ERJ Express. **Published on December 8, 2009 as doi: 10.1183/09031936.00163209**

Lung perfusion and chest wall configuration is altered by glossopharyngeal breathing

Authors:

Leigh M Seccombe[1], Steven CS Chung[1], Christine R Jenkins[1], Clayton J Frater[2], Doug WJ Mackey[2], Mark A Pearson[2], Louise Emmett[2], Matthew J Peters[1].

Departments of Thoracic Medicine[1] and Nuclear Medicine[2] Concord Repatriation General Hospital, Concord, Sydney, New South Wales, Australia.

Corresponding author:

Leigh M Seccombe,

Thoracic Medicine,

Concord Repatriation General Hospital,

Hospital Road, Concord NSW 2139 Australia

leigh.seccombe@sswahs.nsw.gov.au,

Phone: +61 2 97677065

Fax: +61 2 97675090

ABSTRACT:

Glossopharyngeal insufflation is used by competitive breath-hold divers to increase lung gas content above baseline total lung capacity (TLC) to improve performance. Whilst glossopharyngeal insufflation is known to induce hypotension and tachycardia, little is known about the effect on the pulmonary circulation and the structural integrity of the thorax.

Six male breath-hold divers were studied. Exhaled lung volumes were measured before and after glossopharyngeal insufflation. On two study days, subjects were studied in the supine position at baseline TLC and after maximal glossopharyngeal insufflation above TLC. Tc 99^{m} labelled macro aggregated albumin was injected and a computed tomography (CT) of the thorax was performed during breath-hold. Single photon emission CT (SPECT) images determined flow and regional deposition. Registered CT images determined change in the volume of the thorax.

CT and perfusion comparisons were possible in four subjects. Lung perfusion was markedly diminished in areas of expanded lung. 69% of the increase in expired lung volume was via thoracic expansion with a caudal displacement of the diaphragm. One subject who was not proficient at glossopharyngeal insufflation had no change in CT appearance or lung perfusion.

We have demonstrated areas of hyperexpanded, underperfused lung created by glossopharyngeal insufflation above TLC.

Key Words: Glossopharyngeal insufflation, perfusion imaging, pulmonary perfusion, breath-hold diving, hyperinflation

INTRODUCTION

Breath-hold diving, or freediving, is a highly organized, increasingly popular extreme sport. Many competitive breath-hold divers perform glossopharyngeal breathing both as a training exercise and just prior to a dive or submersed breath-hold. Glossopharyngeal breathing, a pump-like action involving the glossopharyngeal structures and larynx that forces air into the airways [1], was originally developed as a therapeutic technique for neuromuscular patients to help expand tidal volume and cough effectiveness [2,3]. The increase in expired lung volume above baseline total lung capacity (TLC) using glossopharyngeal breathing is achieved by a combination of an increase in the Euclidian size of the lung and gas compression [4,5]. Participants refer to this technique as lung packing, however this is described in the literature as glossopharyngeal insufflation (GI) [1,5,8].

In theory, GI above TLC has the potential to assist breath-hold diving performance by increasing available oxygen stores and providing a volume buffer against the compressive effects of hyperbaria. Improvements in both static apnoea duration and breath-hold diving performance have been shown [6]. The potential for adverse cardio-circulatory effects is also clear. The extremely high transpulmonary pressures achieved, of up to 80cmH_2O [5], are associated with tachycardia, hypotension and biventricular systolic dysfunction [7]. In keeping with these observations, adverse neurological symptoms (e.g. pre-syncopal episodes, light-headedness) have been associated with this manoeuvre [7,8] and are seen at the peak of GI rather than after a

long breath-hold suggesting that these are related to a circulatory effect rather than hypoxia or hypercapnia.

In selected individuals, the increase in expired lung volume above baseline TLC using GI can be as much as 3 litres [4,5]. It has been estimated that approximately 30% of the additional entrained air can be attributed to gas compression [4,5] and the remainder an effect of an increase in the Euclidian size of the lung. While some of this increase could be from displacement of structures within the thorax (heart, vessels, oesophagus) most of it may be related to change in the configuration of chest wall and diaphragm. Individual case reports on breath-hold divers have described pulmonary hyperinflation with cardiac compression, aortic stretch and reduction in blood flow using magnetic resonance imaging [9,10].

