

Name *	Randall Grout
Email *	randall-grout@uiowa.edu
Educational Level *	Other
If Selected Other	M2
College *	College of Medicine
Department *	Radiology; Doris Duke Clinical Research Fellow
Title of Research *	Total Pulmonary Arterial Volume: A New Quantitative CT Phenotype of Emphysema?
Other Authors *	Zhiyun Gao, PhD (UI College of Engineering); Punam Saha, PhD (UI College of Engineering); Eric A. Hoffman, PhD (UI College of Medicine, College of Engineering)

Introduction & Purpose *

A hypothesis regarding the etiology of COPD describes endothelial dysfunction as a precursor to smoking-associated emphysema. We introduce a non-invasive, imaging-based measure of total pulmonary vascular volume (TPVV) as a phenotype of emphysema susceptibility. TPVV is defined as the volume of the lumen and wall of the pulmonary vascular tree. We hypothesize that increased TPVV may serve as an index of distal pathology, including endothelial dysfunction and the resultant subclinical pulmonary hypertension. Since venous volume may mask changes in the arterial tree, we refine these measures to isolate the total pulmonary arterial volume (TPAV).

Experimental Design *

Full-lung, non-contrast CT scans from 3 subjects (one each of non-smoker, smoker without emphysema on CT, and smoker with CT emphysema) randomly selected from a prior TPVV study were segmented into arterial and venous trees using a novel algorithm. Right lower lobe vessel volumes were determined inside a lobe mask reduced to 50% volume with 3D morphology preserved.

Results *

We successfully designed and implemented a system for objectively quantifying TPVV and TPAV. In a preliminary comparison with our previous method, TPAV normalized to the lobar region showed 4.5% vascular volume in the emphysematous subject compared to 3.8% in the two non-emphysematous subjects, whereas TPVV normalized to total lung volume did not distinguish between these subjects.

Conclusions *

Our standardized system objectively isolates a consistent portion of the arterial tree in unenhanced CT image datasets, and preliminary data suggests that TPAV may be more sensitive than TPVV in distinguishing emphysema-susceptible subjects. Recent research has shown endothelial dysfunction occurs early in COPD, and we show that pulmonary vascular volumes quantified through non-contrast CT can index the resultant subclinical pulmonary hypertension. TPAV may serve as a useful metric in determining the etiology and possible therapies for emphysema-type COPD.

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