regression, three stable, three moderate), 16 PG (four regression, four stable, four moderate), 17 LAWVR (six regression, seven stable, four moderate). Image quality of micro-TEE was scored 1 for 2D and 2 for Doppler. In three patients on circulatory assistance, micro-TEE could be performed in PICU with good tolerance and acceptable quality.

Conclusions.— TEE is a tool to evaluate hemodynamic significance of post-operative residual lesions. This new microprobe offers the possibility to perform TEE in small infants in the OR and in unstable patients with poor imaging TTE in PICU.

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03 Coronary artery compression during transcatheter right-ventricular outflow tract treatment: Incidence, diagnosis and outcome

Alain Fraisse a,b,c, Anass Assaïdib,c,e, Sophie Malekzadeh-Milamib,c,e, Mehul Patelb,c,e, Damien Bonnetb,d,e, Younes Boudjemlineb,c,d,e

a Cardiologie Pédiatrique, Hôpital de la Timone-Enfants, 264, rue St-Pierre, 13385 Marseille Cedex 05, France
b Centre de Référence Malformations Cardiaques Congénitales Complexes (M3C), Necker Hospital for Sick Children, Assistance Publique des Hôpitaux de Paris, Pediatric Cardiology, Paris, France
c Centre de Référence Malformations Cardiaques Congénitales Complexes (M3C), George-Pompidou European Hospital, Assistance Publique des Hôpitaux de Paris, Unit for Adults with congenital heart defects, Paris, France
d Université Paris Descartes, Sorbonne Paris Cité, Paris, France
e Hospital Bordeaux, Unit for children and Adults with congenital heart defects, Bordeaux, France

Background.— Coronary compression (CC) may occur during percutaneous pulmonary valve implantation (PPVI) and is potentially life threatening when undiagnosed before right ventricular outflow tract (RVOT) stenting. We sought to evaluate its incidence, diagnosis and outcome.

Methods.— All consecutive patients who underwent transcatheter RVOT treatment from May 2008 to December 2011 in two institutions were studied. Baseline demographics, diagnosis and outcomes of CC were reviewed with analysis of risk factors.

Results.— CC occurred in six out of 100 patients (6%) at a median age of 24 (13 to 49) years, with RVOT conduit stenosis as the primary lesion in all cases. The initial congenital heart disease was pulmonary atresia-ventricular sepal defect (n = 3), complex transposition of the great arteries (n = 2) and critical aortic stenosis status-post Ross operation (n = 1). The RVOT initial median conduit diameter at surgical implantation was 23 (17 to 24) mm and conduit types were homograft (n = 3), bioprosthesis (n = 2) and a pericardial patch (n = 1). CC was diagnosed by coronary angiogram during balloon dilation of the RVOT in all cases whereas it was suspected on pre-procedure computed tomography (CT-scan) in only two cases. Compression occurred on the left anterior descending coronary artery in four cases and on a right coronary artery that arose from the proximal left anterior descending coronary artery in two patients (single coronary artery). No risk factor was found but there was a significantly higher incidence of CC in one of the two institutions (P = 0.04). CC was well-tolerated and resolved after the balloon was deflated in all the cases. No patients underwent RVOT stenting or PPVI. Surgical conduit replacement was electively performed in three patients. Two patients with moderate residual RVOT stenosis are followed. One patient with encephalopathy and respiratory insufficiency died 9 months after catheterization.

Conclusions.— CC is efficiently diagnosed by coronary angiogram during balloon dilation in patients undergoing transcatheter interventions on RVOT. Diagnosis by pre-procedure CT-scan is not accurate. No specific risk factors exist. Surgical conduit replacement is indicated when balloon dilation fails to improve the RVOT obstruction.

