

Research Article

Assessment of Pulmonary Artery Pulsatility by Multidetector Computed Tomography in Patients Affected by Chronic Obstructive Pulmonary Disease and Pulmonary Hypertension: Preliminary Data

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The aim was to assess if computed tomography is able to measure pulmonary artery pulsatility in patients affected by chronic obstructive pulmonary disease and to ascertain whether pulsatility is different in patients with and without pulmonary hypertension and whether it is related to haemodynamics. We selected two groups of patients, the first one with pulmonary hypertension and the second one without. In patient with hypertension, pulmonary artery pressure and resistance were increased with the increased diameters (transverse 36 ± 5 mm and axial 38 ± 4 mm versus 22 ± 3 and 25 ± 5 , resp.), the increased cross-sectional area (10 ± 08 versus 4 ± 1 cm²), and the reduced pulsatility (21 ± 7 versus $10\% \pm 5\%$). Arterial stretching was decreased in patients with hypertension (10 ± 5 versus $21\% \pm 7\%$) and significantly related to pulmonary vascular resistances and pressure. Cardiac output measured by tomography was significantly related to that obtained by Fick method and was not different in the two groups. The diameters allow to identify patients with PH, assuming a cut-off of 28 mm and assuming a pulsatility of right branch of 26% as well. These preliminary observations indicate tomography as a suitable technique, being able to measure the pulsatility and the dimensions of the arteries and the right ventricular functional parameters.

1. Introduction

The assessment of pulmonary hypertension (PH) during the chronic obstructive pulmonary disease (COPD) is important, since PH worsens quality of life, prognosis, effort tolerance, and outcome in acute respiratory failure [1–3]. Precapillary PH can be seen in 30% to 43% of patients affected by COPD, reaching values higher than 45 mmHg in 5% [1–3]. PH is determined by both functional vasoconstriction and vessels remodeling [3–5] that determine the right ventricular afterload leading to effort intolerance, poor quality of life, cardiac failure, and reduced life expectancy. The assessment of pulsatility (Puls) of pulmonary artery (PA) in PH looks like a promising tool, since it looks related to severity, progression of disease, functional capacity, prognosis, and survival [6–9]. The invasive right heart catheterization (RHC) is the gold

standard for the diagnosis of PH, and the assessment of reversibility of PH with vasodilators, the measure of Puls by intravascular ultrasound (IVU) [10–14]. The noninvasive cardiac echocardiography (US) allows the measurement of the pulmonary artery dimensions, the right ventricular shape, the ejection fraction, and the noninvasive estimation of the systolic pulmonary artery pressure [15–18], but the applicability of US in patients affected by COPD is limited because of the reduced acoustic window [15], due to pulmonary hyperinflation. Within the imaging methods, magnetic resonance imaging (MRI) is extensively studied and allows the measurement of both morphologic and functional data such as Puls and pulmonary blood flow [19–21], but it is not widely available. Multidetector computer Tomography (MDCT) and cardiac software imaging are diffusely employed and available; they allow the detailed examination

of heart, coronary vessels, left ventricular function, and pulmonary arteries diameters [22–28]. Our aims are to study whether MDCT allows the assessment of Puls in COPD, whether Puls is different between patients with and without PH, and whether it is related to hypertension.

2. Methods

2.1. Selection of Patients. All the patients were affected by COPD (diagnosed according to ATS statement and GOLD criteria) [29], and they were examined as soon as they consecutively come in the ward of pulmonary division. Patients were assigned to a specific GOLD stage, according to post bronchodilator FEV1.

The inclusion criteria were (a) presence of COPD and (b) the written informed consent and motivation. The exclusion criteria were (a) the significant cardiac diseases such as valvulopathies or cardiac failure defined by a fraction ejection of left ventricle lesser than 50% (measured by echocardiography), (b) the presence of other pulmonary diseases as pulmonary fibrosis, tuberculosis, or chronic embolic disease (already diagnosed by scintigraphy), (c) the allergy to iodine compounds, (d) the recent significant radiation loads due to isotopic or radiological procedures, (e) the psychiatric disorders as claustrophobia or panic attacks, (f) the sleep apnoea syndrome (diagnosed by polysomnography), and (g) the absence of acute relapses. All the enrolled patients were examined by clinical check, pulmonary functional testing, blood gas analysis, echocardiography, six minutes walking test (6 MWD), RHC in the first day, and chest CT examination one day after. The study started on April 2008 and ended one year later. The operator was unaware of the presence of precapillary PH that was diagnosed under RHC by a mean pulmonary artery pressure higher than 25 mmHg with a capillary pressure lower than 15 mmHg measured during RHC. Within one year, only twenty patients with PH were recruited because of the low prevalence of the disease and the refuse of the informed consent; the enrolled patients were compared to the first consecutive twenty patients without PH.