In normal lung, there are regional differences in the relationship between alveolar ventilation and capillary blood flow. The underperfusion of apical lung areas is related to a direct effect of gravity on blood flow and an indirect effect of gravitational distortion or lung ‘sagging’ in the normal upright lung [11,12]. Zone 1, as described by West [11], has absent capillary perfusion because alveolar pressure exceeds pulmonary arterial pressure. In health these conditions should not exist. If there is a marked increase in alveolar pressure, a significant fall in pulmonary artery pressure or severe distortion of the alveolar capillary bed, Zone 1 conditions could exist during GI above baseline TLC.

By any measure, the potential physiological changes during GI at extremely high lung volumes are significant. In this study, we sought to explore the changes in dynamic and regional lung perfusion that occur under these conditions and also assess using computed tomography (CT) imaging, in a larger number of subjects than had previously been reported, the accompanying volume changes of lung and thorax.

METHODS

Six competitive breath-hold divers who practice GI were recruited. All subjects were non-smokers and did not have any known lung or heart disease.

Lung function

Baseline respiratory function was measured according to ATS/ERS criteria [13,14] (Sensormedics Vmax Encore, Yorba Linda, CA.) with predicted values derived from the recommendations of the ECCS [15]. Exhaled vital capacity (VC) following maximal GI was recorded.

Imaging

Pulmonary perfusion scintigraphy was performed by intravenous injection of 150 MBq of technetium 99^{m} labelled macro aggregated albumin. A hybrid dual-headed gamma

camera (Precedence, Philips, Milpitas, CA.) was used to acquire dynamic (0.3 sec for 400 frames), planar (8 routine views) and single photon emission computed tomography (SPECT) (15 sec at 32 steps per head over an interval of 360°) images of the lungs. The same camera acquired a 6 – slice CT of the thorax.

Subjects underwent measurements at baseline TLC (TLC) and following maximum GI above baseline TLC (TLC_{GI}). These were performed at least 72 hours apart. After cannulation, the subject lay supine on a gantry with arms extended behind their head. Subjects were asked to breath-hold during the dynamic phase of the perfusion imaging (approximately 1 min) at TLC or TLC_{GI} and then immediately following, subjects underwent static and SPECT imaging (approximately 20 mins), where the subject breathed normally in the same position.

A low-dose CT was performed immediately after perfusion scan completion with subjects replicating the same state as in the perfusion imaging. Subjects were asked to breath-hold for the duration of the CT (approximately 80 sec).

All imaging was analysed blind to the subject number, packed or unpacked state and the relative proficiency of subjects in GI.

Processing and display

Images were analysed independently (Astonish, Philips Medical Systems, Milpitas, CA.). CT images were registered for each subject for direct TLC and TLC_{GI} comparison using cross-correlation (Syntegra v2.3.1, Philips Medical Systems, Milpitas, CA.) including lung and skeletal tissue. Lung tissue was segmented from the CT images using ITK-SNAP (University of Pennsylvania, PA.) and subsequent analysis performed using the image processing tool ImageJ (National Institutes of Health, Bethesda, MD).

Imaging interpretation

SPECT: Images were analysed separately by two Nuclear Medicine Physicians and the average of the two scores were taken. The analysis was based on a previously reported digitized model of the anatomy of human lung [16]. Each subsegment was graded as either 0 (normal perfusion), 1 (mild perfusion reduction), 2 (moderate perfusion reduction) or 3 (perfusion absent).

Dynamic views: Time activity curves of the radiotracers were generated with time to maximum and any radiotracer counts in the superior vena cava, right upper, middle and lower lobes and left middle lobe documented.

CT: TLC Euclidian lung volume was subtracted from TLC_{GI} (from the CT registration) for each subject for each CT slice in the transaxial view.

The study was reviewed and approved by the Ethics Review Board of the Sydney South West Area Health Service. Each subject gave written informed consent.

Statistical Analysis

Results are expressed as mean (SD). A two-tailed t-test was used to assess change in physiological parameters between TLC and TLC_{GI}. Inter-observer variability and the comparison of the perfusion intensity scores were performed using a Wilcoxon signed rank test. A *P*-value less than 0.05 was considered statistically significant.

