Case Report

Pneumomediastinum after lung packing.

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Jacobson FL, Loring SH, Ferrigno M. Pneumomediastinum after lung packing. Undersea Hyperb Med 2006; 33(5):313-316. Lung packing (glossopharyngeal insufflation) consists of forcing air into the lungs, using glossopharyngeal muscle contractions similar to swallowing. Breath-hold divers perform this technique after a maximal inhalation prior to diving, thus increasing initial lung volume. However, as suggested by previous authors, this breathing maneuver could theoretically lead to lung rupture. Here we report a pneumomediastinum found on chest CT scan in a diver during a physiological study, when glossopharyngeal insufflation increased the volume of gas in the lungs by 1,040 ml (over his total lung capacity); at the same time, his transpulmonary pressures increased up to 4.1 kPa. We discuss the possibility that the very high transpulmonary pressures during lung packing caused this pneumomediastinum.

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INTRODUCTION

Lung packing by glossopharyngeal insufflation (GI), also known in the medical community as glossopharyngeal breathing (1), consists of forcing air into the lungs, using glossopharyngeal muscle contractions similar to swallowing. Typically, a breath-hold diver performs this technique after a maximal inhalation prior to diving, thus increasing both the initial lung volume at the surface and the volume of gas available at depth for pressure equalization (cf. 2). This technique is reported to increase vital capacity (VC) by up to 39% (3). However, as first suggested by Vann (4), the large increases in airway pressure recorded during lung packing, reportedly up to 6.4 kPa in (3) and 8.0 kPa in (4), could lead to lung rupture. Although lung damage is a theoretical risk, to our knowledge, such an event has never been reported in the literature, despite the frequent use of lung packing by breath-hold divers for almost 50 years (5).

CASE REPORT

A 30 year old Caucasian male elite breath-hold diver participated in a study of both glossopahryngeal insufflation and glossopahryngeal exsufflation (GE), another breathing maneuver by which air can be extracted from the lungs using glossopharyngeal muscle contractions (2). The study was approved by the institutional Human Research Committee and the subject gave informed consent. This breath-hold diver had reached a maximal depth of 61 m in the ocean and could hold his breath for 6 min 21 s in a pool, and had used both GI and GE maneuvers frequently during his training for at least 3 years, without signs or symptoms

of lung damage. He had never smoked and he had no other medical conditions, nor history of previous lung diseases or thoracic surgery. He was fit, weighed 70 Kg and was 182 cm tall. In this study, he underwent measurements of respiratory mechanics and computed tomography (CT) scans of his chest at different lung volumes, as detailed below.

Pulmonary mechanics were measured with an esophageal balloon-catheter and flow meter to obtain the static deflation pressure-volume (P-V) characteristic of the lungs. The esophageal balloon-catheter, 10 cm long and containing 0.5 ml air, was passed through his nose to position its tip at 40 cm from the nares. Proper positioning of the balloon was confirmed by finding negligible change in transpulmonary pressure (airway pressure minus esophageal pressure) during respiratory efforts made against an occlusion. The transpulmonary pressure was determined at total lung capacity (TLC), i.e., the lung volume achieved after a maximal inhalation, and at TLC augmented by GI. The transpulmonary pressure was 2.4 and 2.5 kPa in two satisfactory maneuvers at TLC, and increased to 2.9 and 4.1 kPa in maneuvers after GI.

Multidetector CT scans of the diver's chest were performed on a Siemens Sensation 64 slice CT Scanner (Siemens Medical Solutions of Siemens AG Erlangen, Germany) using 120 kV and variable mAs, ranging from 60 to 270, based on automated evaluation of body part for thickness and density. Four data acquisitions were made through the entire chest, sequentially at TLC, GI, Residual Volume (RV) and GE; lung volume estimates were obtained from 3D reconstruction of CT images (Voxar 3D software, Duluth, GA 30097). With GI maneuvers, right lung volume increased from 4760 ml at TLC to 5345 ml; the left lung volume increased from 4410 ml at TLC to 4865 at GI. On all four data acquisitions, CT scans revealed a small stable pneumomediastinum, shown in Fig 1. This small amount of mediastinal air, probably less than 10 ml, was not visible on chest radiograph. He did not exhibit any signs or report any symptoms, and no other abnormalities were found on the CT scans of his chest.

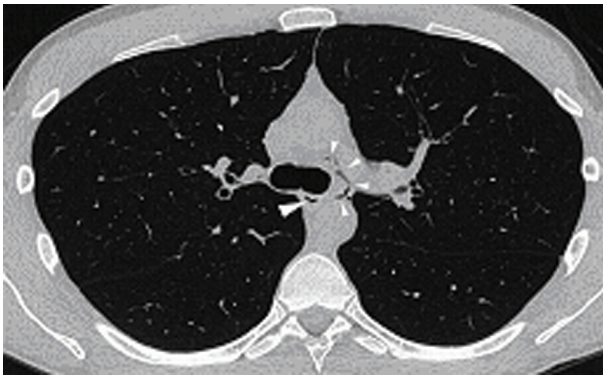

Fig. 1. Axial CT image at TLC shows a small pneumomediastinum; gas is visible adjacent to the left mainstem bronchus, anterior to the aorta, as indicated by small arrows. The large arrow points to the small gas collection normally present in the esophagus.