The goal to access the applicability of MDCT in the assessment of Puls was accomplished examining the success rate in the imaging of PA diameters and surface area in diastole and systole. The difference of Puls within patients with and without PH was obtained comparing the mean by a statistical package and observing the relationship between Puls and PAP or RVP.

2.2. Physiological Measurements. Lung volumes were measured by a whole body chest plethysmography (Pulmobox 6200, SensorMedics, California, USA). Measured volumes were referred to ERS normal standards [30]. Vital capacity (VC) and forced expiratory volume in the 1st second (FEV1) were reported.

Blood gas analysis was carried out by radial artery puncture (performed at rest whilst breathing air) and automated analysis (Rapidlab Bayer, FRG), measuring arterial oxygen tension (PaO₂) and haemoglobin oxygen saturation (SaO₂).

Mixed venous oxygen tension (PvO₂) and saturation (SvO₂) were obtained by pulmonary artery sampling.

Effort tolerance was measured by 6 MWD [31] and dyspnoea by WHO rating scale [32]. RHC was performed by cubital vein catheterization, according to Seldinger's technique using Swan-Ganz catheters (Baxter, USA) [33].

2.3. Haemodynamic Measurements. Pulmonary artery pressure (PAP) was assessed both in stable state and under NO (20 ppm) inhalation. Cardiac output (Q') was obtained using Fick method by means of simultaneous pulmonary and radial arteries sampling (to measure oxygen content) together with the measurement of oxygen consumption (Cortex Metalyzer, Germany). Pulmonary wedge pressure (Pw) measurement allowed pulmonary vascular resistances (PVRs), computed dividing pressure drop by cardiac output (PAP – Pw/Q'). All patients showed Pw lesser than 15 mmHg.

2.4. Imaging Measurements. Computed tomography was accomplished by a 64-detector tomograph (Philips Brilliance 64, Germany). A first whole chest scanning was performed by a low dosage radiation without contrast medium with high resolution technique (100 kV; 95 mAs; 64 × 0, 625 mm; t rot 0, 75 sec; pitch 1.078) held at functional residual capacity and in forced expiration to check for concomitant pulmonary diseases or associated conditions. A "test bolus" technique was used before injecting the contrast medium (Iomeprol 400, Bracco, Italy) to compute the time necessary for the simultaneous imaging of both the pulmonary trunk and the right ventricle, choosing as region of interest (ROI) the pulmonary trunk, the right atrium and the left ventricle. Afterwards the acquisition with contrast medium started, using cardiac gating (120 kV; 350 mAs; 64 × 0, 625 mm; t rot 0, 4 sec; pitch 0.299), from the aortic arch to the cardiac base, including the pulmonary arteries up to two centimetres from the thoracic cage wall in the reconstruction algorithm. The contrast medium was injected at a flow rate of 4 mL/sec and at a concentration of 400 mg/mL, by a double lumen injector, according to the sequence: 80 mL of solution with 60% mdc and 40% sterile saline, followed by 40 mL of contrast medium and 40 mL of sterile saline. Two doctors measured the longitudinal and the transverse diameters of both pulmonary arteries in CT observed under mediastinal gating, choosing a plan perpendicular to the path of vessels. The measures were obtained at the end-systolic and the end-diastolic frames, carefully checking during acquisition that the cardiac frequency was in the range of 55 to 85 bpm. The end-systolic frame happens around the 40% of the cardiac cycle and the end-systolic frame around the 70% (Figures 1 and 2). The cross-sectional area was measured as well, and the pulsatility of arteries was measured by the ratio between the systolic and the diastolic surface areas. By means of a dedicated software, it was possible to measure the ejection fraction and the cardiac output of right ventricle.

