Original articles

Pulmonary effects of ëlung packingí by buccal pumping in an elite breath-hold diver

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Key words
Breath-hold diving, barotrauma, buccal pumping, transpulmonary pressure, lung compliance

Abstract

(Simpson G, Ferns J, Murat S. Pulmonary effects of 'lung packing' by buccal pumping in an elite breath-hold diver. *SPUMS J* 2003; 33: 122-126) Buccal pumping is a technique used by breath-hold divers to increase lung capacity above normal total lung capacity (TLC) and thus increase depth capability. Concern has been expressed that hyperinflating the lungs using the pharyngeal muscles could itself produce pulmonary barotrauma, but transpulmonary pressures after buccal pumping have not previously been measured. We studied a breath-hold diver (SM) using whole-body plethysmography and oesophageal balloon manometry. Spirometry demonstrated that vital capacity could be increased from 7.48 to 9.22 l by buccal pumping. TLC increased from 9.28 to 11.02 l, calculated by assuming a constant residual volume of 1.8 l. At normal TLC, mean maximal pulmonary relaxation pressure measured at the mouth was 8.9 cm H_2O. This rose to 86 cm H_2O following buccal pumping to 'super' TLC. Mean transpulmonary pressure (mouth pressure minus oesophageal balloon pressure) at normal TLC was 31.6 cm H_2O and at super TLC was very similar at 29.3 cm H_2O. There did not seem to be a dramatic alteration in pulmonary compliance at higher lung volumes, although this was technically difficult to measure. These data suggest that buccal pumping itself does not carry a risk of pulmonary barotrauma. We postulate that the lack of rise in transpulmonary pressure relates to increased elastic recoil of the chest wall at volumes greater than normal TLC giving a positive intrapleural pressure and preventing pulmonary over-distension. Splinting of the chest wall has been shown experimentally to reduce the risk of pulmonary barotrauma in anaesthetised animals and fresh human cadavers, probably by a similar mechanism.

Introduction

Breath-hold or 'free' diving is the oldest form of diving and has been an important commercial activity for centuries for gathering pearls, sponges, bêche de mer etc., from the ocean floor. Spear fishing, using breath-hold techniques, remains a very popular pastime. In recent decades, however, breath-hold diving has developed rapidly as a competitive sport. There are four basic disciplines: simple breath-holding for time; constant-weight (unassisted) free diving to a maximum depth possible; distance diving, where the diver covers the maximum possible distance beneath the surface in the water in one breath (with or without fins and often in a pool environment); and variable-weight (assisted) or sled diving. In this last discipline great depths are achieved by increasing the rate of descent using a heavily weighted sled on a line and often using buoyancy aids to speed the return to the surface. The depths attained by free divers using these techniques are astonishing to recreational scuba divers. The official world record has gone from about 30 m in the 1940s to the current 171 m set by Audrey Mestre in October 2002. Mestre tragically died three days after she set her record whilst attempting to break it again. The physiology of breath-hold diving has been reviewed recently by Francis.[1]

In an effort to increase maximum depths attained, some divers have developed a technique, known as 'lung packing' or 'buccal pumping' to increase their lung volume above normal total lung capacity (TLC). This technique involves inspiring to TLC, closing the glottis, and gulping a mouthful of air. The air in the mouth is compressed using oral and pharyngeal muscles and then the glottis is opened and the air forced into the lungs. The pumping movement or 'chip' is then repeated up to 50 times. Buccal pumping was developed by spear-fishing breath-hold divers in the Mediterranean many years ago and introduced to sport diving by the US Navy diver Robert Croft in the 1960s.[2]

Increasing the volume of air in the lungs above TLC carries a theoretical risk of inducing pulmonary barotrauma. Though there have been reports of the cardiovascular effects, there is limited information on the pulmonary effects of buccal pumping.[3] It has been shown that relaxation mouth pressure, reflecting intrathoracic pressure, is increased considerably by buccal pumping and this has been interpreted as suggesting a substantial risk of lung rupture.[2] However, there are no data on the effects of buccal pumping on transpulmonary pressure. We report measurements of transpulmonary pressure and lung compliance during buccal pumping performed by an elite breath-hold diver.