RESULTS

The six study subjects were young and healthy. Their demographic, lung function data and breath-hold diving performance history are displayed in Table 1. Despite attempting GI, subject 6 was unable to increase VC with GI on any of three manoeuvres. As had been planned, this subject proceeded to blinded assessments of perfusion and CT analysis. He functioned as a relevant negative control and his results were therefore excluded from the analysis of the effect of GI.

All other subjects were able to perform manoeuvres adequately and were able to increase measured VC with GI. Mean increase in exhaled VC was 1.4 (0.3) L ($P < 0.001$), equivalent to 20 (3) % increase of baseline VC. CT volume comparisons were

not possible in one subject because scanning his greatly increased lung size would have resulted in radiation limits being exceeded. In relation to analysis of lung perfusion, the dynamic views showed that in one subject, the radiotracers did not move into the pulmonary circulation until he had released his breath-hold following TLC_{GI}. His data was not included in the SPECT analysis. Therefore CT comparisons and perfusion comparisons were each possible in four subjects but the subject sets were different.

Perfusion:

SPECT: There was good agreement between observers in the blinded assessment of the change in perfusion intensity between TLC and TLC_{GI} ($P < 0.72$). In the four subjects that we were able to directly compare, total subsegment scores during TLC_{GI} demonstrated a significant reduction in perfusion intensity from TLC values ($P < 0.02$). There was a reduction in perfusion intensity in anterior ($P < 0.03$) and inferior ($P < 0.01$) segments with no change in posterior, upper and middle segments (Figure 1, 2).

Dynamic views: Time activity curves in five subjects demonstrated an increase in time to initial radiotracer activity in the superior vena cava from 10.1 (1.6) to 16.3 (14.1) seconds from TLC to TLC_{GI} which did not reach significance. The high variability in the TLC_{GI} time activity was largely attributable the subject who did not did not experience radiotracer lodgement in the pulmonary microcirculation until release of TLC_{GI} breath-hold. There were no differences in the time to maximum radiotracer counts in the superior vena cava, right upper, middle or lower left lobes or the right middle lobe.

Computed Tomography:

In four subjects, Euclidian lung volume as measured by this modality increased at TLC_{GI} from TLC by 0.97 (0.3) L ($P < 0.05$). Areas of expansion were similar across subjects as demonstrated in the TLC/TLC_{GI} Euclidian volume subtraction trace for each subject in Figure 3. Thoracic expansion was evident with a caudal displacement of the diaphragm (Figures 3,4). Intercostal bulging of lung tissue and displacement of the vascular mediastinum (Figure 4) was also noted in all subjects. The increase in Euclidian lung volume was 69% of the increase in VC using expired gas volume measurement. Therefore, gas compression was responsible for approximately 31% of the increase in expired lung volume after GI.

DISCUSSION

The study of experienced divers who undertake GI above TLC gives an interesting insight into an area of extreme lung physiology. The increase in lung volume seen in this setting occurs through thoracic expansion and a downward displacement of the diaphragm along with a displacement of the vascular mediastinum. In regions of lung where this marked hyperexpansion occurs there is a reduction in perfusion that approaches absent in some areas.

These subjects had larger than predicted lung size. It is not clear whether this represents a consequence of undertaking GI over a long period or a selection effect that allows competitors with larger lungs than predicted to excel at their chosen sport. By any standard, they were able to increase their expired lung volume impressively. Using CT-based Euclidian volumetric comparisons, we calculate that 69% of the additional expired lung volume was achieved through Euclidian lung expansion. Although we did not directly measure alveolar pressure on this occasion, this is identical to the figure estimated in a previous study from measurement of alveolar pressure and a pneumotachograph [4] and validates the volumetric calculations we conducted here.

The observed features of the lung expansion were that it was achieved through thoracic expansion, a lowering of the diaphragm and displacement of the mediastinum. Reflecting the pressure increase, there was also intercostal bulging of lung tissue. We are confident that our CT registration algorithm was valid and that using anatomical and lung references was appropriate. The consistency of change is reassuring. A previous case report, based on dynamic magnetic resonance imaging, suggested that there was a symmetrical expansion in the thorax [10]. A second case report also demonstrated cardiac and vascular mediastinum configurational changes [9].