2.5. Statistical Analysis. Biometric data, pulmonary function, effort performance, and hemodynamic and radiologic variables of patients with and without pulmonary hypertension

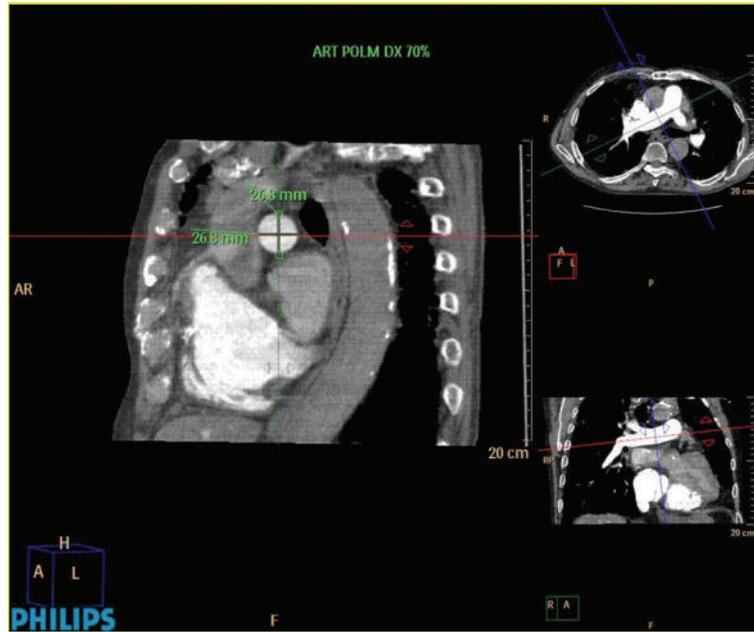


FIGURE 1: Assessment of pulmonary diameters in mediastinal window at end-diastolic phase. The measurement of pulmonary artery diameters was repeated in end-systolic and end-diastolic phases; the ratio allowed the assessment of pulsatility of pulmonary artery.

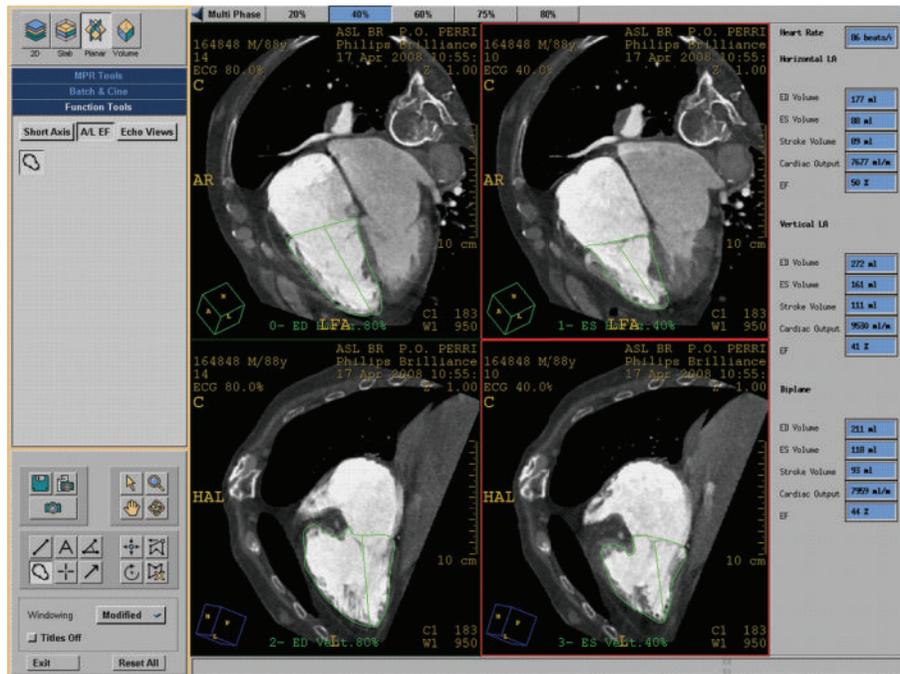


FIGURE 2: Assessment of cardiac performance. Delimitation of region of interest in end-systolic and end-diastolic phases. Computer-aided calculation of systolic volume and cardioutput.

were compared by the analysis of the mean with Student's *t*-test for unpaired data (Tables 1 and 2) by means of computer-aided software (Epistat, USA, and Graphpad Prism, USA). The relationships between pulmonary artery features and hemodynamic variables and between cardiac output assessed

by MDCT and by Fick method were assessed by linear fitting and least square method.