Case report

SM, a 34-year-old dive instructor, was originally referred following a routine occupational diving medical because of concerns about a low forced expiratory volume in one second to forced vital capacity (FEV_1/FVC) ratio. In view of his greater than normal vital capacity it was not felt that this finding was a contraindication to diving. SM is a breath-hold diver who can hold his breath in excess of eight minutes, distance fin-swim underwater over 190 m, and descend to over 150 m and over 90 m in variable-weight and constant-weight breath-hold diving categories, respectively. At his interview, SM mentioned concerns regarding the risk of barotrauma with buccal pumping and requested further investigation.

Methods

Detailed lung-function testing, including whole-body plethysmography, was performed using a Sensormedics Vmax Autobox with Vmax Vision 5.2a software including spirometry with flow-volume loops, both normally and following buccal pumping. Intrathoracic pressure at TLC with and without buccal pumping was estimated by measurement of maximal mouth pressure at full inspiration with the glottis opened and respiratory muscles relaxed. In order to measure transpulmonary pressure (mouth pressure minus intrapleural pressure) an oesophageal balloon was passed and positioned above the diaphragm. A 10 cm balloon was used on a 100 cm oesophageal catheter. The initial trials passing the balloon nasally failed as the subject could not perform buccal pumping satisfactorily because of the consequent leak of air past the soft palate. The balloon was therefore passed orally for the experiments. The balloon was initially positioned 40 cm from the teeth and then withdrawn slightly. Position was checked by monitoring catheter pressure measurements during tidal breathing, confirming negative pressure during inspiration. The balloon was inflated with 0.5 ml of air during the test. Shutter closure was regulated manually. Measurements of static pulmonary compliance and transpulmonary pressures were taken with normal breathing to TLC and following buccal pumping (to 'super' TLC). Intrathoracic pressure at super TLC was derived from transpulmonary and mouth recoil pressure.

Results

The results are summarised in Table 1. They demonstrate an increase in vital capacity of 1.74 l with buccal pumping.

TABLE 1
Spirometry, transpulmonary pressures and lung relaxation pressures at normal TLC and following buccal pumping to super TLC. TLC after buccal pumping and intrapleural pressures are derived values (see text; TLC - total lung capacity)

Parameter	Normal	Buccal pumping
FEV_1 (l) (Forced expiratory volume.sec^{-1})	5.13	5.87
FVC (l) (Forced vital capacity)	7.48	9.22
TLC (l)	9.28	11.02
Mean max. mouth pressure (mouth minus atmospheric, cmH_2O)	8.9	86
Mean transpulmonary pressure (mouth minus oesophageal, cmH_2O)	31.6	29.3
Calculated mean intrapleural pressure (cmH_20)	-22.7	+56.7

FIGURE 1
Flow (l .min^{-1}) pressure (cmH_2O) and volume (l) traces at normal TLC (total lung capacity) during pulmonary compliance and maximal relaxation pressure testing

