Pulmonary edema and hemoptysis after breath-hold diving at residual volume

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Lindholm P, Ekbom A, Öberg D, Gennser M. Pulmonary edema and hemoptysis after breath-hold diving at residual volume. J Appl Physiol 104: 912–917, 2008. First published January 17, 2008; doi:10.1152/japplphysiol.01127.2007.—To simulate pressure effects and experience thoracic compression while breath-hold diving in a relatively safe environment, competitive breath-hold divers exhale to residual volume before diving in a swimming pool, thus compressing the chest even at depth of only 3–6 m. The study was undertaken to investigate whether such diving could cause pulmonary edema and hemoptysis. Eleven volunteer breath-hold divers who regularly dive on full exhalation performed repeated dives to 6 m during a 20-min period. The subjects were studied with dynamic spirometry, videofibersolaryngoscopy, and single-breath diffusion capacity of carbon monoxide (DLCO). The duration of dives with empty lungs ranged from 30 to 120 s. Postdiving forced vital capacity (FVC) was reduced from mean (SD) 6.57 ± 0.88 to 6.23 ± 1.02 liters (P < 0.05), and forced expiratory volume during the first second (FEV1.0) was reduced from mean (SD) 5.09 ± 0.64 to 4.59 ± 0.72 liters (P < 0.001) (n = 11). FEV1.0/FVC was 0.78 ± 0.05 prediving and 0.74 ± 0.05 postdiving (P < 0.001) (n = 11). All subjects reported a (reversible) change in their voice after diving, irritation, and slight congestion in the larynx. Fresh blood that originated from somewhere below the vocal cords was found by laryngoscopy in two subjects. DLCO/VA was 1.56 ± 0.17 mmol kPa−1 min−1 l−1 before diving. After diving, the DLCO/VA increased to 1.72 ± 0.24 (P = 0.001), but 20 min later it was indistinguishable from the predive value: 1.57 ± 0.20 (n = 11). Breath-hold diving with empty lungs to shallow depths can induce hemoptysis in healthy subjects. Edema was possibly present in the lower airways, as suggested by reduced dynamic spirometry.

Today an increasing number of breath-hold divers compete for depth in the sport of apnea (13). Among these divers, there are reports of symptoms suggestive of pulmonary edema after deep dives (5) with some dives resulting in the need for in-hospital oxygen treatment (personal communication from three divers to P. Lindholm). There are also reports of hemoptysis after breath-hold diving (4, 10), and in one study, pulmonary edema was confirmed with chest x-ray after dives to 30 m in the sea (21). There are also anecdotal reports of minor hemoptysis, with small streaks of blood in the sputum after breath-hold diving without any other symptoms suggestive of pulmonary edema (14). In general, minor pulmonary edema usually presents with dyspnea, desaturation, and fatigue, with more serious cases presenting with hemoptysis, and pink frothy expectorations (11, 24). Pulmonary edema of immersion or swimming-induced pulmonary edema has been reported in connection with both surface swimming (1) and SCUBA diving (11).

During deep breath-hold diving, the gas in the lungs is compressed according to Boyle’s law. The divers usually start diving after a full inhalation to total lung capacity (TLC), sometimes assisted by glossopharyngeal insufflation (GI) to add extra gas volume (14, 17). At some depth during the descent, the compression of the inhaled gas will cause the lungs to shrink to a volume equal to their residual volume. It should be noted that the residual volume measured dry is higher than the residual volume when immersed, due to the immersion, which causes central pooling of blood, enabling the lung and chest to be compressed to a lower volume (higher pressure) than can be calculated from dry measurements (6, 23). Theoretically if a diver descends below this “squeeze” depth, there will be an increased strain on the pulmonary capillaries; the pressure difference between the blood pressure in the pulmonary capillaries and the intra-alveolar gas pressure may cause stress failure (25) with leakage of fluid and blood into the lungs, similar to hydrostatic or cardiogenic pulmonary edema (24).