The study was approved by the Ethics Committee of "Ninotto Melli" Hospital. A written informed consent was obtained from each patient.

3. Results

3.1. Population Studied. Patients showed marked airway obstruction and marked dyspnoea (Table 1). The degree of airway obstruction was advanced, and most patients belonged to the 3rd stage of the disease in both groups (Table 1). The frequency of patients in each stage of COPD was similar in the two groups. The difference between patients with PH and without PH was not significant. Hypoxemic hypercapnic chronic respiratory failure could be observed, without significant difference within patients. Patients affected by COPD and PH showed diminished effort tolerance with a significantly lower 6 MWD and an increased PAP and PVR (Table 1), while cardiac output determined by Fick method (Q' Fick) was similar.

3.2. MDCT-Derived PA Diameters and Pulsatility. MDCT allowed suitable imaging and effective measurement in every patient. Pulmonary artery (Table 2) was significantly increased both in axial and in transversal diameters in patients with PH with a mean difference of about 14 mm between COPD and COPD with PH.

The diastolic diameters were about 2 mm less than the systolic ones. Right PA diameters are about 6 mm lower than those of the main PA. Left PA branches were slightly narrower than right ones in both series.

Two patients with PH had a PA diameter between 30 mm and 28 mm, the remaining had a diameter larger than 30 mm, while two patients without PH showed a PA diameter larger than 30 mm. Assuming a cut-off of 28 mm, a sensitivity of 90%, specificity of 85%, a positive predictive power of 85%, and a negative predictive power of 89% could be obtained. Cross-sectional area of right and left arteries was about 40% smaller than PA area with narrower values in the left side. It was significantly increased in patients with PH (Table 2).

The Puls of PA is about 20% in the PA and in the left branch of COPD patients; right PA showed increased values ($38\% \pm 5\%$) (Table 2). The Puls was significantly diminished (mean 10%) in patients with PH both in PA and in branches, mainly in the left side (2%). It was not possible to choose a threshold for the Puls of the main pulmonary artery to diagnose the presence of PH, because of the large transvariance of the distributions between patients with and without PH. Since the stretching of right PA looks enhanced and the difference looks wider, assuming as a threshold the lower limit of right PA Puls (mean minus two standard deviations), a cut-off of 26% is associated with a sensitivity of 90%, a specificity of 85%, a positive predictive power of 90%, and a negative predictive power of 85% could be obtained. Left side was not useful to avoid bias in reading the frames due to very low values.

Aorta diameters were normal in both patient sets (transverse diameter 29 ± 8 mm, axial diameter 29 ± 5 mm, and area $2.64 \pm .4$ cm² in patients with PH and 30 ± 3 mm, 30 ± 3 mm and $2.86 \pm .5$ cm² in patients without PH, resp.), and pulmonary artery/aorta ratio was significantly increased in patients with PH ($1.2 \pm .2$ in patients with PH and $.8 \pm .2$ in patients without PH, resp.).

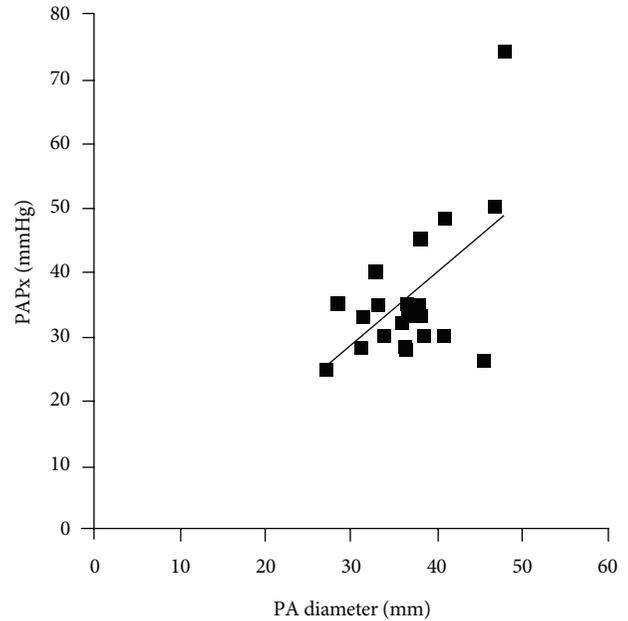


FIGURE 3: Scatter plot of the relationship between pulmonary artery pressure and pulmonary artery Diameter.