DISCUSSION

Our case report suggests the possibility that GI maneuvers can increase transpulmonary pressure enough to cause lung rupture; this may have caused pneumomediastinum in the breath-hold diver described here. However, in the absence of a previous lung CT, the possibility of a pre-existing pneumomediastinum cannot be excluded. While the risk of pulmonary barotrauma from GI has been previously mentioned in the literature (e.g. 3, 4), to our knowledge, this is the first report of pneumomediastinum associated with this breathing maneuver. We found that both lung volume and transpulmonary pressure substantially increased from TLC to TLC augmented by GI, presumably leading to stress failure of the pulmonary epithelium. Then, as originally described by Macklin and Macklin (6), gas enters the perivascular interstitium, dissecting proximally within

the bronchovascular sheath toward the mediastinum. Air can be seen in this pathway in our diver who exhibited pulmonary interstitial emphysema (Fig. 2).

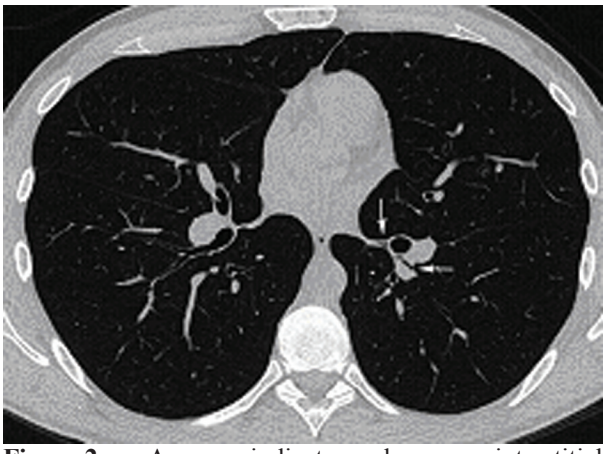

Fig 2. Arrows indicate pulmonary interstitial emphysema, as gas dissects through the interstitium adjacent to the airways.

Our findings and conclusions differ with those of Simpson et al. (7) who measured transpulmonary pressure during GI maneuvers in an elite breath-hold diver. They reported that transpulmonary pressure at TLC did not increase after GI (3.2 kPa at TLC versus 2.9 kPa after GI), despite a large increase in lung volume (from 9.28 to 11.01 l). This increase in volume of gas in the lungs was too great to be explained by displacement of intrapulmonary blood volume (normally about 0.4 l), and must therefore reflect an actual increase in lung size. The authors concluded that "buccal pumping itself does not carry a risk of pulmonary barotraumas," and suggested that increased elastic recoil of the chest wall at volumes greater than normal TLC would "splint" the lungs, thereby preventing pulmonary over-distention and consequent increase in transpulmonary pressure. If the chest wall had in fact "splinted" the lungs, it would have effectively prevented the large increases in lung volume they reported. Furthermore, the slope of the transpulmonary pressure-volume curve near TLC shown in their Fig. 2, suggests the increase in lung volume (from 9.28 l to 11.02 l) should have increased transpulmonary pressure by at least 0.8 kPa, whereas they reported a small decrease. These inconsistencies can be explained by assuming that some or all of the transpulmonary pressures they reported at TLC and after GI were measured when the glottis was closed. We also noted in their Fig. 3 (7), during exhalation after GI, an initial increase in transpulmonary pressure followed by a progressive decrease with exhalation, consistent with an esophageal contraction early in the exhalation. Such peristaltic contractions increase esophageal pressure and could explain the apparent decrease in maximal transpulmonary pressures after GI.

Simpson et al. cited Malhotra and Wright (8) for the possible protection against over expansion of the lungs by a stiff chest wall. These investigators measured the intratracheal pressures at which lung rupture occurred in human cadavers and found that thoracoabdominal binding led to a large increase in the airway (transrespiratory) pressure necessary to cause lung rupture (from 9.7 kPa to 25.3 kPa). However, neither lung volumes nor transpulmonary pressures were measured in that study, and the thoracoabdominal binding likely increased intrathoracic pressures and reduced lung expansion despite high airway pressure.

Pulmonary over-distention has not been reported in ventilator-dependent patients (mostly tetraplegic) who use glossopharyngeal breathing to extend periods of ventilator-free breathing (9). However, these patients perform GI maneuvers from volumes well below TLC and therefore do not over-distend their lungs. On the other hand, spontaneous pneumomediastinum can occur in young, healthy individuals after various strenuous activities, including sports (10). This rare condition is probably associated with increased alveolar volume, treatment is usually conservative, and spontaneous

resolution the rule (11). Retrosternal chest pain is the most frequent symptom and subcutaneous emphysema is the most common physical finding (10). Given the minimal volume of mediastinal air and its limited distributions in our diver, it is not surprising that he was asymptomatic. It is possible that he had a small pneumomediastinum prior to our study, but we suspect this developed during repeated GI maneuvers performed as part of our protocol, particularly in light of the increased maximal transpulmonary pressures and lung volumes measured with these breathing maneuvers.

Our case report of a pneumomediastinum in conjunction with lung packing suggests that these breathing maneuvers, particularly if forceful, can lead to pulmonary over-distention, with air dissecting centrally along the vessels into the mediastinum. If this occurs prior to breath-hold diving, it might predispose to pulmonary barotrauma during ascent (cf. 12).

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