Many competitive divers practice the “thoracic squeeze” effect by exhaling before diving. To simulate pressure effects and experience thoracic compression in a safer environment, divers use glossopharyngeal exsufflation (GE) (14, 17) to further empty the lungs before shallow diving in a swimming pool. This is a method by which the muscles of the glossopharynx are used to draw air out of the lungs and into the mouth. If this maneuver is combined with closing of the vocal cords while expelling the mouthful of air, and repeated a number of times, volumes of around 0.2–0.4 liter may be extracted from the lungs, yielding an effective volume of air below residual volume (RVGE). Subsequent breath-hold diving will compress the chest substantially even if the depth is only around 3–6 m. Divers sometimes use this type of diving for short warm-up dives and sometimes as a training tool to enable training in the safety and comfort of a swimming pool instead of deep sea water. Reports from divers practicing “empty lung diving” include descriptions of feeling of throat congestion, a slightly fluidic cough, minor hemoptysis, and suggestion that the voice may be affected for a few minutes after such dives. The strain on the chest can be felt in the sternal/rib joints with lingering articular soreness for 1–2 days.

We hypothesized that breath-hold diving after exsufflation could cause fluid filtration into the air spaces and also edema formation in the conductive airways. Such an edema would be similar to cardiogenic asthma, reducing dynamic spirometry. Thoracic squeeze has been suggested to cause hemoptysis in deep breath-hold diving. However, both the anatomic origin of the blood and the cause of the hemoptysis is unclear. It was...
therefore considered of interest to perform a study on subjects exposing themselves to pulmonary squeeze.

METHODS

Subjects

The 11 healthy male volunteers were 29 ± 5 (21–35) yr old, weighed 78 ± 8 (68–89) kg, and were 184 ± 5 (176–191) cm tall (values shown as means ± SD, and range). All of the subjects were experienced breath-hold divers. The experimental procedure was conformed in conformity with the principles of the Declaration of Helsinki and had been approved by the Ethics Committee of Karolinska Institutet. All subjects gave their written informed consent before participation. To enable an ethical method to do such a study we only studied divers regularly performing these maneuvers and used rigorous safety procedures with proper medical backup standing by. We believe that the methods used to study the divers pre- and postdiving did not further increase the risk of such diving.

Experimental Procedures

Breath-hold diving protocol. The dives were carried out in a 6-m-deep circular pool with a ladder to the bottom. The water temperature was 35.5 ± 0.5°C. Subjects were asked to do repeated “empty lung diving” according to their own judgment and training routine for 20 min. Each diver was closely followed at all times by a trained safety diver (also breath holding but not diving on empty lungs). The safety diver was within arms reach and in visual contact with the subject during both descent and ascent, as well as monitoring the diver after reaching the surface. No attempt was made by the investigators to interfere with this diving schedule.

Empty lung diving. Before diving, the divers exhaled completely to residual volume and then performed glossopharyngeal exsufflation (14, 17) to further reduce the volume of air in the lungs. The participants dived repetitively according to their own preference.

Measurements. Before diving, the subjects’ static lung volumes were determined with nitrogen washout (O2 breathing). Thereafter, single-breath diffusion capacity for carbon monoxide (DLCO) was measured (18). Dynamic spirometry was performed at least three times; thereafter the upper airways of the subjects were investigated and photographed via nasofiberscopy.

Once these measurements were made, the subjects entered the water. After 20 min of diving, the subject exited the water, dried off quickly (30 s), and dressed in a bath robe. Thereafter DLCO was remeasured. The diver then returned to the pool for one more dive, quickly (30 s), and dressed in a bath robe. Thereafter DLCO was performed.

Lung function tests were carried out according to European Respiratory Society/American Thoracic Society standards (2, 18, 20, 22) using the Sensormedics Vmax pulmonary system (SensorMedics Vmax229 Encore, VIASYS Healthcare Respiratory Technologies, Yorba Linda, CA). All tests and calibration procedures conformed with the specifications set by Senormedics software. Due to the constrained testing protocol, we did not measure a resting arterial oxygen saturation (SaO2) postdiving. (Subjects were either diving, performing lung function testing, or being investigated with laryngoscopy.) A previous pilot study did not show any desaturation a few minutes after breath-hold diving, using a similar dive protocol.