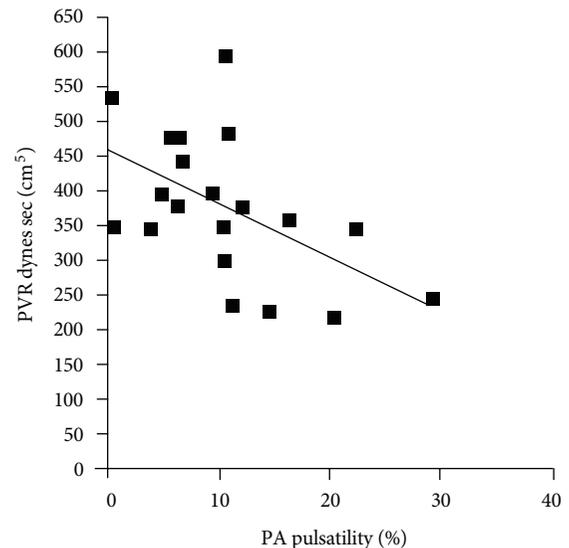


FIGURE 4: The relationship between pulmonary vascular resistance and pulsatility of pulmonary artery.

3.3. The Relationship with Haemodynamics. The Puls was significantly inversely related to PVR ($PVR = 458 - 7.53$ pulsatility ± 95 ; $r^2 = .274$; $P = .0177$) (Figure 4) and PAP ($PAP = 38 - 2.5$ pulsatility ± 5 , $r^2 = .219$; $P = .0371$) (Figure 5). PA diameter was related to PH level (Figure 3) (systolic PA diameter = $-6.1 + 1.14$ PAP $\pm .38$; $r^2 = .33$; $P = .0075$; diastolic PA diameter = $-10 + 1.32$ PAP; $\pm .34$; $r^2 = .38$; $P = .0018$). By grouping of patients according to the WHO staging of PH, PA diameter reached an average value of 32 ± 3 mm in the 1st stage of PH (PAP < 25 at rest but

TABLE 1: Functional values observed in patients affected by COPD with PH under stable state.

	Units	COPD + PH	COPD	<i>P</i>
Age	Years old	66 ± 9	65 ± 10	n.s.
Height	cm	165 ± 8	167 ± 10	n.s.
Weight	kgms	75 ± 13	73 ± 9	n.s.
GOLD st. II	%	25	20	
GOLD st. III	%	55	50	
GOLD st. IV	%	20	30	
FVC	%	49 ± 18	53 ± 15	n.s.
FEV1	%	37 ± 18	39 ± 17	n.s.
PaO ₂	mmHg	57 ± 10	58 ± 9	n.s.
PaCO ₂	mmHg	46 ± 8	46 ± 9	n.s.
PAP	mmHg	37 ± 5	18 ± 5	<.1 × 10 ⁻⁶
Q' Fick	L/m	4.9 ± 1.4	5.1 ± 1.2	n.s.
Q' radiol	L/m	4.1 ± 1.0	4.2 ± 1.4	n.s.
PVR	Dynes sec ⁻¹ cm ⁻⁵	442 ± 192	120 ± 70	<.1 × 10 ⁻⁶
6 MWD	Mt	257 ± 118	370 ± 150	<.02246
Dyspnoea	WHO stage	III ± I	III ± I	n.s.
<i>N</i>	<i>n</i>	20	20	

FVC: forced vital capacity as percentage of predicted values; FEV1: forced expired volume in the 1 second as percentage of predicted values; PaO₂: arterial oxygen tension; PaCO₂: arterial carbon dioxide tension; PAP: mean pulmonary artery pressure; Q': cardiac output measured by radiologic method (radiol) and Fick method (Fick); PVR: pulmonary vascular resistance; 6 MWD: six minutes walking distance; WHO: dyspnoea rating according to WHO stages; and GOLD st. II-III and IV: % of patients belonging to GOLD stages II, III, and IV; Values are expressed as mean ± standard deviation; *N*: number of observations.