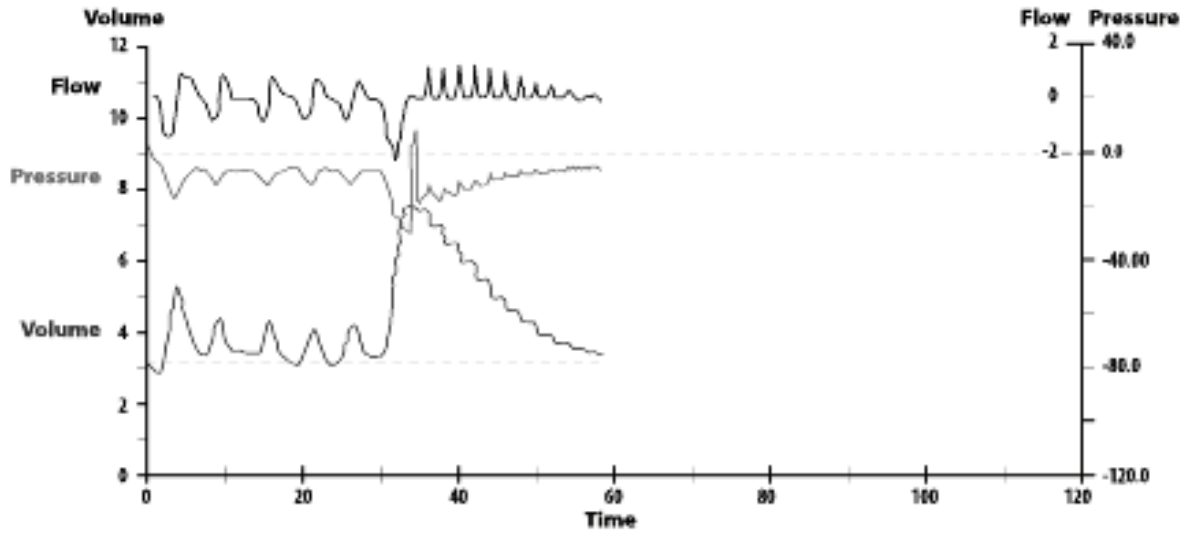
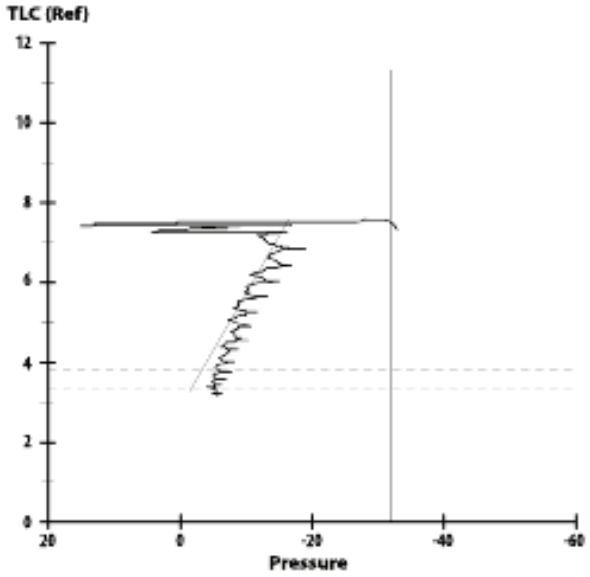


FIGURE 2
Pressure/volume curve from normal TLC showing derivation of static lung compliance (given by the slope of the diagonal line) and maximal trans-pulmonary pressure (indicated by the vertical line) (pressure - cmH_2O, volume - l)



SM found it impossible to perform buccal pumping with a mouthpiece in place so the TLC after buccal pumping is calculated by assuming that the residual volume (RV) of 1.8 l, measured during whole-body plethysmography, remains constant. Vital capacity thus is increased by 23.3% and, assuming RV is constant, TLC by 18.9%.

Figure 1 shows the flow, pressure and volume traces during a static lung compliance measurement at normal TLC. Figure 2 shows the pressure/volume relationships at normal TLC and demonstrates how static lung compliance is derived from this relationship. Maximal transpulmonary pressure is shown by the vertical line.

