Right atrial and ventricular adaptation to acute and chronic right ventricular pressure overload

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Background: In contrast to acute pulmonary hypertension (PH), chronic PH has a unique cardiovascular circulation such that it is not possible to estimate its severity solely from measuring the pulmonary arterial pressure. Chronic PH causes RV remodeling, including hypertrophy and dilatation.

Purpose: We evaluated right atrial (RA) contractility, right ventricular (RV) diastolic and systolic function in adult patients (pts) with acute pulmonary embolism (PE) and chronic pulmonary hypertension (PAH) and assessed their relationship with serum brain natriuretic peptide (BNP).

Methods: Systolic myocardial wave (Sa), early diastolic myocardial wave (Ea), and late diastolic myocardial wave (Aa) at the tricuspid annulus were recorded in 29 pts with PE and in 8 pts with PAH.

A complete 2D and pulsed echocardiographic examination was performed using Philips IE 33 (Philips Medical Imaging Andover, MA)

A novel tissue tracking technique (TAD, QLAB Software, Philips Medical System) was used to detect tricuspid annular motion off-line the apical four chamber using the hinge point of the tricuspid valve leaflets with the septal and lateral aspects of the tricuspid annulus as user-defined anatomic landmarks.

Aa of RV was regarded as the parameter of RA contractility.

Mean RA pressure (RAP) can be estimated using the tricuspid E/Ea ratio.

BNP levels were measured within 24 hours of echocardiographic examination. BNP is produced mainly in ventricular cardiomycytes and its increased in reponse to both the left ventricle and RV volue or pression overload.

Results: All data are presented in table 1.

RA contractilty was increased in PE pts wit BNP> 80pg/mL vs PAH pts. RAP was increased in PAH pts vs PE pts (7.2+/-2.8 vs 5.4 +/- 2.4 p=0.05) .

The BNP level was negatively correlated with TAD (R^2=0.4; p<0.0001), and positively with PASP (R^2=0.53; p<0.0001).

Conclusion: RV function during PE showed no significant change in RV diastolic performance. In PAH pts, RA contractility was decreased and RA pressure was increased.

In pts with acute or chronic right ventricular pressure overload, increased BNP level may be related to decreased RV and RA systolic function.

Apical 4chamber view with TAD in PAH pt
QLAB software Philips Medical System

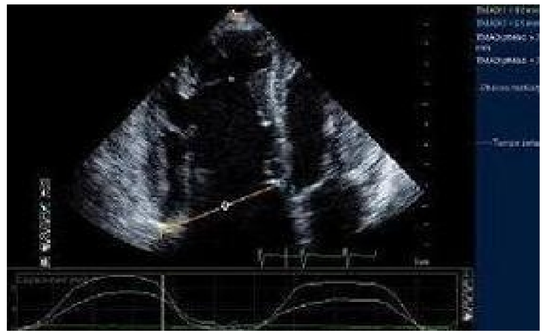

Table 1 :

	PE with BNP < 80	PE with BNP > 80	PAH pts	p*	p+
n	16	13	8		
Age (years)	58,9+/-17,1	73,9+/-7,5	62,4+/-14,9	ns	ns
Male (%)	56,3	46,2	50	ns	ns
Heart rate (bpm)	77,3+/-10,4	86,2+/-15,6	80,6+/-15,4	ns	ns
Body area (m²)	1,92+/-0,24	1,83+/-0,21	1,69+/-0,07	0,02	ns
RVEDD (mm)	32,1 +/-7	35,8+/-6,1	46,9+/-7,2	0,0003	0,002
PASP(mmHg)	35,9+/-6,9	49,8+/-10	80,6+/-15,4	0,0002	0,0009
PA (ms)	96,7+/-30,1	89,2+/-32,5	61,6+/-13,6	0,023	ns
RV FAC (%)	50,4+/-11,6	45,9+/-12,6	38,7+/-11,1	0,04	ns
Sa (cm/s)	13,5+/-3	12,2+/-3,9	10,2+/-2,8	0,02	ns
TAD (mm)	16,6+/-3,5	12,1+/-2,5	10,5+/-3,2	0,0003	ns
RV/ tricuspid:					
E (cm/s)	47,6+/-8,5	48,7+/-12,5	51,3+/-7,9	ns	ns
A (cm/s)	57,4+/-8,5	58+/-15,9	52,6+/-19,9	ns	ns
DT (ms)	164+/-62,9	149,9+/-49,2	147,5+/-36,9	ns	ns
E/A	1+/-,3	0,9+/-0,5	1,1+/-0,5	ns	ns
Ea (cm/s)	10,1+/-3	9+/-3,4	8,2+/-4	ns	ns
Aa (cm/s)	14,7+/-4,5	16,4+/-4,8	11,9+/-1,1	ns	0,046
E/Ea	5,1+/-2,4	5,6+/-2,6	7,2+/-2,8	0,04	ns
BNP (pg/mL)	39,8+/-24,7	227+/-154	443+/-23,8	<0,001	0,011

DT: deceleration time of the E wave- RVEDD: right ventricular end diastolic diameter- PA: pulmonic valve acceleration time-p*: comparaison between pts with PE and BNP level < 80 pg/mL versus PAH pts - p+: comparaison between pts with PE and BNP level > 80 pg/mL versus PAH pts - ns: not significant

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