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04 The influence of closure of patent ductus arteriosus on left and right ventricular systolic size and function

Laurianne Le Gloan a, Jean-Boïnt Thambob, Julie Chabaneixb, Jean-Marc Langlard a, Véronique Gourmay, Cécile Pascal a, Bénédicte Romerofe, Quentin Hauet a, Nadir Benbrik b, Olivier Baron b, Patrice Guérina

a Université Paris Descartes, Sorbonne Paris Cité, Paris, France
b Service des cardiopathies congénitales de l’enfant et de l’adulte, University hospital of Bordeaux, Bordeaux-Pessac, France

Background.— Patent ductus arteriosus is responsible for left ventricular diastolic and right ventricular systolic overload. Little is known about hemodynamic changes after percutaneous and surgical closure.

Methods.— We conducted a bicentric prospective study of children, with patent ductus arteriosus, evaluating the evolution of systolic and diastolic ventricular function before, 1 day and 1 month after successful closure, by means of transthoracic echocardiography.

Results.— Thirty-three children, age 3 months to 14.5 years, underwent successful closure of their patent ductus arteriosus, 32 percutaneously and one surgically. All patent ductus arteriosus were closed for hemodynamic reasons, with a mean diameter of 3.6 ± 0.9 mm. There was a significant increase in systolic (100.6 ± 15.1 versus 95.4 ± 15.3 mmHg, P < 0.05) and diastolic (53.2 ± 17.1 versus 47.8 ± 17.2 mmHg, P < 0.05) blood pressures, immediately after the suppression of the shunt. Left ventricular fractional shortening and end-diastolic volumes index were significantly lower the day after closure (respectively 34.7 ± 5.5 versus 37.8 ± 4.7% and 47 ± 16.2 versus 54.6 ± 20.1 mL/m²) and remained low compared to the preclosure state at follow-up (respectively 33.8 ± 5.4 versus 37.8 ± 4.7% and 47 ± 12.7 versus 54.6 ± 20.1 mL/m²). A non-significant decrease in left ventricular filling pressures was observed. Right ventricular systolic parameters (TAPSE) were significantly lower 1 month after closure (19.3 ± 2.9 versus 21.4 ± 4.4 mm, P < 0.05).

Conclusions.— Changes in left ventricular volume and function and in right ventricular function occur immediately after patent ductus arteriosus closure, and remain at 1 month of follow-up. Further studies are warranted to increase the number of patients and the duration of follow-up.

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05 A 10-year study of planned delivery of foetuses with prenatally diagnosed congenital heart disease in a single institution

M. Bensemlia a,b, F. Raimondi a,b, L. Fermont a,b, J. Le Bidois a,b, L. Salomon a,b, D. Bonnet a,b

a M3C-Necker Enfants maladies, AP-HP Université Paris Descartes, Sorbonne Paris Cité, Paris, France
b Maternité, Necker-Enfants maladies, AP-HP, Université Paris Descartes, Sorbonne Paris Cité, Paris, France

Objectives.— To describe the aims and rationale for planned delivery in a tertiary referral centre for foetuses with prenatal diagnosis of congenital heart disease.

Methods and results.— Two thousand one hundred and thirty consecutive foetuses with congenital heart disease diagnosed from
Long-term follow-up after heart transplantation in very young children

M. Veyrier a, C. Ducrue c, R. Henaine b, A. Bozio a, F. Sassolas a, J. Ninet b, S. Di Filippo b

 a Department of Pediatric and Congenital Cardiology, University of Lyon Medical Center, Lyon, France
 b Department of Cardiothoracic Surgery, University of Lyon Medical Center, Lyon, France

Heart transplantation in young children and infants may be controversial. The aim of this study was to review long-term follow-up of heart transplanted small children and assess prognosis and outcomes.

Material and methods.— Patients who underwent orthotopic heart transplantation (OHT) within the first 3 years of life were included in the study. Demographics, clinical data, events, outcomes and survival were assessed.