The larynx was examined in all divers immediately after the lung function tests had been performed. The nose was anesthetized in the conventional way using a nasal spray containing 34 mg/ml lidocaine hydrochloride, 0.17 mg/ml oximethazoliumhydrochloride, and Metagin (preservative). All patients were examined before and after the dive with a conventional fibernasolaryngoscope transnasally (Olympus ENF type 4) connected to a cold light supply (Olympus ILK 3). All subjects were also examined transorally with a 70° Hopkins rod laryngoscope (Karl Storz) connected to the same cold light supply. Rod laryngoscopes generally provide better optical quality, but due to anatomic differences, all regions of the larynx could not be visualized by this method in all subjects. Images were recorded using a 3 CCD camera unit (Panasonic GP-US-502) transferred by S-video connection to a video recorder (Sony DCR PC 100) and digitized to JPEG format for storage and later study.

Subjective voice changes, feelings of secretion on vocal cords, needs to clear the throat, blood taste, and other subjective symptoms noted by the subjects were estimated subjectively and recorded in the written experimental record.

Hemoglobin concentrations in capillary blood were measured with a B-Hemoglobin analyzer with an accuracy of ± 1.5% (HemoCue AB, Angelholm, Sweden), results being given in grams per deciliter. Tests were done in duplicate and averaged both pre- and postdiving. Tests were carried out while subjects rested between dynamic spirometry maneuvers, with the subjects sitting.

Statistical Analyses

t-tests for dependent samples were used for pre- and post-data testing. The data are presented as means ± SD, and differences with a P value of <0.05 were considered significant. An ANOVA test with repeated measures (Statistica, Statsoft, Tulsa, OK) was used for those measurements that involved more than two conditions. For post hoc comparisons, the Fisher least significant difference test was used.

RESULTS

All divers reached the bottom of the pool when diving. Some divers remained upright on their knees with their chest at ~5-m depth, while others lay down on the bottom and so effectively reached 6-m depth for their lungs. Divers were encouraged to perform multiple short dives so not to risk hypoxia from breath-holding nor hypoxia of ascent. Most dives lasted 30 s but dive times of up to 2 min were recorded. (The boundaries of the ethics approval was not to interfere with the divers own procedures; thus we did not limit dive time.) There were no incidents of loss of consciousness or loss of motor control due to hypoxia (13) during the experiment. A few subjects showed objective signs of minor hypoxia, such as lip cyanosis, after surfacing.

Spirometry-Static Lung Volume

Nitrogen washout from functional residual capacity (FRC) gave TLC of 8.23 ± 1.21 liters (n = 9) (highest TLC of 2 measurements), with a corresponding RV of 1.33 ± 0.46 liters and VC of 6.89 ± 0.94 liters. The vital capacity (VC) was 0.18 ± 0.18 liters higher than forced vital capacity (FVC) (prediving) (n = 9, P = 0.02). Nitrogen washout and slow VC was not tested after diving. The predicted value for TLC in this group was 7.7 ± 0.3 liters, and the predicted RV was 1.8 ± 0.1 liters (22).

Dynamic spirometry was tested to achieve three technically acceptable measurements pre- and postdiving. Data were averaged for all three measurements. Postdiving FVC decreased 5.4 ± 7.1% (n = 11, P = <0.05) and forced expiratory volume during the first seconted (FEV1.0) decreased 10.0 ± 7.0% (n = 11, P < 0.01). (For absolute values and FEV1.0/FVC, see Table 1.) Prediving FVC was 120 ± 14% of predicted values (22) (n = 11) (range 105–146%).

DLCO/alveolar ventilation (VA) was 1.56 ± 0.17 mmol·kPa⁻¹·min⁻¹·l⁻¹ before diving. After diving, the
DLCO/V\(_A\) increased to 1.72 ± 0.24 mmol·kPa\(^{-1}\)·min\(^{-1}\)·l\(^{-1}\), and then 20 min later it had decreased to 1.57 ± 0.20 mmol·kPa\(^{-1}\)·min\(^{-1}\)·l\(^{-1}\) (ANOVA \(P = 0.001, n = 11\)). Post hoc testing showed significant differences between postdiving data and both predive and recovery data (\(P = 0.001\)). There was no significant difference between predive and recovery values. There were no significant changes in inspiratory vital capacity (IVC) or V\(_A\) between conditions.

