

# Hemodynamic assessment of pulmonary artery on smokers with 3.0T phase-contrast MR imaging: initial experience

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TARGET AUDIENCE Physicians and Scientists who interested in pulmonary MRI

## INTRODUCTION & PURPOSE

Pulmonary vascular remodeling and dysfunction associated to tobacco smoking might pave the way for the subsequent development of pulmonary hypertension. Its prognosis is dreadful and its underlying mechanisms are unknown in humans. Smokers exhibited an impaired pulmonary endothelium-dependent vasodilation compared with non-smokers. Phase-contrast magnetic-resonance imaging (PC-MRI) is a proven non-invasive technique for quantifying velocity and blood flow in the great arteries. Our aim was to quantitatively investigate pulmonary blood flow of main pulmonary artery and its changes induced by smoking with 3.0T PC-MRI.

## METHOD & MATERIALS

The smoker group consisted in 10 men, mean aged  $50.0 \pm 12.17$  years, with a median cumulated smoking history of 50 pack·year (range 10–100). Our control group consisted in 10 men never smoking who all had no detectable intrinsic lung and heart diseases, their mean age was  $42.33 \pm 18.77$  years. The smoker group and control group all exhibited a normal lung function. They underwent PC-MRI of main pulmonary artery. PC-MRI scans were performed with flow quantification from the slices transverse to the ascending aorta and pulmonary trunk. The long axis image of MPA was prescribed parallel to the MPA long-axis in the axial image, as shown in Fig.1. An image plane was positioned perpendicular to the long-axis image of MPA, 1.5 cm–2 cm above the level of the pulmonary valves (Fig.2), resulting in magnitude and phase-map images. All imaging was done on a 3.0T TwinSpeed MRI Scanner (GE Healthcare). Institutional Review Board approval was obtained for the study protocol, and informed consent was obtained from all volunteers. Main measurement parameters included peak positive velocity, average flow, average positive flow and distensibility on Report Card 4.0 software. We quantitatively evaluated hemodynamic changes of pulmonary artery between smokers and non-smokers. Results are expressed as means  $\pm$  SE. For between-group comparisons, t-test was used for variables.

**RESULTS** Average flow and average positive flow of main pulmonary artery in smoker and non-smokers were  $62.16 \pm 8.83$  ml/beat,  $62.16 \pm 8.83$  ml/beat and  $86.90 \pm 9.29$  and  $93.30 \pm 8.07$  ml/beat, respectively. Average flow and average positive flow of main pulmonary artery in smokers group were significantly lower than these in non-smokers group, ( $t = -3.93$ ,  $p = 0.02$ ;  $t = -4.70$ ,  $p = 0.01$ ). Meanwhile, Peak positive velocity ( $98.33 \pm 29.29$  cm/s) and distensibility ( $33.44 \pm 4.46$ ) of main pulmonary artery in smokers group were also lower than these ( $124.33 \pm 34.29$  cm/s and  $40.43 \pm 20.03$ ) in non-smokers group, but there were no statistical differences between them ( $t = 1.14$ ,  $P = 0.29$ ;  $t = 0.84$ ,  $P = 0.41$ ).

## DISCUSSION & CONCLUSION

MR assessment of the pulmonary arteries provides detailed morphological images and flow information to diagnostic standards. MR imaging, is not restricted by conventional acoustic imaging planes, allowing the display of the entire flow jet in any plane or direction. PC-MR techniques are the best available in vivo tests of flow. Smoking can significantly enhance hypoxic pulmonary vasoconstriction, the process may be associated with smoking affect pulmonary vascular and alveolar macrophage arachidonic acid metabolism, increased pulmonary vascular resistance. Released when burning tobacco contains many harmful of chemical substances, such as nicotine, tar, carbon monoxide (CO), CO-derived free radicals. Smoke harmful components in peripheral arteries due to increased resistance and afterload, leading to elevated blood pressure. Cigarette smoking is related to endothelial dysfunction, increased expression of growth factors and inflammatory cell infiltrate in pulmonary arteries. These factors may induce smooth muscle cell proliferation and increase pulmonary vascular resistance. The findings of this study suggest that PC-MRI may reflect pulmonary blood flow change induced by smoking, which is useful to quantitatively evaluate and monitor pulmonary blood flow change in smokers. A better quantitative evaluation of the pathophysiological change of pulmonary circulation induced by smoking helps to monitor pulmonary hypertension and guide smoking cessation.

**REFERENCE** [1] Priscilla Henno, Jean-François Boitiaux, et al, Cigarette Smoke-Induced Lung Endothelial and Alveolar Epithelial Injury Tobacco-associated pulmonary vascular dysfunction in smokers: role of the ET-1 pathway, Am J Physiol Lung Cell Mol Physiol 2011;300: 831–839 [2] E. Ferrer, V.I. Peinado, J. Castan, et al, Effects of cigarette smoke and hypoxia on pulmonary circulation in the guinea pig Eur Respir J 2011; 38: 617–627

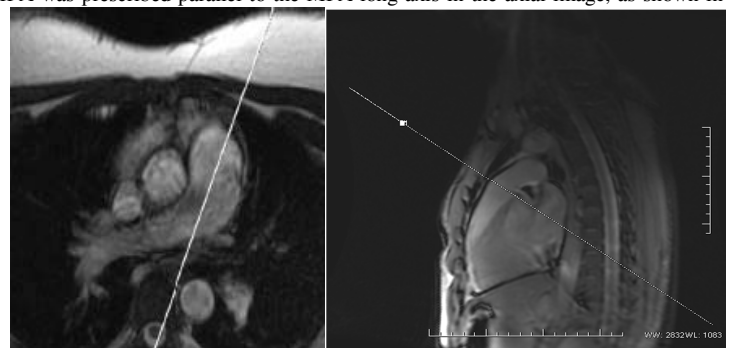


Fig1 The long axis image of MPA was Fig2 An image plane was positioned prescribed parallel to the MPA perpendicular to the long-axis image of MPA, long-axis in the axial image 1.5 cm–2 cm above the level of the pulmonary valve

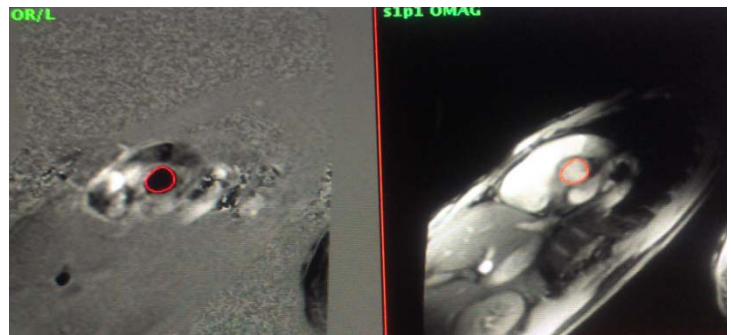


Fig3 and Fig4 MPA phase-map and magnitude images