Regional differences in lung perfusion can be understood by following West's 3 Zone model which is based on the inequality of blood flow in the lung due to hydrostatic effects [11]. The situation of normal blood flow (Zones 2 and 3) is where pulmonary arterial pressure is greater than alveolar and pulmonary venous pressures. Zone 1

describes a situation where alveolar pressure becomes greater than pulmonary arterial pressure and therefore there is no blood flow. In normal lung, Zone 1 conditions should not exist. The relative lack of perfusion to apical regions in an upright lung is caused not by a gravitational loss of vascular pressure driving perfusion to the apices but by the distortion in the pulmonary capillary bed due to the lung sagging under its own weight within the thorax.

In this study, with patients lying supine, gravitational effects would have a smaller influence on distribution of lung perfusion than in erect subjects. While there clearly was a vertical gradient to some extent, the axis of the reduction in perfusion was more obliquely placed with a greater reduction in basal, anterior areas than there was in anterior areas towards the lung apices. Instead of this being all gravitational, we believe that the re-distribution must be related either to a marked increase in alveolar pressure in all or expanded lung and/or an increase in pulmonary vascular resistance in expanded lung. The latter would be caused by capillary distortion and would be one extreme on the u-shaped curve that normally relates lung volume to pulmonary vascular resistance [17].

Previous studies have shown a transient decrease in lung elastic recoil following GI [4,5,18] which is considered to be a result alveolar stretch associated with repeated and prolonged hyperinflation. Another possible mechanism, as reflected in this data, could be related to hyperinflation-related decrease in pulmonary blood content related to capillary distortion that could in turn increase lung compliance. The effect would be

similar to that seen with exsanguination [19]. The relatively rapid return of compliance to normal, as has been described [18], would be expected as pulmonary capillaries refilled.

One open question is whether the repeated performance of these manoeuvres has a beneficial or deleterious effect on the lung itself. Baseline VC and chest expansion has been shown to significantly increase by 2% and 10% respectively following only 5 weeks GI training in elite female swimmers [20]. We were able to compare static lung function in one subject who participated in an earlier study [4] (Table 2). Over five years, there has been an increase in baseline TLC and VC that may be performance enhancing but at the expense of a reduction in FEV_1/VC ratio. In retrospect, the low ratio at the earlier time of testing may also have been an effect of using this breathing technique in earlier years. The summative effect of these changes on lung performance requires further careful investigation, particularly where this technique is practiced for many years.

As it might relate to competitive performance in near normobaric conditions (static breath-hold and dynamics within a pool setting), these data could be of interest to breath-hold divers. Presumably, the added gas content achieved by compression is equally distributed to all lung segments. However, that which is added to poorly perfused lung would contribute little to systemic oxygenation unless there is effective collateral ventilation during and after GI. Also, it could be predicted that the alveolar pressure of oxygen would fall quite quickly in the perfused lung but remain higher in that

less well perfused. To the extent that gravity and body posture influence lung perfusion, changes in posture during face-immersion breath-hold for example, may lead to greater ‘extraction’ of alveolar oxygen.

There were several methodological and practical challenges associated with our study that are consistent with what is known about this manoeuvre. We discovered that one subject could not successfully use GI to increase his baseline VC and this may occur in a subgroup of competitive breath-hold divers who are uncertain about their competency with this manoeuvre. We also observed syncope in one subject during two separate occasions while attempting TLC_{GI} which has been previously reported [7,8]. Two subjects were studied while breathing tidally for their TLC perfusion scans, however due to hydrostatic similarities between TLC and tidal breathing, we did not believe this to be a limitation.

The subject that was unable to effectively perform GI served as a very useful negative control. There was no change in perfusion intensity or lung shape and the calculated difference in lung volumes between study days was 10mL. In a frank discussion subsequent to his participation, he admitted that he found no performance benefit from GI and no longer used this manoeuvre in competition. One subject was lost from each of the CT and perfusion comparisons. Whilst we could have studied a greater number of subjects, the consistent pattern evident is such that additional information from more subjects would be unlikely to change our conclusions and the radiation exposure could not be justified. Further studies on these subjects, to determine reproducibility of

findings, were not possible because research radiation limits would have been exceeded.