Hemoglobin concentration was 15.4 ± 2 g/dl before diving and 16.2 ± 1.0 g/dl postdiving (\(n = 8, P = 0.07\)). Recovery values of Hb were not measured. Our subjects’ Hb were higher than the “normal” Hb for DLCO calculations [14.6 g/dl (18)]. If adjusted (adj) DLCO is calculated for the pre- and postdiving data in the eight subjects who had their Hb measured, taking account of the Hb values [\(\text{DLCO}/\text{V}_{A\text{adj}} = \text{DLCO}/\text{V}_A/[1.7 \text{ Hb/(10.22 + Hb)}]\)], then there was a nonsignificant increase from prediving DLCO/V\(_{A\text{adj}}\) of 1.55 ± 0.19 mmol·kPa\(^{-1}\)·min\(^{-1}\)·l\(^{-1}\) to the postdiving values of 1.65 ± 0.24 mmol·kPa\(^{-1}\)·min\(^{-1}\)·l\(^{-1}\) (\(n = 8\) \((P = 0.12)\) (t-test).

**Laryngoscopy**

After diving, fresh blood was found by laryngoscopy in two subjects (Figs. 1 and 2). This blood originated from somewhere below the vocal cords and was documented by digital camera. A more subtle finding was a short-lasting blood congestion in the midline on the laryngeal side of the epiglottis, possibly caused by squeeze or direct mechanical irritation from GE. One of these two subjects also had an acute cough while exiting the pool. Five subjects showed increased redness (clinical judgment) after diving, and two showed subglottal swelling, while excess mucus was apparent in four divers. All subjects had a mild change in their voice after 20 min of diving (subjective). No subject showed any bleeding from upper respiratory tract, sinuses, or nose.

**DISCUSSION**

The principal finding of the present study was that diving that involved thoracic squeeze resulted in reduced dynamic spirometric values. Also, in two divers, fresh blood, whose origins could not be traced to the upper airways or sinuses, was found on the glottis. Finally, nonadjusted diffusion capacity was increased directly after the dives.

**Empty Lung Diving and Thoracic Squeeze**

To simulate the thoracic squeeze effects of deep diving in the ocean, competitive breath-hold divers have invented a more convenient training method: they dive in shallow water, often in a swimming pool after complete exhalation to RV. This method is similar to the study on a single subject reported by Craig (6), except that the present-day breath-hold divers dive on air, not after preparatory breathing of 100% oxygen. Craig showed that when diving to 4.75-m depth after exhalation to residual volume, there was no change in esophageal pressure, indicating a blood shift into the thorax of 0.6 liter. Many of the
divers in the present study further reduced the amount of air in the lungs by GE before diving (12, 14, 17). We did not measure the volume of GE in the divers who used it in the present study, and neither did we measure the effective volume of gas in the lungs when the divers surfaced (the duration of the dives would consume oxygen from the pulmonary stores, further reducing the gas volume) since we did not want to interfere with the practice of this diving method for reasons of safety and ethics. The protocol approved by the ethics committee was based on the fact that these divers practiced diving on empty lungs as part of their usual training procedures and that our measurements would not interfere or generate additional risk. From personal experience and a few pilot experiments, we designed the 20-min time and 6-m depth to cause a moderate effect. We did not expect to find hemoptysis occurring within this protocol, but since hemoptysis/bleeding was confirmed by laryngoscopy it is possible that minor bleeding happens during such training even though it may not be noted by the divers. Divers practice these maneuvers and dives to increase their chest flexibility and their tolerance to compression (not confirmed experimentally). In fact, the divers’ RV, as measured by standard spirometry (N2 washout), averaged 27% lower than predicted, while TLC was slightly elevated (107% of predicted value). It can be speculated that the divers’ practice has given them greater thoracic flexibility or ability to exhale a larger proportion of their lung capacity. Commercial divers (using breathing apparatuses) have been shown to have larger lungs and lower FEV1/FVC than predicted from a normal population, yet are still considered healthy (7) (similar to our findings of FEV1/FVC of 0.78 prediving).

The compression of the thorax and lungs in our experimental dives can be correlated to deep diving after inhalation to full lung volume by calculation of the “equivalent squeeze depth” (ESD), i.e., the theoretical depth at sea where a similar compression would be experienced as would be seen during exhalation/exsufflation and diving in shallow water:

\[
ESD = 10 \times \left\{ TLC \times \left[1 + (\text{depth}/10)\right]/(RV - GE) - 1 \right\}
\]

where ESD/10 + 1 = pressure at depth in atmospheres absolute, and volume of lung at depth = \(RV_{\text{surface}}/(1 + \text{depth}/10)\) with depth in meters. If we calculate ESD from our subjects’ dry TLC and RV values (without GE), this would correspond to an ESD of 91 ± 30 m (mean ± SD) (n = 9) when diving to 5-m depth after exhalation to RV (not RVGE). This calculation however, does not include gas uptake from the lung due to metabolism (oxygen) and increased pressure (nitrogen) during diving in the ocean; such gas consumption from the lung would reduce actual ESD.