TABLE 2: Pulmonary artery features in COPD.

	COPD + PH	COPD	Units	<i>P</i>
Main pulmonary artery				
Transverse systolic <i>d</i>	36 ± 5	22 ± 3	Mm	<.1 × 10 ⁻⁶
Axial systolic <i>d</i>	38 ± 4	25 ± 5	Mm	<.1 × 10 ⁻⁶
Systolic Area	10.8 ± 2	4.3 ± 1	cm ²	<.1 × 10 ⁻⁶
Transverse diastolic <i>d</i>	34 ± 2	20 ± 5	mm	<.1 × 10 ⁻⁶
Axial diastolic <i>d</i>	36 ± 3	22 ± 4	mm	<.1 × 10 ⁻⁶
Diastolic area	9.6 ± 1	3.4 ± 1	cm ²	<.1 × 10 ⁻⁶
Pulsatility	10 ± 05	21 ± 07	%	<.1 × 10 ⁻⁶
Right pulmonary artery				
Transverse Systolic <i>d</i>	30 ± 1	21 ± 8	mm	<.1 × 10 ⁻⁶
Axial Systolic <i>d</i> .	30 ± 1	21 ± 7	mm	<.1 × 10 ⁻⁶
Systolic area	7.1 ± 1	3.6 ± .8	cm ²	<.1 × 10 ⁻⁶
Transverse diastolic <i>d</i>	28 ± 2	17 ± 6	mm	<.1 × 10 ⁻⁶
Axial diastolic <i>d</i>	29 ± 2	16 ± 6	mm	<.1 × 10 ⁻⁶
Diastolic area	6.4 ± .9	2.2 ± .6	cm ²	<.1 × 10 ⁻⁶
Pulsatility	10 ± 05	38 ± 05	%	<.1 × 10 ⁻⁶
Left pulmonary artery				
Transverse Systolic <i>d</i>	28 ± 2	18 ± 5	mm	<.1 × 10 ⁻⁶
Axial Systolic <i>d</i>	29 ± 1	19 ± 4	mm	<.1 × 10 ⁻⁶
Systolic area	6.3 ± .9	2.7 ± .6	cm ²	<.1 × 10 ⁻⁶
Transverse diastolic <i>d</i>	28 ± 2	16 ± 3	mm	<.1 × 10 ⁻⁶
Axial diastolic <i>d</i>	28 ± 2	19 ± 5	mm	<.1 × 10 ⁻⁶
Diastolic area	6.3 ± .8	2.1 ± .5	cm ²	<.1 × 10 ⁻⁶
Pulsatility	2.1 ± .5	21 ± .5	%	<.1 × 10 ⁻⁶

COPD + PH: pulmonary artery diameter in patients with COPD and pulmonary hypertension; COPD: pulmonary artery diameter in patients with COPD and normal pulmonary artery pressure; transverse systolic *d*: transverse systolic diameter; axial systolic *d*: axial systolic diameter; and systolic area: sectional systolic area.

Mean ± SD; *P*: level of probability, n.s.: not significant; right: right pulmonary artery; left: left pulmonary artery; pulsatility: ratio between systolic and diastolic cross-sectional areas. Number of observations: 20 for each set.

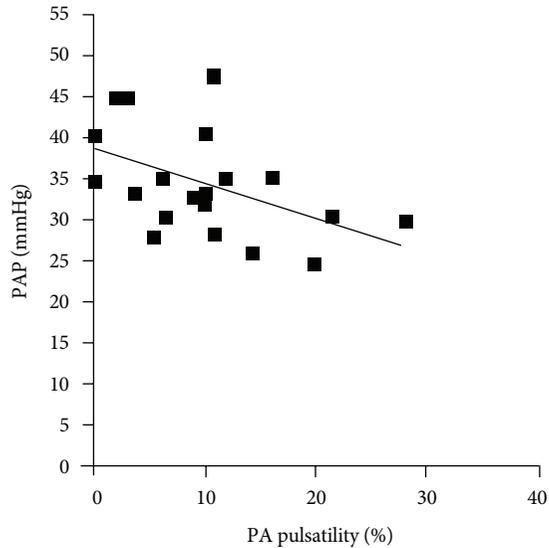


FIGURE 5: The relationship between pulmonary artery pressure and pulsatility of pulmonary artery pressure.