In summary, dynamic pressure changes in the chest during GI expand lung areas that are aerated but poorly perfused. This primarily affected anterior and inferior regions as the subjects lay supine. This may be related to increased alveolar pressure, pulmonary capillary pressure decreases or a combination of the two. GI caused thoracic hyperinflation in a pattern that was consistent between subjects.

ACKNOWLEDGMENTS

We acknowledge “Sydney Freedivers” for their support and enthusiasm for our research and their assistance in recruiting subjects. We are also grateful to Dr Roberto Russo, Dr Lloyd Ridley and Peter Rogers for their assistance with data analysis.

None of the authors have a conflict of interest to declare. This data has not been submitted anywhere else for review or publication. All authors are fully aware of the content and have approved this submission.

REFERENCES

1. Lindholm P, Norris CM Jr., Braver JM, Jacobson F, Ferrigno M. A fluoroscopic and laryngoscopic study of glossopharyngeal insufflation and exsufflation. *Respir Physiol Neurobiol* 2009; 167: 189-194.

2. Dail CW, Affeldt JE, Collier CR. Clinical aspects of glossopharyngeal breathing. *JAMA* 1955; 158: 445-449.

3. Feigelson CI, Dickinson DG, Talner NS, Wilson JL. Glossopharyngeal breathing as an aid to the coughing mechanism in the patient with chronic poliomyelitis in a respirator. *N Engl J Med* 1956; 254: 611-613.

4. Seccombe LM, Rogers PG, Mai N, Wong CK, Kritharides L, Jenkins CR. Features of glossopharyngeal breathing in breath-hold divers. *J Appl Physiol* 2006; 101: 799-801.

5. Loring SH, O'Donnell CR, Butler JP, Lindholm P, Jacobson F, Ferrigno M. Transpulmonary pressures and lung mechanics with glossopharyngeal insufflation and exsufflation beyond normal lung volumes in competitive breath-hold divers. *J Appl Physiol* 2007; 102: 841-846.

6. Overgaard K, Friis S, Pedersen RB, Lykkeboe G. Influence of lung volume, glossopharyngeal inhalation and $P_{ET}O_2$ and $P_{ET}CO_2$ on apnea performance in trained breath-hold divers. *Eur J Appl Physiol* 2006; 97: 158-164.

7. Potkin R, Cheng V, Siegel R. Effects of glossopharyngeal insufflation on cardiac function: an echocardiographic study in elite breath-hold divers. *J Appl Physiol* 2007; 103: 823-827.

8. Novalija J, Lindholm P, Loring SH, Diaz E, Fox JA, Ferrigno M. Cardiovascular aspects of glossopharyngeal insufflation and exsufflation. *UHM* 2007; 34: 415-423.

9. Lindholm P, Nyŕen S. Studies on inspiratory and expiratory glossopharyngeal breathing in breath-hold divers employing magnetic resonance imaging and spirometry. *Eur J Appl Physiol* 2005; 94: 646-651.

10. Eichinger M, Walterspacher S, Scholz T, Tetzlaff K, Röcker K, Muth C-M, Puderbach M, Kauczor H-U, Sorichter S. Lung hyperinflation: foe or friend? *Eur Respir J* 2008; 32: 1113-1116.

11. West JB. Regional differences in the lung. *Chest* 1978; 74: 426-437.

12. Arai TJ, Henderson AC, Dubowitz DJ, Levin DL, Friedman PJ, Buxton RB, Prisk GK, Hopkins SR. Hypoxic pulmonary vasoconstriction does not contribute to pulmonary

blood flow heterogeneity in normoxia in normal supine humans. *J Appl Physiol* 2009; 106:1057-1064.

13. ATS/ERS Task Force: Standardisation of lung function testing. Standardisation of spirometry. *Eur Respir J* 2005; 26: 319-338.

14. ATS/ERS Task Force: Standardisation of lung function testing. Standardisation of the measurement of lung volumes. *Eur Respir J* 2005; 26: 511-522.

15. European Community for Coal and Steel. Standardized lung function testing. *Bull Eur Physiopathol Respir* 1983; 19, Suppl 5: 7-21.

16. Magnussen JS, Chicco P, Palmer AW, Van der Wall H, Vu DH. Creation of a three-dimensional model of human segmental lung anatomy. *Am J Roentgenol* 2000; 174: 1333-1336.

