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Right ventricle function analysis by using RV papillary muscle

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Introduction: Right ventricular dysfunction is a common long-term complication in patients after the repair of congenital heart disease. Previous investigators have examined the cellular and molecular mechanisms of left ventricular (LV) remodeling, but little is known about the stressed RV. Our purpose was to provide a detailed physiological characterization of a model of RV hypertrophy.
Method: A right lateral thoracotomy was performed on SD rat at the weight of 200g. Then, the main pulmonary artery was banded with a 5-0 suture, tied tight against a 25G needle. RV pressure was examined by using 1.2F micro cath. at 4weeks postoperative. The rats with elevated RV pressure (>80mmHg) were enrolled for more further study. Age matched SD rats were examined for control. We used aequorin method to evaluate tension development with Ca2+ in right ventricular papillary muscle in place of RV function.
Results: RV pressure was significantly difference between PAB and control(88.2±13.9mmHg vs 21.8±5.1mmHg, P<0.001). There was no significant difference in cardiac output(0.38±0.09 vs 0.37±0.11; NS). RV weight increased by 70% without significant change in LV weight. We confirmed that peak Ca2+ in PAB was significantly higher than that in control (2.35±0.03 vs 1.52±0.11 μM, p<0.05). Interestingly, peak tension of both cardiac muscle was not significantly different at 4 weeks after operation (26.52±9.91 vs 38.03±5.97 mN/mm2). We also analyzed time courses in tension and Ca2+. Relaxation time in PAB was not significantly different from that in control (64.00±2.89 vs 70.67±2.82 msec), however, decay time of light in PAB was significantly slower than that in control (45.33±3.76, 27.33±1.15 msec, p<0.05).
Conclusions: RV hypertrophy could have impairment of Ca2+ handling possibly due to impairment of Ca2+ handling. This model, and the method are useful to understand the RV dysfunction. More further study will be needed to establish the strategy for RV failure.