

AP Target Symposium

AP Target Symposium 2 (I-APT2)

Dealing with the borderline Right Ventricle - Fontan vs One-and-a-Half Ventricle Repair vs Biventricular Repair: what are the criteria and how to get there –

Chair: Munetaka Masuda (Department of Cardiovascular Surgery, Yokohama City University Hospital, Japan)

Chair: Hiroyuki Yamagishi (Department of Pediatrics, Keio University School of Medicine, Japan)

Fri. Jul 7, 2017 4:15 PM - 5:45 PM ROOM 3 (Exhibition and Event Hall Room 3)

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[I-APT2-01] Circulation and morphology of the borderline right ventricle

○ Hiroyuki Yamagishi (Department of Pediatrics, Keio University School of Medicine, Japan)

As the normal heart has two separate ventricles, namely right and left ventricle, “two-ventricle repair” is an ideal surgical strategy for complex congenital heart diseases. There are, however, defects that cannot be easily septated surgically commonly undergo the Fontan operation, or “one-ventricle repair.” And occasionally, there are hearts with two ventricular cavities and two atrioventricular valves, but the morphologic and physiologic characteristics of the right-sided ventricle are insufficient and subject to the “one and a half ventricle repair.” The strategy how to deal with the borderline right ventricle is extensively discussed in the session.

Congenital heart defects involving hypoplasia of the right or left ventricle account for 25% of all mortality from congenital heart disease in children and may be the result of defects in expansion of a precursor pool of ventricular cardiomyocytes. Cell lineage analyses have demonstrated that two progenitor cell populations, the first heart field (FHF) and second heart field (SHF), are derived from the lateral plate and splanchnic mesoderm, respectively. The FHF forms the crescent shaped heart primordium that gives rise to the linear heart tube and later contribute to most of the left ventricle. The SHF cells, initially medial and caudal to the FHF, migrate through the pharyngeal mesoderm into the heart tube and contribute to the outflow tract, right ventricle and atria.

As for the ventricular development, a “ballooning” model has been proposed in which growth of the ventral aspect of the linear heart tube gives rise to the outer curvature of the looped heart and results in ventricular expansion. Our observation using model mice represents some of the more convincing functional evidence supporting this model. Dissection of the complex molecular pathways involved in ventricular specification, differentiation and growth would provide the basis for understanding the pathogenesis of hypoplastic ventricle syndromes.