17. West JB. Measurement of pulmonary blood flow. *In:* Kelly PJ ed. Respiratory physiology, the essentials. 6th Edn. Lippincott Williams and Wilkins, Baltimore, MD, 2000; p. 35.

18. Tetzlaff K, Scholz T, Walterspacher S, Muth CM, Metzger J, Roecker K, Sorichter S. Characteristics of the respiratory mechanical and muscle function of competitive breath-hold divers. *Eur J Appl Physiol* 2008; 103: 469-475.

19. Cotes JE, Chinn DJ, Miller MR. Lung and chest wall elasticity. *In:* Lung function. 6th Edn. Blackwell Publishing, Malden, MA, 2006; p.123.

20. Nygren-Bonnier M, Gullstrand L, Klefbeck B, Lindolm P. Effects of glossopharyngeal pistoning for lung insufflation in elite swimmers. *Med Sci Sports Exerc* 2007; 39: 836-841.

TABLES

TABLE 1: Baseline lung function and diving performance

in six male breath-hold divers

	Mean (SD)	Range
Age, yr	30 (7)	20-36
Height, cm	181 (5)	174-190
BMI	25.0 (2.0)	22.5-27.4
FEV_1,% pred	119 (13)	99-132
FVC, L	6.79 (0.99)	5.67-8.62
FVC,% pred	129 (16)	115-158
TLC, L	8.42 (1.25)	6.81-10.60
TLC,% pred	114 (15)	97-139
PB static breath-hold (min)	6:33 (1:02)	5:30-8:00
PB dynamic distance (m)	168 (35)	135-223

BMI: body mass index; FEV_1: forced expiratory volume in 1 second; FVC: forced vital capacity; TLC: total lung capacity; PB: personal best; dynamic distance: apnoeic distance in a 50m swimming pool.

TABLE 2: Baseline lung function in Subject 1 as

compared to previous measurements in 2004 [4].

	2009	2004
Age, yr	30	25
Height, cm	184	186
Mass, kg	89	90
FEV_1, L	5.87	6.20
FEV_1,% pred	129	130
FVC, L	8.62	8.11
FVC,% pred	158	142
FEV_1/FVC, %	68	76
TLC, L	10.60	10.08
TLC,% pred	139	130

FEV_1: forced expiratory volume in 1 second; FVC: forced vital capacity; TLC: total lung capacity.

FIGURE LEGENDS

Figure 1. Mean reduction in perfusion intensity from baseline to maximum glossopharyngeal insufflation above total lung capacity for each lung subsegment in four breath-hold divers. White denotes a less than 5% reduction, light grey a 6-15% reduction and dark grey a 16-35% reduction. Subsegment number as marked per Magnussen model [16].

