EI of PA was (10%). PA was more asymmetric in bicuspid valves rather than in tricuspid valves (EI 14% vs 7%, p = 0.002).

PA diameter didn't differ significantly between 2D-TTE, Dh, CT scan and angiography, and were significantly lower than mean 3DD and Dv. There was a very strong correlation between 3DD and perioperative measurement. PA was conserved in 53.3% without significant residual stenosis immediately and after a 20 month of follow-up (except 1 patient requiring balloon dilatation).

Conclusion 3D-TTE in patients with TOF is an accurate method to describe PV, and to measure PA size. Thus could help the surgical repair and the preservation of PV function.

Conflict of interest The authors have not transmitted any conflicts of interest.

P4

Right ventricular activation mapping to determine electrical activation pattern in patients with repaired tetralogy of Fallot

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Background Patients after repaired tetralogy of Fallot (TOF) frequently have right ventricular (RV) dysfunction and prolonged QRS duration (QRSd), the latter is considered as a sudden death risk factor. It has been suggested that QRSd mainly reflects abnormalities of the RV outflow tract (RVOT) rather than the RV body itself. We aimed to better understand the RV electrical activation pattern in these patients using activation mapping.

Methods 51 Adults (33±13 yo, mean QRS duration 153±21ms) referred for either catheter ablation or pulmonary valve replacement late after TOF repair underwent a MRI, with fibrosis analysis, and an invasive RV activation mapping (201±32 sites per patient; Carto 3 – Biosense Webster). RV total activation time (RVTAT) was defined as the duration between the first and the last RV EGM.

Results The delay between QRS onset and earliest RV EGM was 28±23 ms traducing the absence of RV purkinje activation and the left to right ventricle activation. We observed in all patients a single RV septal breakthrough (mid-septal in 79%, septo-basal in 14% and apico-septal in 7%) followed by 2±spreads of activation: a first wave from the septum to the RV anterior wall through the RVOT with fragmented EGM in the infundibulum; a second wave from the septum to the RV free wall through the apex with slow conduction. The RV free wall was the latest activated in all the patients (Figure 1). RVTAT (127±20 ms) was correlated to QRS duration (r=0,72; p<0,001) and to RVOT scar surface area in MRI (r=0,62; p<0,001). These activation parameters were correlated with fibrosis revealed by MRI.

Conclusion RV delayed activation in patients with repaired TOF traduces an homogeneous activation pattern that is not only the consequence of an infundibular disease but also reflects a slow conduction in the RV free wall.

Conflict of interest The authors have not transmitted any conflict of interest.

P5

Children with tetralogy of Fallot exhibit accelerated maturation of the cardiac tissue into adult phenotype

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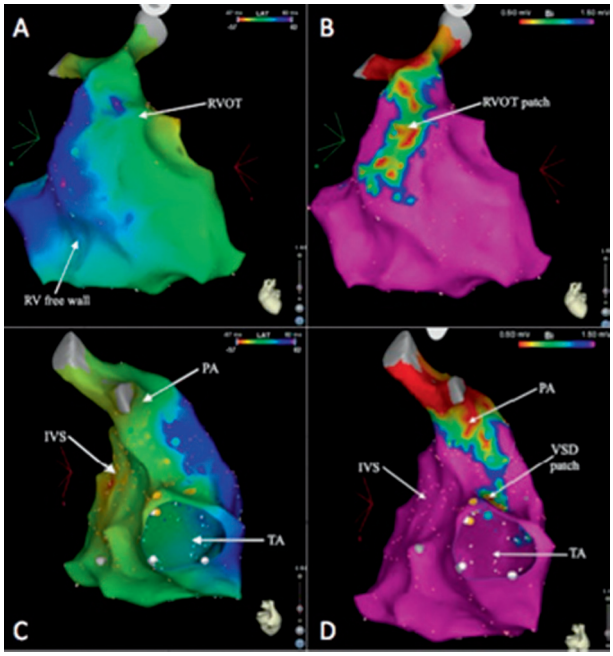

P4 – Figure 1: Anterior and posterior views of RV activation maps (A and C) and RV voltage maps (B and D) in the same patient – RVOT = right ventricular outflow tract; – VSD = vetricular septal defect, IVS = interventricular septum, TA = tricuspid annulus, PA = pulmonary annulus

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Shortly after birth, under changes in loading conditions of the heart, a shift from predominantly hyperplasic to hypertrophic cardiac growth occurs. In rodents, at birth, neonatal proliferative cardiomyocytes (CMs) exhibit a fusiform shape and differentiated into non-proliferative rhod-shape CM around P20 defined as the mature state. In human, CM differentiation ended around 6 years old but the exact timing still remains unknown. Moreover, numerous stimuli most probably contribute to the CM maturation, including pressure. In a human model of pressure overload named tetralogy of Fallot (ToF), we thus hypothesized the occurrence of an earlier maturation of the cardiac tissue.

Methods We prospectively included 15 children around 6 month-old (min. 3.5 max. 27), who required surgery for the management of ToF. We assessed criteria of maturation from right ventricle tissue of infundibulum that was resected during the surgery.

Results As previously described, heart sections analysis revealed a marked sub-endocardial fibrosis (473±444µm) and a significant fibrosis of the interstitium (13.0±6.3%). This criteria was correlated to the severity of the disease represented by the degree of desaturation (r=0,623; p=0,017). In all children analysed, CMs were hypertrophied but unlike healthy myocardium, CM size was heterogeneous (CV=40,4%), with alternating immature and mature area. In mature area, as expected, CMs proliferation stopped as indicated by the loss of Ki67 staining and exhibit a mature rod-shape. Ultrastructurally, CMs had structured intercalated disk and elongated contractile apparatus with an alignment of Z-strikes and apparent I-band. The lateral membrane between two CMs was compacted with periodic crests and holes.

Conclusion Our data highly suggest that the increase of pressure during childhood may act as a maturation factor. Myocardium in ToF is heterogeneous, with mature and immature zone associated with fibrosis, which are a potential substrate for arrhythmias.

Conflict of interest The authors have not transmitted any conflicts of interest.

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