had improvement of their respiratory functional tests. All patients had improvement of there right ventricular function (Fig. 1) and none of them developed any chronic lung allograft dysfunction.
Median follow-up after lung transplant was 397 months (min: 265; max: 562). One patient died of pulmonary infection complication at 14.5 months after lung transplant.
Conclusion DLT followed by percutaneous ASD closure is an efficient therapeutic approach in patients with end-stage ASD associated PAH that may offer an alternative option to HLT.
Keywords Lung transplant; Atrioseptal defect; Pulmonary arterial hypertension; Eisenmenger syndrome
Disclosure of interest The authors declare that they have no competing interest.

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Early detection of left ventricular failure in right ventricular congenital heart diseases

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Abstract
Background Early detection of left ventricular (LV) failure is crucial to improve prognosis of patients with right ventricular (RV) overload.
Objectives We aim to assess whether LV function is precociously affected in 2 surgical porcine models of moderate (Fallot repaired) and severe RV dysfunction (progressive pulmonary hypertension HP) at in vivo and in vitro levels.
Methods Three animals of each surgical group were compared with 6 controls/Sham. 4 months after surgeries, LV function was evaluated using echocardiography/strain compared with conductance catheter. At cellular level using isolated cardiomyocytes, calcium transients amplitude with relaxation time associated and sarcomere shortening were recorded using Ionoptix system. T-tubules network integrity (DI-4-ANEPPS) and colocalization (Immunofluorescence) between main Excitation/Contraction (EC) actors (Ca2 + v1.2-Ryr) were analyzed. Contractile reserve was evaluated by adrenergic stimulation in-vivo and in-vitro (Dobutamine-isoproterenol).
Results Despite RV dysfunctions in both groups, LV present hemodynamic impairment only in HP group (Longitudinal strain 9 versus 18%. Conductance catether with dobutamin: Elastance arterial 7.7 versus 1.32, SV 14 versus 75 ml and tau (relaxation) 49 versus 27, $P < 0.05$). In cardiomyocytes, we observe decrease of Ca2+ transient amplitude and cardiomyocytes contraction, acceleration of Ca2+ relaxation time, T-tubule network desorganisation and Cav1.2/Ryr decoupling (Fig. 1).
In vivo and in vitro, adrenergic stimulations increase dysfunction.
Conclusion In vitro experiments pointed early abnormalities in LV EC particularly after adrenergic stimulation. A better understanding of cellular alterations could lead to survival improvement.
Disclosure of interest The authors declare that they have no competing interest

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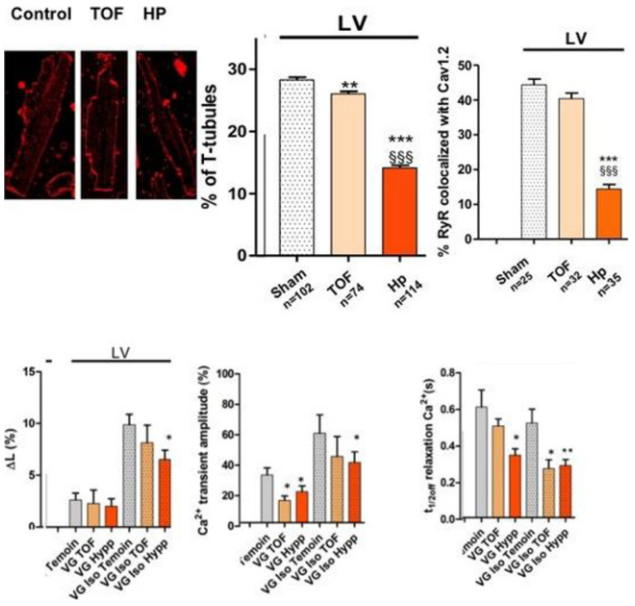

Fig. 1