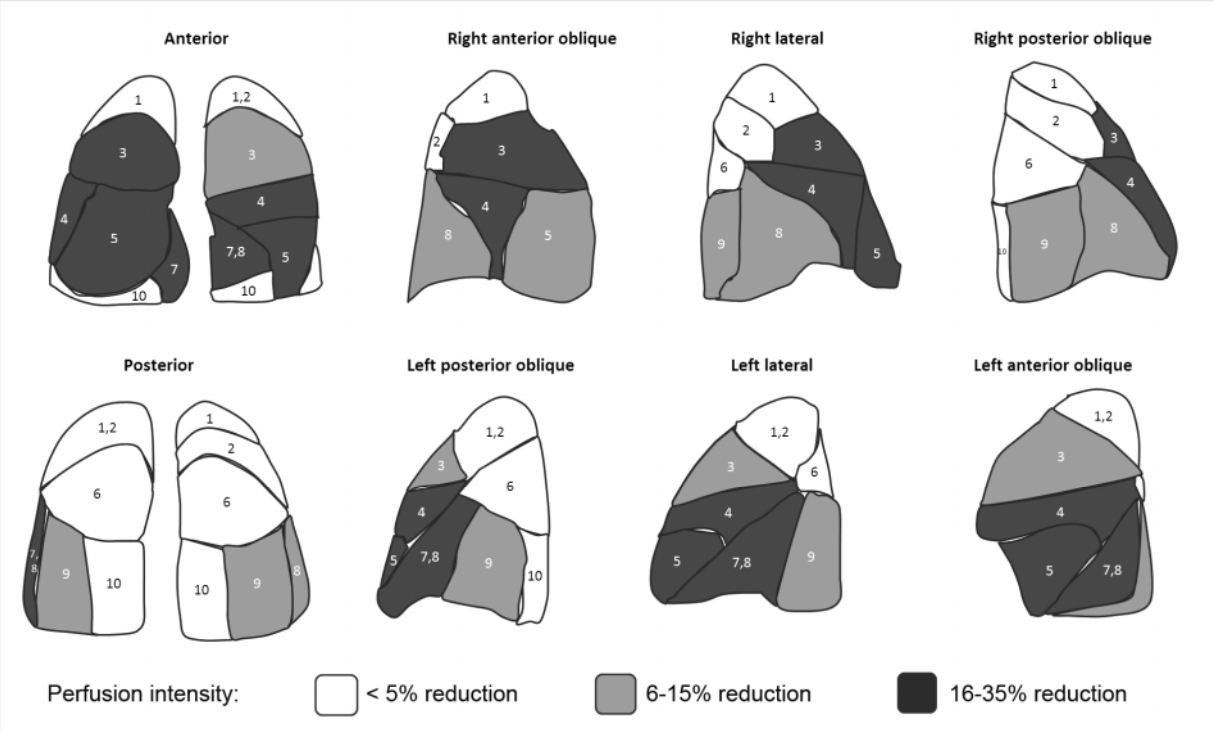


Figure 2. Single photon emission computed tomography image of Subject 1 in the supine position at baseline and at maximal glossopharyngeal insufflation above total lung capacity (TLC_{GI}) in the coronal (posterior), sagittal (right lateral) and axial views.

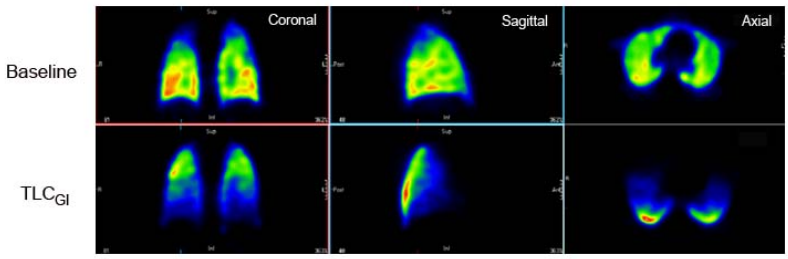


Figure 3. Lung volume per CT slice in the transaxial zone for four breath-hold divers during maximal glossopharyngeal insufflation above total lung capacity (TLC_{GI}) and at baseline total lung capacity (TLC). Solid line is the subtraction of the two.