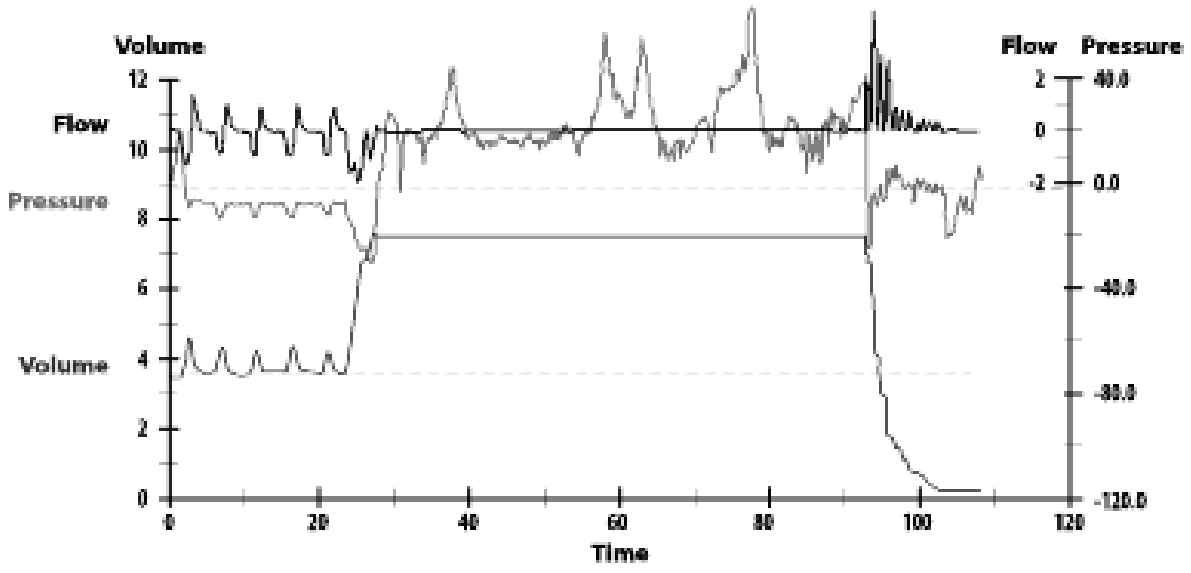
Figure 3 shows the flow, pressure and volume traces during buccal pumping with subsequent compliance and pressure measurement. It can be seen that, during the pumping manoeuvres, pressure rises in the mouth considerably and that, during the early part of the compliance measurement, flow from the lungs is rapid.

Results of mean maximal intrathoracic pressure (mouth pressure minus atmospheric pressure), mean transpulmonary pressure (mouth pressure minus oesophageal pressure) and calculated mean intrapleural pressure are shown in Table 1. It can be seen that there is no significant difference between the mean transpulmonary pressures at normal or at super TLC, though maximum mouth pressure rises considerably as does intrapleural pressure following buccal pumping.

Discussion

Barotrauma means damage caused by pressure. Increased pressure will not cause rupture of air spaces in the lung unless it is allowed to expand beyond the elastic limit of its tissues. It is, therefore, perhaps unfortunate that almost all the published literature on barotrauma concentrates on pressure changes rather than volume changes, and this includes this paper. Whether or not an airspace in a lung ruptures depends on how much it is allowed to expand and

FIGURE 3
Flow ($l.min^{-1}$) pressure (cmH_2O) and volume (l) traces after buccal pumping to super TLC (total lung capacity) during pulmonary compliance and maximal relaxation pressure testing (note high pressures in buccal pumping phase and rapid deflation on passive exhalation)



this is proportional to the pressure gradient across the walls. In the case of the whole lung, this is the transpulmonary pressure, i.e., central airway pressure minus intrapleural pressure. It is accepted that transpulmonary pressures of around 100 cm of water are sufficient to cause lung rupture in some circumstances.

