

In summary, the results suggest that environmental temperature and relative humidity contribute to the variability of condensate pH.

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#### STATEMENT OF INTEREST

None declared.

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DOI: 10.1183/09031936.00128007

## Isolated diastolic dysfunction of right ventricle: stress-induced pulmonary hypertension

To the Editors:

We read with great interest the article of HUEZ *et al.* [1]. Their results, which were mostly noninvasive, suggest that isolated longitudinal diastolic dysfunction of the right ventricle may be a sign of stress-induced (or latent) pulmonary hypertension. The aim of our letter is to confirm this observation with the help of our results based on invasive measurements.

In total, 58 patients (mean age 54±8 yrs, 48 female) were examined. These comprised 15 healthy subjects who had no signs or symptoms of heart disease and 43 consecutive patients suffering from connective tissue disease (CTD), of whom 38 had systemic sclerosis, two had systemic lupus erythematosus, two had mixed CTD and one had polymyositis. Patients in the latter group were referred to the University of Pécs (Pécs, Hungary) on suspicion of pulmonary artery hypertension (PAH). Patients with atrial fibrillation and severe mitral or tricuspid insufficiency were excluded from the study.

The local ethics committee approved the study. All subjects had given written informed consent prior to undergoing echocardiography and right heart catheterisation.

Echocardiography was performed using an Aloka ProSound 5500 ultrasound system (Aloka Co. Ltd, Tokyo, Japan). Left ventricular ejection fraction was measured by Simpson's method. Right ventricular end-diastolic diameter was obtained from standard parasternal long axis view using M-mode measurements. Transmitral flow was recorded from the apical four-chamber view. The peak of the early (E) diastolic

velocities was measured. Systolic pulmonary artery pressure (PAP) was estimated as a sum of the pressure difference across the tricuspid valve calculated using the modified Bernoulli equation and an estimate of mean right atrial pressure. Myocardial systolic, early (E') and late diastolic velocities were measured from apical four-chamber view at the lateral border of the mitral and tricuspid annulus using tissue Doppler imaging. The mitral E/E' ratio was calculated. Doppler measurements were obtained from at least three consecutive beats during end-expiratory apnoea.

All patients with CTD underwent right heart catheterisation. A Swan-Ganz catheter was introduced to a main pulmonary artery branch. If the resting mean PAP ( $P_{pa}$ ) was <30 mmHg, a 3 min bench-fly physical stress test was performed using two 1-kg dumbbells.  $P_{pa}$  was measured at rest and at peak exercise.

Differences between groups were tested for significance using ANOVA. *Post hoc* tests were performed by Scheffe's method. Comparisons of nonparametric data were performed using Chi-squared tests. A p-value of <0.05 was considered significant.

Thirteen patients belonging to the CTD group had resting ( $P_{pa}$ : 37.9±10.9 mmHg) PAH diagnosed by right heart catheterisation. In six patients, normal PAP values were measured at rest (22.8±1.7 mmHg) while elevated values were measured at peak exertion (39.8±6.3 mmHg).

Considering these results, the CTD patients were divided into the following three groups: 24 patients without PAH, six patients with stress-induced PAH and 13 patients with resting

**TABLE 1** Main characteristics of the study population

	Normal subjects	Patients with CTD			p-value
		Without PAH	Stress-induced PAH	Resting PAH	
Patients n	15	24	6	13	
Age yrs	50±6	54±7	57±9	55±8	NS
BSA m <sup>2</sup>	1.74±0.15	1.76±0.16	1.76±0.15	1.71±0.26	NS
Male/female n	3/12	3/21	0/6	4/9	NS
Left ventricular EF %	63.5±2.3**	61.2±3.3	63.3±2.7	59.0±3.7	<0.01
Mitral E/E'	5.9±1.3	7.1±2.4	8.8±1.9	8.5±4.4	NS
RV diameter mm	27.9±2.5***	31.8±4.1**	29.7±3.7*	38.4±8.2	<0.001
PASP estimated mmHg	24.6±2.4***	30.0±7.0***	31.5±4.2**	52.7±18.7	<0.001
Tricuspid S cm·s <sup>-1</sup>	13.9±2.6**	13.1±2.7	12.1±2.1	10.6±2.4	<0.01
Tricuspid E' cm·s <sup>-1</sup>	11.0±1.7	9.7±2.3	8.3±2.2 <sup>#</sup>	8.4±1.1 <sup>#</sup>	<0.01
Tricuspid A' cm·s <sup>-1</sup>	13.4±3.0	13.7±2.8	12.9±2.4	12.0±3.6	NS

Data are presented as mean±SD, unless otherwise stated. CTD: connective tissue disease; PAH: pulmonary artery hypertension; BSA: body surface area; EF: ejection fraction; E: early diastolic velocity; E': myocardial early diastolic velocity; RV: right ventricle; PASP estimated: systolic pulmonary artery pressure estimated by echocardiography; S: myocardial systolic velocity; A: myocardial late diastolic velocity. \*: p<0.05 versus resting PAH; #: p<0.05 versus normal; \*\*: p<0.01 versus resting PAH; \*\*\*: p<0.001 versus resting PAH.

PAH. Table 1 outlines the principal characteristics of our patient groups. The four groups were matched in age, sex distribution and body surface area. The left ventricular ejection fraction was significantly lower in patients with resting PAH than in the healthy volunteers, but the difference was clinically insignificant. No significant difference was found between mitral E/E' values in the four groups.

The diameter of the right ventricle was significantly larger in patients with resting PAH than in subjects in the other three groups. In the group of patients with stress-induced PAH, only diastolic, while in patients with resting PAH combined systolic and diastolic deterioration of right ventricular longitudinal function was found.

PAH is a severe, potentially life-threatening complication of CTDs, such as systemic sclerosis, systemic lupus erythematosus, mixed CTD and, to a lesser extent, rheumatoid arthritis, polymyositis and primary Sjögren's syndrome. The pathophysiological mechanisms leading to PAH and consequentially to right heart dysfunction are thought to be similar in these diseases [2].

Determination of the tricuspid annular velocities provides an excellent tool for assessing the global systolic and diastolic function of the right ventricle [3]. The long axis function is guided by subendocardial fibres, which are most vulnerable to transitional or permanent pressure overload in patients with stress-induced or resting pulmonary artery hypertension. Our results suggest that the isolated diastolic dysfunction of the right ventricle is a sign of stress-induced pulmonary hypertension, while the resting elevation of pulmonary artery pressure is characterised by combined systolic and diastolic dysfunction. The latter observation is in accordance with the result reported by RUAN and NAGUEH [4] regarding patients with idiopathic pulmonary hypertension. Elevated ventricular pressure, however, is not the only cause of the right ventricular dysfunction in patients with connective tissue disease. Disturbance of

myocardial microcirculation and interstitial fibrosis as the hallmarks of primary myocardial involvement may also damage the function of the subendocardial fibres [5]. Nevertheless, tissue Doppler imaging seems to be a promising new method to confirm or eliminate suspicion of stress-induced pulmonary hypertension in patients with connective tissue disease when the noninvasively estimated pulmonary artery pressure is normal at rest.

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#### STATEMENT OF INTEREST

None declared.

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DOI: 10.1183/09031936.00124207