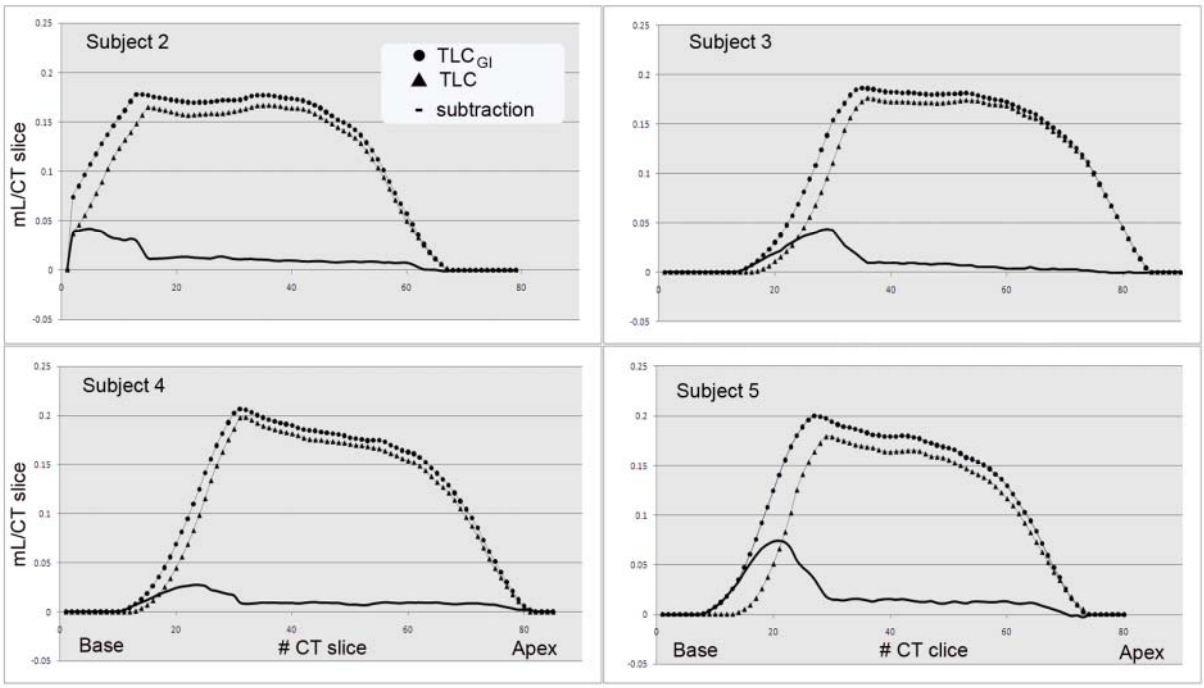
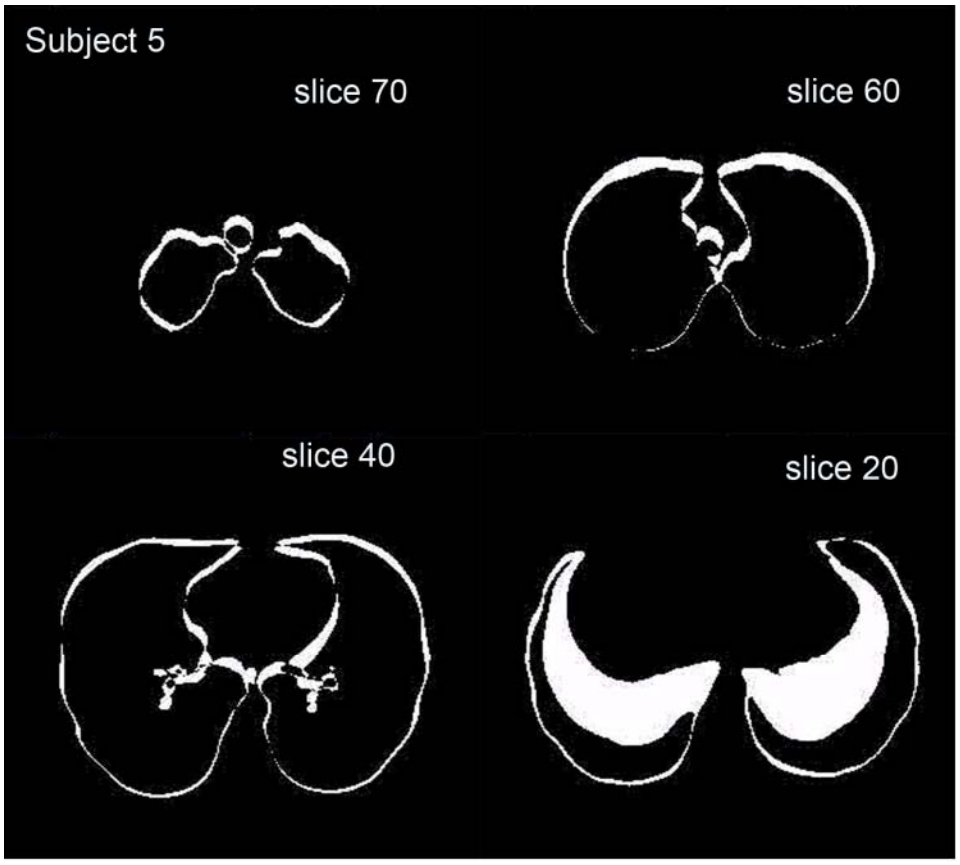


Figure 4. Lung volume difference (white areas) between registered CT scans performed at maximal glossopharyngeal insufflation above TLC and baseline TLC in Subject 5. CT slices displayed are 70 (near apex), 60, 40 and 20 (near base) in the transaxial view.


Subject 5
slice 70
slice 60
slice 40
slice 20