CHEST

Original Research

COPD

Reduced Intrathoracic Blood Volume and Left and Right Ventricular Dimensions in Patients With Severe Emphysema*

An MRI Study

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Background: **Left ventricular (LV) filling is impaired in patients with severe emphysema manifesting in small end-diastolic dimensions. We hypothesized that the hyperinflated lungs of these patients with high intrinsic positive end-expiratory pressure will decrease intrathoracic blood volume (ITBV) and ventricular preload. We therefore measured ITBV, and LV and right ventricular (RV) dimensions and function using MRI techniques in patients with severe emphysema.**

Methods: **Patients with severe emphysema (n = 13) and matched healthy volunteers (n = 11) were included. The magnetic resonance (MR) examination consisted of three parts: (1) evaluation of RV and LV dimensions and function and interventricular septum curvature using cine MRI; (2) quantification of aortic flow using MR phase velocity mapping; and (3) calculation of the cardiopulmonary peak transit time (PTT) from the pulmonary artery to the ascending aorta using contrast-enhanced, time-resolved, two-dimensional MR angiography.**

Results: **There were no differences between the groups regarding age, height, or weight. In the emphysema patients, ITBV index (− 35%), LV end-diastolic volume index (LVEDVI) [− 21%], RV end-diastolic volume index (− 20%), cardiac index (− 22%), and stroke volume index (SVI) [− 40%] were lower compared to control subjects. LV and RV end-systolic volumes, LV wall mass, septal curvature, and PTT did not differ between the groups. LVEDVI (r = 0.83) as well as SVI (r = 0.82) correlated closely to ITBV index. SVI correlated closely to LVEDVI (r = 0.84).**

Conclusions: **LV and RV performance is impaired in patients with severe emphysema because of small end-diastolic dimensions. One possible explanation for the decreased biventricular preload in these patients is intrathoracic hypovolemia caused by hyperinflated lungs.**

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Key words: biventricular end-diastolic volumes; biventricular function; cardiac output; emphysema; intrathoracic blood volume; mean transit time; MRI

Abbreviations: AO = ascending aorta; BSA = body surface area; CI = cardiac index; CO = cardiac output; ITBV = intrathoracic blood volume; ITBVI = intrathoracic blood volume index; LV = left ventricular; LVEDV = left ventricular end-diastolic volume; LVEDVI = left ventricular end-diastolic volume index; LVSVI = left ventricular stroke volume index; MR = magnetic resonance; NONMEM = nonlinear mixed effect modeling; PA = pulmonary artery; PEEPi = intrinsic positive end-expiratory pressure; PTT = peak transit time; qf = quantitative aortic flow; ROI = regions of interest; RV = right ventricular; RVEDVI = right ventricular end-diastolic volume index; RVSVI = right ventricular stroke volume index; SV = stroke volume; SVI = stroke volume index

Patients with severe lung emphysema have poor quality of life because of impaired lung function and considerable reduction in exercise tolerance.[1] The functional features consist of severe expiratory airflow obstruction and considerable hyperinflation due to destruction of lung parenchyma and loss of lung elasticity. Intrathoracic (intrapleural) pressure is increased due to generation of a high intrinsic positive end-expiratory pressure (PEEPi).[2,3]

We have studied[4] left ventricular (LV) diastolic

and systolic function in patients with severe emphysema and found that LV function is impaired in patients with severe emphysema due to small LV end-diastolic dimensions, *ie*, a decrease in LV preload. The present investigation is an extension of that study, and the aim was to evaluate whether or not the hyperinflated lungs in patients with severe emphysema will cause blood to pool peripherally and thus decrease intrathoracic blood volume (ITBV) and LV and right ventricular (RV) end-diastolic volumes (preload), stroke volume (SV), and stroke work.[5] We therefore measured ITBV and LV and RV dimensions and function in patients with severe emphysema using MRI techniques.