>25 mmHg on effort), 34 ± 3 mm in the 2nd one (PAP > 25 and <35 mmHg), 36 ± 3 mm in the 3rd one (PAP > 35 and <45), and up to 46 ± 3 mm in the 4th stage (PAP > 45 mmHg). The cardiac output measured by CT ($Q'_{rad} = 4.1 \pm 1$ L/m) underscores the blood flow measured by Fick method (Q'_{Fick}). The two measurements are significantly related ($Q'_{Fick} = 1.4 + .96 Q'_{rad} (+.09)$; $P < .001$).

3.4. Additional Outcomes. CT allowed the assessment of pulmonary nodules (2 patients), arteriovenous malformations (one patient), and severe coronary pathology (three cases). The average absorbed dose was 8 ± 2 mSv.

4. Discussion

Our study indicates for the first time the chance to apply CT cardiac imaging software in the study of pulmonary vessels and right ventricular performance. This method allows to appreciate significant differences of PA dimensions, cross-sectional areas, and PA stretching in patients with and without PH. The measurements are related to physiological measurements obtained under RHC.

The main limitation of the current study is the low number of observations, due to the low prevalence of the disease and to the difficulties in the recruitment because of the lack of compliance of patients; thus the current data can be regarded just as preliminary outcomes to be confirmed by multicentric studies.

The first outcome is that, according to our results, MDCT cardiac software imaging technique is able to measure the features of pulmonary arteries as well as the right ventricular performance, and it can be used in every COPD patient, while US applicability is limited by the reduced acoustic window and MRI is still not diffuse everywhere and is more expensive and time consuming. MDCT allows as well

an effective imaging of the associated pulmonary such as coronary diseases and pulmonary emboli. The applicability is not fully extensible in other pulmonary diseases associated with PH such as interstitial lung disease, because of the interference of fibrosis upon reading of frames and differences in involvement of vessels [34, 35].

The second observation is the observation of a diminished arterial Puls in patients with PH. The applicability in the early diagnosis of PH looks limited, because the Puls of main PA ($21\% \pm 07\%$ in COPD versus $10\% \pm 05\%$ in COPD + PH) cannot play a role because of the large overlap of the distributions between patients with COPD and COPD with PH, although the pulsatility of right PA (38 ± 05 in COPD versus $10\% \pm 05\%$ in COPD + PH) looks as a promising index to be verified in more extensive studies.

MDCT-derived pulsatility fairly agrees with that measured by IVU and MRI [11–21], indicating a mean pulsatility of $20\% \pm 5\%$. The pulsatility significantly diminishes in patients affected by PH, because of arterial remodelling leading to arterial stiffness [16, 17].

The relationship between the pulsatility of central arteries and PVR, mainly determined by peripheral vessels, can be explained observing that pathology determines contemporary structural changes in both central and peripheral vessels. The inverse relationship between Puls and PAP depends on the fact that as peripheral vessel's pathology worsens, determining an increase in PVR and PAP, the pulmonary artery dilates according to its elastic modulus and becomes proportionally stiffer with the enlargement of diameters together with the lack of progression of elastic waves into the peripheral units. Furthermore, as the PAP and the right ventricular afterload (determined by the vascular impedance) increase, the right ventricular performance is impaired with the decrease in the systolic volume (the main determinant of pulsatility) and the evidence of pulmonary valve regurgitation [36–38].

The reported value of pulsatility in the prognosis [9] can be explained by the observed relationship between Puls and haemodynamics: the pulmonary artery stretching is dependent on the degree of PH, but it determines as well the ventricular/arterial coupling. Controversial reports indicate higher values in patients responders to vasodilators, suggesting a possible role in the evaluation of reversibility of PH and the selection of patients responders to calcium channel blockers [39, 40].