Edema and Dynamic Spirometry

The theoretical basis for our hypothesis was that during diving on empty lungs, the residual gas volume in the lungs will be affected by the external pressure and the volume will be compressed according to Boyle’s law.

When the middle ear is exposed to increased ambient pressure without proper pressure equilibration, an increased volume of the mucosal lining has been measured (3). The change in volume of the inner ear mucosal lining was shown to be directly related to the change in ambient pressure (3). In the middle ear the pressure effect on the mucosa depends on the state of the eustachian tube, the tympanic membrane, the enclosed gas volume, and the mucosal compliance. Similarly, the effect of the mucosal lining in the airways would be dependent on the enclosed gas volume in the airways, and the compliance of the mucosal membranes.

During this type of diving, it is possible that a relatively lower pressure will develop in the airways with respect to the ambient pressure. This would cause a larger filtration pressure in the mucosal membranes lining, e.g., the small airways (bronchioles). A fluid extravasation causing an interstitial edema in this region would induce swelling and narrowing of both bronchi and bronchioli. A narrowing of conductive airways would affect airway conductivity and thus reduce forced expiratory flow. A more severe narrowing could induce clinical obstructive effects, and a parallel could be argued to cardiac asthma where a blood and fluid overload (due to cardiac failure, not lung underpressure) causes edema in the lungs with resulting obstructive signs. In the present experiments, the FVC decreased, but the decrease in FEV1.0 was greater, causing a significant fall in FEV1.0/FVC, although there was no significant changes in the static spirometry (IVC). These results present indirect support for our hypothesis that an interstitial edema was formed in the conductive airways making the subjects’ more obstructive postdiving.

Apnea for 15 s with cold water face immersion has been shown to decrease forced expiratory flow by 17% immediately after apnea in a group of five subjects responding to cold water face immersion (19). The suggested mechanism for the diving response-induced bronchoconstriction would be vagally mediated (cholinergic), and it was blocked by ipratropium. Dry and warm breath-holds created a lesser degree of bronchoconstriction, which was not affected by ipratropium. Also 1 min of cold showers to the trunk has been shown to reduce large airway conductance by 21% (9). To avoid the effects of hypothermia, cold face immersion, and peripheral vasconstriction, our subjects dived in thermoneutral water. The vagal effect of a diving response (as shown on the heart) is immediately reversed when breathing commences. Thus a lingering effect would most likely not have been the case when our subjects performed the dynamic spirometry (during a time frame of about 1–4 min postdiving).

Pulmonary squeeze as a consequence of thoracic compression below RV has long been recognized as a risk with deep breath-hold diving. There have been case reports, mainly of hemoptysis and pulmonary edema as suggested by computer tomography in a few cases and in fewer still by bronchoscopy with bronchoalveolar lavage some time after admission to hospital (4, 10). Bleeding from the upper airways after diving could theoretically come from several anatomic locations. For example, a small hemorrhage from the paranasal sinuses into the posterior nasopharynx could manifest as expectorations of bloody mucus without overt nosebleed. In our two cases where bleeding was observed, however, the source of the bleeding was conclusively shown to have an origin below the vocal cords, and none of the subjects had any bleeding evident in the nasal cavities. It is possible that it originated in the alveoli where the barrier between blood and airway is at its thinnest, as has been previously postulated. The cause of bleeding would then likely be caused by the direct pressure differential across the alveolar membrane. Another possibility could be that the
bleeding occurs in some of the larger airways as a consequence of mechanical shearing forces caused by the compression. MRI studies have been performed in subjects performing GE starting at RV (14). Although bleeding was not observed in these subjects, it was clearly obvious that there were significant forces applied to the larger airways. For example, the posterior wall of the trachea was seen to invaginate into the tracheal lumen. Mechanical shear caused by voluntary diaphragmatic contractions has been suggested as a cause of hemoptysis by Kiyon et al. (10). Other complaints associated with the GE and noted among the divers such as the need to clear the throat, a feeling of change in the voice and the need to cough associated with the findings of increased redness in the epiglottis and subglottal swelling, could possibly be explained by a similar mechanism, i.e., edema and blood congestion due to pressure differences in the incompressible regions of the larynx. An alternate explanation is that the mechanical stress in the larynx is not caused by the pressure differences but by direct mechanical stress during GE when the glottis and larynx is used as a valve during the exhalation. In such cases these findings would be particular to the practice of GE in the training situation and not present during “real” dives.