Materials and Methods

The local Ethics Committee of the Medical Faculty of Göteborg University approved the study protocol, written informed consent was obtained from all subjects, and the study complied with the recommendations found in the Declaration of Helsinki.[6] Criteria for inclusion in the emphysema group (n = 13) were a history of lung emphysema based on physical examination, chest radiography, and pulmonary function test results: FEV_1 from 15 to 30% of predicted value; total lung capacity > 120% of predicted value; and residual volume > 200% of predicted value. Furthermore, the emphysema patients should have no history of cardiac disease, a normal echocardiographic examination (LV ejection fraction > 50% and systolic pulmonary artery [PA] pressure < 55 mm Hg). These criteria are in concordance with commonly accepted guidelines for single-lung transplantation.[7] The control group consisted of healthy volunteers (n = 11) with no history of cardiopulmonary disease and were matched for age, gender, and body size.

Imaging Protocol

Each subject underwent a single magnetic resonance (MR) examination (1.5-T MR system, Philips Intera, R9.3; Philips Medical Systems; Best, the Netherlands). [Figs 1–6]. Heart rate was continuously recorded, and systolic and diastolic arterial BPs (by sphygmomanometer) were recorded before the start of imaging. Subjects in the emphysema group were allowed to breathe oxygen-enriched air during the entire examination.

Cardiac Volumes and Function by Cine MRI

Values for cardiac volume and function by cine MRI were obtained once in all participants. During the examination, multiple slices through the heart were acquired to encompass completely the ventricle in multiple phases within the cardiac cycle by using ECG triggering. The images were acquired during breath-hold in expiration with approximately 12 sections through the left ventricle in the short-axis view. In the short axis, the contours describing the endocardial and epicardial border of the myocardium were delineated, and the following LV and RV parameters were evaluated using commercially available software (Easy Vision 5.1; Philips Medical Systems): ejection fraction, end-diastolic volume, end-systolic volume, and SV. Endocardial contours were traced on the diastolic and systolic images, and the ventricular volume (diastolic or systolic) equals the sum of all the endocardial areas (of the diastolic or systolic images, respectively) multiplied by the slice thickness. The LV wall mass was calculated by tracing the epicardial borders in diastole to obtain an epicardial volume. The volume of the myocardium was defined as the epicardial volume minus LV end-diastolic volume (LVEDV). Multiplication of this value by the specific gravity for muscle (1.05 g/mL) yields the myocardial mass.[8,9] Papillary muscles were included in the volume and excluded in the mass determination.[10] Septal bowing was measured in the short-axis image plane as the septal curvature, defined as 1/septal radius in centimeters, as described by Roeleveld et al.[11] The cine image with the lowest septal curvature was used for quantification.

Quantification of Aortic Flow Using MR Phase Velocity Mapping

Quantification of aortic flow using MR phase velocity mapping was performed once during breath-holding using a phase-contrast ECG-triggered two-dimensional fast-field echo sequence at the level of the PA, perpendicularly to the ascending aorta (AO). SV was evaluated using the Easy Vision 5.1. Circular regions of interest (ROI) were placed over the AO. The contours of the ROI were delineated around the internal border of the vessel of interest on all images by automated contour detection. SV (quantitative aortic flow [qf]-SV) was computed by integrating the flow over a complete cardiac cycle.

Contrast-Enhanced, Time-Resolved, Two-Dimensional MR Angiography of the Heart and Lungs for Calculation of the Peak Transit Time

Contrast-enhanced, time-resolved, two-dimensional MR angiography of the heart and lungs for calculation of the peak transit time (PTT) was performed twice in each patient for evaluation of reproducibility. A two-dimensional, T1-weighted, flow-compensated fast-field echo sequence was applied at the level of the pulmonary trunk and AO during IV gadolinium bolus injection (2 mL bolus of gadopentetate dimeglumine; Magnevist; Berlex Laboratories; Wayne, NJ), followed by 20 mL of saline solution, injected at a rate of 5 mL/s using an automated power injector (Spectris Solaris; Medrad; Indianola, PA). Two-dimensional data sets were acquired at 0.559- to 1.1-s intervals (approximately 2 Hz) for 25 to 30 s after contrast material injection. Subjects were requested to hold their breath in expiration during the MR angiographic examination for at least 30 s or as long as was comfortable. Time-intensity curves were generated for bolus transit through the ROI (Intera R 9.3; Philips Medical Systems): the outflow part of the PA and the AO. The PA and AO curves were fitted to functions describing peak/pulse curves (PA) and multicompartment impulse response exponential decay curves (AO) using software (Origin Version 7; Origin Lab Corporation;

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