MDCT allows as well to study the additional features of pulmonary arteries, such as the diameters. According to preliminary reports in the literature [24–29] and our results, the diameters allow the detection of PH with fairly good sensitivity and specificity. Assuming 28 mm as the upper limit of normality (mean + 2SD) of PA, a sensitivity of 90%, a specificity of 85%, and a positive predictive value of 96% can be obtained by our study with outcomes similar to those obtained in larger series [24–29]. The variance observed around the slope of the relationship between diameters and PAP can be explained by several reasons: (1) differences in the amount of elastic bundles of the main pulmonary artery tissues; (2) dilation determined by previous loads, such as higher PAP peaks preceding the measures, heavier than

the actual measured PAP; (3) differences in cardiac output; (4) different body surface area, since cross-sectional area of PA and left branch corrected by body surface area were significantly related to PAP; and (5) time of measurement referred to the natural history of disease, since pulmonary artery progressively increases even in case of reduction of PVR by vasodilators, due to intrinsic vessel properties independent of pressure and flow changes. Other relevant findings in the literature are the arterial/bronchial ratio $> 1,1$ in lobar arteries and the ratio PA/Aorta higher than 1 and close to $1.2 \pm .3$, as found in our patients as well [24–29].

In conclusion, MDCT could be applied in every COPD patient, allowing the measurement of PA pulsatility, lesser in patients affected by PH and closely related to the haemodynamic variables. Pulsatility can contribute to raise the suspicion of the existence of PH together with the measurement of diameters. MDCT allows as well an insight in to the performance of RV by means of the assessment of the cardiac output related to the hemodynamic measures, and it offers the chance to verify additional thoracic or cardiac lesions, justifying the radiation dose absorbed. These findings are relevant not only as scientific outcomes but as well in clinical practice because they offer a suitable and also available technique to be applied once in primary health care when the suspect of PH is raised.

5. Summary

The aim was to assess if multidetector computed tomography (MDCT) is able to measure pulmonary artery (PA) pulsatility (Puls) in patients affected by chronic obstructive pulmonary disease (COPD) and to ascertain whether Puls is different in patients with and without pulmonary hypertension (PH) and whether it is related to haemodynamics. Statistical analysis by Student's *t* test allowed the comparison of data between patients with and without PH. The linear fitting and the least square method allowed to assess a significant relationship between haemodynamics and imaging. MDCT allowed the imaging and the measurement of PA features in all the patients. We selected two groups of COPD patients, the first one with PH, and the second one without PH. In patients with COPD and PH PAP and PVR were increased (PAP 37 ± 18 versus 18 ± 5 mmHg, PVR 442 ± 192 versus 120 ± 70 dynes $s^{-1} cm^{-5}$) and PA showed increased diameters (transverse 36 ± 5 mm, axial 38 ± 4 mm versus 22 ± 3 and 25 ± 5 , resp.), increased cross-sectional area (10 ± 08 versus 4 ± 1 cm^2), and reduced pulsatility (21 ± 7 versus 10 ± 5 %). Arterial stretching was decreased in patients with PH ($10\% \pm 5$ versus 21 ± 7 % in non-PH) and significantly related to pulmonary vascular resistances (PVRs) (PVR = $458 - 7.53$ pulsatility ± 95 ; $r^2 = .274$; $P = .0177$) and pulmonary artery pressure (PAP) PAP = $38 - 2.5$ pulsatility ± 5 , $r^2 = .219$; $P = .0371$). Cardiac output measured by MDCT was significantly related to that obtained by Fick method (4.1 ± 1.0 L/m versus 4.9 ± 1.4 , resp.) and was not different in the two groups. The diameters of PA allow to identify patients with PH with a confident specificity and sensitivity: assuming a cut-off of 28 mm for the diameter of PA, it was possible to

diagnose pulmonary hypertension with a sensitivity of 90%, a specificity of 85%, a positive predictive power of 85%, and a negative predictive power of 89%. While the distribution of PA Puls was overlapping, the Puls of right PA, assuming a cut off of 26%, allowed to diagnose PH with a specificity of 85%, a positive predictive power of 90%, and a negative predictive power of 85%. These preliminary observations, to be confirmed in multicentric and more extensive studies, indicate MDCT as a suitable technique in COPD, able to measure the Puls and the dimensions of pulmonary arteries as well as the right ventricular functional parameters.

Abbreviations

Puls:	Pulsatility
MDCT:	Multidetector tomography
PH:	Pulmonary hypertension
PAP:	Pulmonary artery pressure
PA:	Pulmonary artery
PVRs:	Pulmonary vascular resistances
Q':	Cardiac output
COPD:	Chronic obstructive pulmonary disease
RHC:	Right heart catheterization.

Conflict of Interest

The authors declare that they have no conflict of interests, and the study was supported by no grant.

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