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Study of the time-relationship of the mechano-electrical interaction in an animal model of tetralogy of Fallot : Implications for the risk assessment of ventricular arrhythmia

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Background : Long-term outcome of tetralogy of Fallot(TOF) is determined by progressive RV dysfunction, due to chronic pulmonary regurgitation(PR), and the risk of sudden death by ventricular arrhythmia. Although the electro-mechanical interaction is well-known, its time-relationship remains ill-defined.
Methods : According to contemporary surgical repair, PR was induced by transannular patch with limited RV scarring, in pigs of 25 kg. Biventricular mechano-electrical assessment was based on presuure-volume loops, after respectively 3 (n=8) and 6 (n=7) months, and compared to control animals (n=5). Electrophysiological testing included registration of endocardial monophasic action potentials(MAP) at 3 RV locations (inlet-apex-outflow) and 2 LV locations (septal-lateral), intraventricular conduction velocity, and induction of ventricular arrhythmia based on a burst pacing protocol (minimal cycle length 200 ms).
Results : Data are shown in the table. RV dilation and dysfunction is observed at 3 months, and progressed significantly at 6 months, achieving at that time the critical threshold for pulmonary valve implantation. Depressed RV contractility is associated with impaired LV contractility at 6 months. According to an increased QRS duration, the MAP duration and MAP dispersion at RV and LV were prolonged at 6 months. The RV conduction velocity decreased significantly at 6 months, compared to control and 3 month animals. This was associated with an increased activation time on the 3 RV locations at 6 months. The delayed intraventricular LV conduction was not significant. No sustained ventricular arrhythmias were induced.
Conclusion : Progressive RV dysfunction is associated with altered electrical properties, observed at 6 months of pulmonary regurgitation, and predominantly affecting the RV. Despite significant RV dilation and concomitant electrical disturbance, ventricular arrhythmia were not induced. The data suggest that the hemodynamical RV deterioration after TOF repair is effectively preceding the risk of inducing sustained ventricular arrhythmia, questioning the need for electrophysiological testing preoperative to pulmonary valve implantation, when extensive RV scarring is absent.

Hemodynamics	Control (n=5)	3 months (n=8	6 months (n=7)	p-value ANOVA
RVEDVi (ml/m²)	59 ± 16	122 ± 18	142 ± 13	<0,001
RVESVi (ml/m²)	27 ± 10	74 ± 15	96 ± 7	<0,001
RVEF (%)	55 ± 6*	39 ± 6	32 ± 5	<0,001
LVEDVi (ml/m²)	78 ± 10	85 ± 17	58 ± 6	0,002
LVESVi (ml/m²)	36 ± 8	43 ± 10	36 ± 10	0,331
LVEF (%)	54 ± 9	49 ± 10	39 ± 15	0,102
QRS duration (ms)	76 ± 12	88 ± 12	97 ± 13	0,039
MAP duration (ms)				
RV-inlet	260 ± 5	268 ± 12	313 ± 24	<0,001
RV-apex	253 ± 13	262 ± 26	296 ± 37	0,037
RV-outflow	252 ± 21	264 ± 29	308 ± 30	0,005
LV-septal	249 ± 24	274 ± 22	321 ± 43	0,003
LV-lateral	248 ± 12	265 ± 22	327 ± 49	0,001
Activatie time (ms)				
RV-inlet	16 ± 12	33 ± 10	42 ± 10	0,011
RV-apex	7 ± 4	18 ± 6	21 ± 5	0,005
RV-outflow	16 ± 7	36 ± 9	40 ± 13	0,005
LV-septal	5 ± 4	19 ± 10	26 ± 16	0,063
LV-lateral	6 ± 8	25 ± 12	27 ± 16	0,077
Conduction velocity(m/s)				
RV	2,4 ± 0,6	2,1 ± 0,2	1,8 ± 0,2	0,035
LV	2,4 ± 0,7	2,3 ± 0,4	1,9 ± 0,2	0,107