Results.— Among 96 paediatric heart transplantsations performed in a French single-centre, 25 patients who underwent OHT at ≤ 3 years of age, were included in the study (10 males, 15 females). Among them, 10 (40%) were on VAD support at the time of OHT. Age at OHT was 1.5 ± 0.9 years (median 1.2). Underlying cardiac disease was congenital in four (16%) or idiopathic cardiomyopathy in 21 (84%). Post-transplant follow-up was 7.1 ± 7 years (range 1 day to 22.7) and was > 10 years in seven cases (28%). Three patients died at first day, second year and fourth year post-transplant. Mean age of survivors at the time of the study was 9.1 ± 7.3 years (range 1.5 to 23.6). One acute rejection episode occurred at first month post-transplant and one at 11th year. One patient had post-transplant lympho-proliferative disease at 14th year post-transplant and was successfully cured. Graft coronary disease occurred in two cases (8%), who underwent second heart and kidney transplantation at 16th and 22nd year after first transplant. All other cases were free from coronary disease with normal graft function. End-stage renal failure occurred in the two re-transplanted cases. Significant severe renal dysfunction was present in three cases (no dialysis), moderate in three cases, and 17 had normal renal function. Linear growth ranged within normal in all patients, except the two cases with end-stage renal failure, despite continuous low dose steroid therapy in 80% of the survivors. All patients are in NYHA class I, except the two re-transplanted cases who were in NYHA class IV at the time of second transplant. Patient survival was 96% at 1-year, 90.7% at 3-year and 83% at 10-year post-transplant follow-up. Graft survival was respectively 96%, 90.7%, 83% and 66% at 1-, 3-, 10- and 16-year follow-up. Conclusion.— Long-term survival of very young heart transplant recipient is fairly good, with a low incidence of graft coronary disease and optimal functional status and growth.

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07 Anatomy of the ventricular septal defect in congenital heart defects: Random or systemic association?

Mériem Masta f e a, Damien Bonnet b, Lucile Houyel a

 a Hôpital Marie-Lannelongue-M3C, Le-Plessis-Robinson, France
 b Hôpital Necker-enfants malades-M3C, université Paris Descartes, Paris, France

Introduction.— A ventricular septal defect (VSD) is part of most congenital heart defects (CHD).

Aim of the study.— To determine the distribution of the anatomic types of VSD in CHD.

Material and methods.— We analyzed morphologically 1178 heart specimens with CHD from the anatomic collection of the French Reference Center for Complex CHD. Special attention was paid to the localization of the VSD: muscular, membranous, located between the two limbs of the septal band, inlet. The specimens were classified according to the anatomic and clinical classification of CHD (ACC-CHD).

Results.— A VSD was present in 67% of all hearts and was:

—constant, of a single type, in tetralogy of Fallot and variants and common arterial trunk: outlet, complete atrioventricular canal (CAVC): inlet, and double-inlet left ventricle (DILV): muscular;
—not constant with a predominant type, in 96% of double discordance (inlet 82%), 62% of heterotaxy syndromes (Htx, inlet 93%) 93% of interrupted aortic arch (outlet 80%), 87% of double outlet right ventricle (outlet 77%);
—not constant, of variable type, in 68% of aortic coarctation (CoA: outlet 44%, membranous 35%, muscular 21%), 54% of transposition of the great arteries (TGA: outlet 40%, membranous 25%, muscular 25%, inlet 10%);
—rare, in anomalies of pulmonary veins (5%), Ebstein anomaly (14%), double-inlet right ventricle (10%), coronary anomalies (25%);
—isolated in 10% of all VSD: outlet 44%, membranous 36%, muscular 18%, inlet 2%.

Associations were:

—outlet VSD: 60% "conotruncal" defects (CTD), 10% TGA;
—inlet: 57% CAVC, 13% DD, 10% Htx;
—muscular: 33% DILV, 26% TGA, 13% isolated;
—membranous: 30% TGA, 28% isolated, 16% CoA.

Conclusion.— The VSD is an integral part of the phenotype in some CHD (CTD, CAVC, and DILV). In CoA and TGA the VSD is not constant and its anatomic distribution is similar to that in isolated VSD, indicating a likely random association. This reinforces the hypothesis of different genetic mechanisms in TGA and CTD. This original approach, using the anatomic characteristics of one part of the phenotype, could provide new insights in the grouping and aetiology of CHD.

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