Viability of Oxygen-enhanced Ventilation Imaging of the Lungs Using Ultra-short Echo Time MRI

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Purpose: To assess the viability of oxygen-enhanced ventilation images using ultra-short echo time magnetic resonance imaging (UTE-MRI).

Methods: We evaluated the oxygen enhancement of the pulmonary T2*, and pulmonary signals in each TE (0.2, 0.8, 1.4, 2.0 ms) in 21 nonsmokers.

Results: The oxygen enhancement of pulmonary signals was the most significant (32%) at the 0.2 ms TE, the second in the pulmonary T2* (~18%).

Conclusions: Pulmonary images using UTE-MRI are useful for ventilation imaging.

Keywords: oxygen-enhancement, ultra-short echo time, pulmonary T2*

Introduction

Oxygen-enhanced pulmonary magnetic resonance imaging (MRI) is widely used for ventilation imaging with few adverse effects.1 Oxygen molecules, which are paramagnetic substances, dissolve into the blood and produce a T1 shortening effect in the lungs.2,3 Most oxygen-enhanced ventilation imaging in MRI uses a half-Fourier acquisition single-shot turbo spin echo (HASTE) sequence.1–3

Another MRI technique, the gradient echo sequence (GRE) with ultra-short echo time (UTE), can depict a pulmonary signal before attenuating and can calculate a T2* value as a quantitative marker of the lungs.4–6 In this study, we assessed whether oxygen-enhanced UTE-MRI would be useful for ventilation imaging.

Materials and Methods

Our institutional ethics committee approved this study. All 21 participants, who were healthy nonsmokers with normal respiratory functions, gave written informed consent. All MR images were obtained with a 1.5T apparatus (Achieva 1.5T, Philips Healthcare, Best, the Netherlands) during quiet breathing in the coronal plane. The sequence was a non-gated three-dimensional GRE with radial sampling, reduction of readout time and a half-sinc RF pulse. We obtained pre- and post-oxygen images in multiple echo times: 0.2, 0.8, 1.4, and 2.0 ms. Fixed parameters were the repetition time, 4.4 ms; flip angle, 10°; field of view, 350 mm; matrix, 128 × 128; slice thickness, 2.73 mm × 128; and bandwidth, 1,446 Hz/pixel, using sensitivity correction. MR signals from the first echo for each echo time were used.

One radiologist (T.S.), with ten years of experience, measured twelve regions of the bilateral lungs for each echo time twice. The mean values were used for analysis. The T2* value was calculated by fitting the signal intensity versus the echo time curve to the following formula:

\[ S(t) = S(0) \exp(-t/T_{2*}) \]  

(1)

The Oxygen enhancement effect (OEE) was calculated using equation (1):

\[ \text{OEE} = \left( \frac{SI_{\text{Post}} - SI_{\text{Pre}}}{SI_{\text{Pre}}} \right) * 100 \]  

(2)

Where SI means the pulmonary signal intensity, Post means post-oxygen-enhancement, and Pre means pre-oxygen-enhancement. In the case of the pulmonary T2*, SI was replaced with the pulmonary T2*. In comparing the pulmonary signals for each echo time and T2* values in pre- and post-oxygen-inhalation, we used a paired t-test. A P value < 0.05 was considered significant.

Results

None developed adverse effects after oxygen inhalation. The pulmonary signals at 0.2 ms and 0.8 ms significantly increased (+32%, +8%, respectively), and pulmonary T2* reduced after...
Fig 1. (A) Oxygen enhancement effects (OEEs) at (A) each echo time and (B) pulmonary $T_2^*$. Pulmonary signals showed exponential ($T_2^*$) decay along echo time in both pre-O$_2$ and post-O$_2$ states. Significant positive OEEs were observed: 32% for 0.2 ms and 8% for 0.8 ms ($P < 0.05$), but there were no significant OEEs for 1.4 ms and 2.0 ms. The slope of the regression line (solid line; $0.771 = 1/1.29$) in the post-O$_2$ inhalation which revealed the reciprocal numbers of the pulmonary $T_2^*$ was steeper than that (dotted line; $0.643 = 1/1.55$) in the pre-O$_2$ inhalation. The steep slope in the post-O$_2$ state indicated shortened pulmonary $T_2^*$ after oxygen inhalation. (B) The pulmonary $T_2^*$ significantly reduced after oxygen inhalation, from 1.55 ms to 1.29 ms ($-18\%$, $P < 0.05$). *$P < 0.05$. NS, not significant.

Fig 2. Lung images for a 42 year-old nonsmoking healthy volunteer: at the shortest echo time of 0.2 ms, (A) pre-oxygen inhalation; (B) post-oxygen inhalation; (C) subtraction of pre- from post-oxygen inhalation image (WL 14000, WW 408000). At the longest echo time of 2.0 ms, (D) pre-oxygen inhalation; (E) post-oxygen inhalation; (F) subtraction of pre- from post-oxygen inhalation image (WL 14000, WW 408000). At an echo time of 0.2 ms, (A) pulmonary signals were observed in the bilateral lungs even before pre-oxygen inhalation, and (B) they were significantly enhanced after oxygen inhalation compared with pre-oxygen inhalation (44%). However, at an echo time of 2.0 msec, pulmonary signals between (D) pre- and (E) post-oxygen inhalation did not significantly change ($-4\%$). (C) At 0.2 ms, an oxygen-enhancement effect was found in the subtraction image, though (F) at 2.0 ms, most pulmonary signals were offset with noise.
oxygen inhalation, from 1.55 ms to 1.29 ms (–18%) \((P < 0.05, \text{paired } t\text{-test})\), although those at 1.4 ms and 2.0 ms did not (Figs. 1 and 2).

Discussion

Oxygen-inhalation UTE-MRI was safely performed on all participants. No adverse effects have been reported following oxygen-inhalation.\(^1\)–\(^3\) The OEE was the most significant at the shortest echo time of 0.2 ms. The shorter echo time emphasizes T1 effect in the GRE sequences. Ventilation imaging is affected by T1 shortening effects depending on ventilation to perfusion ratio, diffusing capacity, and hematocrit.\(^7\)

Pulmonary \(T_2^*\) was also significantly shortened by 18% after oxygen inhalation, which was larger than the about 10% reduction of pulmonary \(T_2^*\) by longer echo times from 1 to 3 ms.\(^7\) The OEE in the pulmonary \(T_2^*\) was smaller than that at the 0.2 ms echo time. To calculate the pulmonary \(T_2^*\), we included pulmonary signals at the longer echo times, 1.4 ms and 2.0 ms, where a lack of significant changes would counterbalance the larger changes at the shorter echo times.

The OEE of the pulmonary \(T_2^*\) might have different ventilation information from the \(T_1\) effect. The OEE of the pulmonary \(T_2^*\) depends on the inhaled gas.\(^7\)–\(^8\) The pulmonary \(T_2^*\) is affected by the air-tissue interfaces of the alveoli, and differences in pulmonary \(T_2^*\) after oxygen-inhalation depend on the susceptibility difference between the gas-tissue interfaces, because differences in lung tissue susceptibility composed of blood are very small.\(^7\)–\(^8\)

Conclusion

Oxygen-enhanced UTE-MRI was safely performed and could be used for ventilation imaging with a short echo time. Pulmonary \(T_2^*\) was also significantly shortened after oxygen inhalation, which might be another quantitative marker after oxygen inhalation.

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Conflicts of Interest

The authors declare that they have no conflicts of interest.

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