Barotrauma has been reported after breath-hold ascent using scuba from a depth of around one metre, and this fits with experimental results.[4] Malhotra and Wright studied fresh, unchilled, human cadavers and showed that the intratracheal pressure at which pulmonary rupture occurred was around 75 mmHg (100 cm of water; 10 kPa).[5] They found that in cadavers whose chests and abdomens were tightly bound these pressures were very much higher. They postulated that this was because the overexpansion of the lungs was limited by the binding. The same authors produced similar findings in vivo using anaesthetised rabbits, confirming previous, similar experiments performed in the 1930s by Polak and Adams and confirmed by Schaefer et al in the 1950s using dogs.[6,7] Using these results and some arbitrary but reasonable estimates of alveolar pressure at TLC and lung compliance, the Thoracic Society of Australia and New Zealand (TSANZ) calculated that the increase in lung volume above which rupture occurs is 0.765 l.[8]

Our subject demonstrated an increase in lung volume by buccal packing that is more than double the figure calculated by the TSANZ to cause lung rupture. We have confirmed the rise in central airway pressure reported by Ornhagen et al that led them to be concerned about the danger of lung rupture.[2] However, direct measurements of transpulmonary pressure show no great increase, which can only mean that the intrapleural pressure has risen considerably during the course of buccal pumping. This would seem to be analogous in some way to the protective effects afforded by thoraco-abdominal binding in cadavers.

We would postulate that, in a young, fit, living and conscious subject, increasing the volume of air in the lungs by buccal pumping in fact hyper-pressurises rather than hyper-inflates the lungs as well as displacing blood from the pulmonary vessels, and that there is splinting of the lungs by the chest wall. This view would be supported by other data from Ornhagen et al who could show no convincing radiographic evidence of lung hyper-expansion following buccal pumping.[2]

It would seem from these results that buccal pumping is not of itself a major risk for pulmonary barotrauma occurring at the surface. Obviously, once descent occurs the risk vanishes. There are some caveats. Non-homogeneous compliance within the lung could cause problems, but the same situation should have occurred in the experimental models on which the maximum pressure figures are based. Secondly, the high pressures generated in the oropharynx and used to force air through the glottis into the lung have not been taken into account and this may be the most risky part of the manoeuvre. Many free divers in the world are performing this manoeuvre, yet there is no epidemiological evidence for or against the safety of buccal pumping. No case reports of barotrauma associated with buccal pumping have appeared.

Perhaps of equal concern are the cardiovascular effects of buccal pumping. Anecdotally, divers have reported fainting following buccal pumping, and it has been shown that the raised intrathoracic pressure associated with this manoeuvre reduces blood pressure, presumably by impeding venous return to the heart.[3]

What advantage do divers obtain by buccal pumping? For many years it was assumed that the depth limit for a breath-hold dive occurred when the total lung capacity had been compressed in accordance with Boyle's law to the lungs' residual volume. Most healthy individuals would reach this limit at a depth of 30 to 50 m. Once divers regularly started exceeding this theoretical limit, another mechanism was sought that would prevent lung squeeze. This seems to be transfer of blood into the pulmonary circulation.

Measurement of thoracic blood volume by impedance plethysmography in the 1960s showed that divers did transfer about one litre of blood to the pulmonary circulation during deep breath-hold diving.[9] Using the known lung volumes and diving records of another famous breath-hold diver, Jacques Mayol, it was possible to calculate that he too transferred approximately one litre of blood to his pulmonary circulation to enable him to achieve his record. In the case of our subject, without buccal pumping or blood transfer the maximum theoretical breath-hold diving depth is given by the formula:

$$(TLC/RV-1) \times 10 = 41 \text{ msw}$$

Assuming SM buccal pumps to the degree demonstrated in this paper he would reach residual volume at 51 msw. Currently, his maximal depth without buccal pumping is 154 msw. Assuming that this is achieved after inhaling to a TLC of 9.28 l, at this depth the thoracic gas volume would be 0.56 l. The calculated blood shift to the pulmonary circulation is 1.23 l, similar to previous findings in other divers.[10] Assuming this is the maximum amount of blood shift that he can achieve, the maximum depth he could achieve with buccal pumping would be $(11/0.56 - 1) \times 10 =$ 186 msw. That is to say, buccal pumping would be worth a theoretical 32 m of extra depth to our subject.

In the light of this sort of reward in such a competitive sport it seems unlikely that the practice of buccal pumping will disappear and further studies are indicated to confirm or refute its safety.

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