survival rate was 97% at 90 days. Post operative complications were uncommon (VT in 6%, Mechanical Ventilation> 24 hours in 6%, renal dysfunction in 10%) except for the LCOS (46%). Prolonged CPB duration> 80 min (p<0.01) and aortic cross-clamp (p=0.03) increased LCOS (OR33 and 6). Age, RV or LV volumes and function, and preoperative additionnal lesion were not significatively predictive of perioperative complications.

Conclusion: these data underline the major role of myocardial protection during PVR in TOF patients. Short beating heart normothermic CPB leads to a decrease LCOS.

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Factors determining the nature of progression of discrete fixed subaortic stenosis

Leila Bazdah, Hédi Baccar, Wejdène Ouechtati, Slim Sidhom, Sami Marouène, Imène Fradi, Hbib Ben Ahmed, Aymen Ben Yaala
Hôpital Charles Nicolle, cardiologie, Tunis, Tunisie

Introduction: In discrete fixed subaortic stenosis, surgery is indicated when the systolic gradient (Gmax) between the left ventricle (LV) and the aorta exceed 50 mmHg or in the presence of significant aortic regurgitation (AR).

The aim of this study is to determine the factors that influence the progression of the obstruction and the appearance of AR.

Methods: retrospective serial echocardiographic review of 19 patients, mean age 16 years (2 years-38 years), with fixed discrete subaortic stenosis that don't require surgery (initial Gmax at inclusion < 50 mmHg and without any symptom). The mean follow up was 5,42 years. The progression of gradient is defined by the formula (Gmax at follow up – initial Gmax)

Results: the mean velocity of increasing of Gmax was 2 mmHg/year. This progression was correlated to the patient's age (cut off=15 years, r=–0,5; p=0,02), and the initial value of the Gmax (cut off = 40 mmHg, r=0,43; p=0,04).

The appearance or the aggravation of AR was determined by : the initial grade of AR (r=0,64 ; p=0,003), initial Gmax (r=0,65 ; p=0,002), progression's velocity of G max (r=0,47 ; p=0,04), and distance between the membrane and the aortic cusps (cut off=5 mm, r=0,49 ; p=0,03). LV hypertrophy was influenced by the velocity of progression of obstruction (> 2 mmHg/year)

Conclusion: the identification of factors determining the evolution of discrete subaortic stenosis (age < 15 years, initial Gmax >40 mmHg, distance membrane- cusps >5 mm) allows an adequate screening of patients that will require early operation.

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Familial atrioventricular node reentrant tachycardias

Béatrice Brembilla-Perrot (1), Anne Moulin-Zinsch (2), Jean Marc Sellal (1), Jérôme Schwartz (1), Pierre Yves Zinzius (1), Mahesh Pauriah (3), Gabriel Cismaru (3), Daniel Beurrier (1), Christian De Chillou (1), Pierre Louis (1), Olivier Selton (1), Arnaud Terrier De La Chaise (1), François Marçon (2)
(1) CHU of Brabois, cardiologie, Vandoeuvre Les Nancy, France – (2) Cardiologie pédiatrique, CHU de Brabois, Vandoeuvre Les Nancy, France – (3) Cardiologie, CHU de Brabois, Vandoeuvre Les Nancy, France

Familial Wolff-Parkinson-White (WPW) syndrome and gene (PRKAG2) was reported**.** Rare case reports suggest that genetic factors may play a role in the formation of accessory atrioventricular pathways (AP) and dual AV nodal pathways. In this last case, nothing is known, probably because atrioventricular node re-entrant tachycardia (AVNRT) is associated with a good prognosis. The purpose of study was to look for familial forms of AVNRT.

Methods: 792 patients with a preexcitation syndrome, 251 patients with a normal ECG but with atrioventricular re-entrant tachycardia (AVRT) using a concealed AP and 969 patients with AVNRT were referred in our department. Electrophysiological study (EPS) was performed in all subjects. The familial forms in these groups have been sought. Only patients with documented and/or induced supraventricular tachycardia were retained as having tachycardia.

Results: Among patients with a preexcitation syndrome, we did not find familial form of preexcitation syndrome. Sister of a young patient who had presented a WPW-related ventricular fibrillation complained of tachycardia, but AVNRT was identified at electrophysiological study (EPS). Two other patients had a family history of unexplained sudden death, but no ECG was recorded before death in these patients. Among patients with AVRT and normal ECG, one familial form was noted (father and daughter) (0.4%). Among patients with AVNRT, 7 families were identified (0.7%): mother and son (n=4), mother and daughter (n=1), brother and sister (n=1), father and daughter (n=1). The frequency of familial AVNRT was significantly higher than in patients with a preexcitation syndrome (p <0.025). The frequency of familial AVNRT was probably underestimated because frequently patients reported a familial history of tachycardia but the nature of the tachycardia could not be proven.

Conclusions: A genetic origin of some AVNRT exists in some families. Its frequency is approximately 0.7% in our population. Although patients with AVNRT are not at risk of life-threatening arrhythmia, the history of tachycardia in family may lead to look for the same tachycardia. The frequency is higher than in preexcitation syndrome.