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Title: Exhaled nitric oxide in reactive pulmonary hypertension

Prof. Caterina 23419 Bucca caterina.bucca@unito.it MD [1], Dr. Pierluigi 23445 Omedè pierluigiomede@gmail.com MD [3], Dr. Walter 23446 Grosso Marra grossomarra@yahoo.it MD [3], Dr. Irene 23447 Parisi ire.parisi@gmail.com MD [1], Dr. Arianna 23457 Vaudano vaudano.arianna@hotmail.it MD [1], Dr. Marta 23458 Malandra mirtilla8@hotmail.it MD [1], Dr. Monica 23464 Masoero raerte@yahoo.it MD [1], Prof. Luisa 23465 Brussino luisa.brussino@unito.it MD [1], Dr. Corrado 24402 Magnino corrado.magnino@gmail.com MD [2] and Dr. A. 90000 Milan alberto.milan@unito.it MD [2]. [1] Clinical Pathophysiology, University, Torino, Italy, 10126 ; [2] Dip. Medicina Ed Oncologia Sperimentale, University, Torino, Italy, 10126 and [3] Cardiologia 1, AOU San Giovanni Battista, Torino, Italy, 10126 .

Body: Background. Pulmonary hypertension (PH) frequently complicates heart failure. In some patients, pulmonary vessels undergo reactive changes due to the chronic elevation of the left ventricular pressure, resulting in severe pulmonary hypertension and increased transpulmonary gradient (TPG). There is evidence that nitric oxide (NO) synthesized by the respiratory epithelium plays a role in the regulation of pulmonary artery pressure. Aims and objectives. To evaluate whether exhaled NO has a role in reactive PH Methods. Seven patients with reactive PH (rPH) were compared to 14 patients with passive PH (pPH) and to 15 control patients without PH. All the patients underwent heart catheterization, lung function tests and exhaled NO (FENO), assessed at multiple flow-rates. Alveolar NO and bronchial NO flux (J'awNO) were calculated using the slope–intercept model. Results. The results are displayed in the Table.

Comparison of haemodynamics, lung function and J'awNO among CP, rPH, pPH patients

	CP	rPH	pPH
TPG ratio	8.27 ± 0.70	20.71±2.41*§	9.57±0.80
Pcwp mmHg	11.73±1.07	25.33±3.16*	23.64±1.64
FEV1/VC%	74.51±2.73	63.50±4.10*	71.84±2.56
FEF50%	69.91±12.46	29.14±4.99*§	52.08±6.94
TLCO%	67.93±6.97	38.00±8.71*§	65.14±4.42
J'awNO nL/min	99.88±17.14	24.17±7.63*§	89.83±16.07

* significantly different from CP; § significantly different from PHp

Patients with PHr had significantly lower FEV1/VC% ratio, lung diffusion (TLCO) and J'awNO. J'awNO was

closely inversely related to TPG (r=0.385, p=0.032). Conclusion. It is still unknown why some patients develop severe and/or fixed PH with the same degree of elevated left-sided filling pressure. Our findings suggest that decreased bronchial NO flux and lung diffusing capacity may contribute to reactive PH.