There are three possible outcomes of an increased ambient pressure: collapse of parts of the lung with atelectasis, hydrostatic pressure leading to fluid filtration into the alveolar space, and, third, rupture with bleeding and blood filling the void spaces.

The compression experienced in the experimental dives for a subject with a residual gas volume at the surface of, for example, 1.5 liters would be 0.5 liter at 5-m depth. If that volume in full should be countered by fluid filling of the lung, it would cause a massive edema with a severe restrictive reduction in lung volume. A more likely outcome is that part of this lowered lung volume is achieved by atelectases. The transpulmonary capillary pressure normally associated with capillary failure can be as low as 24 mmHg, with consistent failure occurring at pressures of 40 mmHg (25), but these figures are relevant to the transmembrane pressure difference between a capillary and an air-filled alveoli. If atelectasis occurs, the capillaries in these areas would be supported by other structures limiting the pressure gradient and fluid extravasation (i.e., no alveolar space for the fluid to go to).

In this study the DLCO measured directly after the dives showed a slight increase in diffusion capacity. This could indicate that no interstitial edema had been formed during these dives. However, the DLCO can theoretically be divided into several resistance elements in series and described thus:

$$1/D_l = 1/D_m + 1/V_\phi$$

where $D_l$ is the measured diffusion capacity, $D_m$ is the diffusion capacity of the alveolar membranes and other structures between the alveolar air and the red blood cells, $V$ is the amount of red blood cells available in the pulmonary capillary bed, and $\phi$ is the carbon monoxide conductance of red blood cells, i.e., the rate of uptake per volume blood. As can be seen from this equation, the measured increase in DLCO after the dives could, in principle, be due to two opposing influences. The pulmonary squeeze may have caused a decrease in membrane conductance ($D_m$) due to fluid filtration, but this effect may have been offset by a large increase in pulmonary capillary blood volume or extravasation of blood into the alveolar space. Free blood in the alveolar space would bind CO, and it has been shown that the largest increase in $D_l$ (50% increase in the transfer factor) after free diving occurred in a subject who developed pulmonary edema (21).

Another factor that may affect the DLCO is the blood concentration of hemoglobin (Hb). In the present study Hb increased by 0.8g/dl between pre- and postdiving. This difference was not statistically significant, but the possibility remains that part of the increase in DLCO postdiving was caused by a hemoconcentration due to immersion/breath holding (8). Calculation of Hb-adjusted DLCO/V˙A showed that although the Hb increase accounted for part of the increase in DLCO, this did not explain the whole difference, although the results were no longer statistically significant.

The fact that the DLCO in the present study returned to predive values within 20 min postdive seems to preclude any major extravasation of blood but would be in line with the time course of cardiovascular changes postimmersion (15, 16).

Since DLCO decreases with increasing alveolar PO2 due to the competitive binding to Hb between oxygen and carbon monoxide, the diffusion effects can be partitioned by measuring DLCO at different alveolar oxygen concentrations. In principle, it would have been possible to determine whether the measured changes in DLCO were due only to increased red cell conductances. Unfortunately, the present experimental schedule did not allow for more DLCO measurements, within the short time period required. However, future studies should center on describing the time course of the DLCO changes more accurately and investigating the effects on the different resistance elements by using gases with other oxygen concentrations than air.

In conclusion, hemoptysis can occur in healthy subjects even after shallow breath-hold dives preceded by full expiration and GE. Laryngeal symptoms are substantiated by laryngoscopic findings suggesting that considerable mechanical stress occurs in the larynx in association with thoracic squeeze, in accordance with previous MRI findings in the trachea during GE maneuvers. Edema is possibly also caused in the lower airways, as suggested by the reduced dynamic spirometry.

GRANTS

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REFERENCES


