BRIEF REPORT

Glossopharyngeal Insufflation and Breath-Hold Diving: The More, the Worse?

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Objective.—The glossopharyngeal insufflation maneuver (lung packing) is largely performed by competitive breath-hold divers to improve their performance, despite observational evidence of fainting and loss of consciousness in the first seconds of apnea.

Methods.—We describe here the time course of hemodynamic changes, induced by breath-holding with and without lung packing, in 2 world-class apnea competitors.

Results.—When compared with apnea performed after a deep breath (100% vital capacity), lung packing leads to a decrease in cardiac output, blood pressure, and cerebral blood flow during the first seconds after the beginning of apnea. The major hemodynamic disorders were observed in diver 1, who exhibited the greater increase in pulmonary volume after lung packing (+22% for diver 1 vs +10% for diver 2). After the initial drop in both cardiac output and blood pressure, the time course of hemodynamic alterations became quite similar between the two apneas.

Conclusions.—Some recommendations, such as limiting the number of maneuvers and performing lung packing in the supine position, should be expressed to avoid injuries secondary to the use of glossopharyngeal insufflation.

Key words: cardiac function, echocardiography, apnea, cerebral blood flow, heart lung interaction, transcranial Doppler ultrasound, immersion pulmonary edema

Introduction

The glossopharyngeal insufflation maneuver, also called lung packing, is used to increase the volume of air in the lungs above physiological total lung capacity. After taking a deep breath, the divers pump air into the lungs using the oropharyngeal musculature. It has been previously reported that breath-hold divers were able to increase gas volume up to 4 L above their total lung capacity (commonly from 0.5 to 3 L). Apnea divers use lung packing to increase both diving depth and duration by increased oxygen stores, attenuation of the consequences of hyperbaria on the chest, and facilitation of pressure equalization in the ear during descent toward

the bottom.¹ However, lung packing also leads to profound hemodynamic changes resulting in a prompt drop in systemic arterial blood pressure.² The major decrease in cardiac output observed after lung packing has been related to an increase in transpulmonary pressure and autonomous nervous system alterations.

Observational evidence of fainting and loss of consciousness, called “packing blackout,” in the first seconds of apnea³ supported the suggestion that lung packing could be responsible for a major decrease in cerebral blood flow. Conversely, during a conventional apnea without lung packing, syncope occurred after several minutes of breath-holding or immediately after the end of apnea. Therefore, we hypothesize that in the first seconds of apnea the decrease in cerebral blood flow is more pronounced after lung packing than after deep breath. We compared the cardiovascular alterations induced by a 4-minute duration apnea performed at 100% of vital capacity (VC) and after lung packing in 2 world-class competitive breath-hold divers (BHD).

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Methods

The 2 world-class competitive BHD studied (diver 1, aged 32 years, height 182 cm, weight 73 kg; diver 2, aged 35 years, height 193 cm, weight 80 kg) are participating in apnea world championships. One of them is the current world champion of free diving (constant weight apnea without fins). They regularly use lung packing during competition and have determined their own optimal lung packing effort to improve their performance. Studies were carried out in a quiet, temperature-controlled room maintained at 27°C to 28°C. All investigations were performed in the morning, 3 hours after a light breakfast. The 2 subjects performed warm-up apneas before each session. The local Ethics Committee at Aix-Marseille University approved the study, and the subjects provided informed consent.

A preliminary study was conducted to assess the respiratory changes induced by lung packing. Dynamic spirometry and end-tidal O_2 concentration measurements were performed using a spirometer and gas analyzer (K4B2 PFT, Cosmed, Rome, Italy). Pulmonary function was studied according to American Thoracic Society standards. Forced expiratory volume in 1 second (FEV_1) and VC were measured at baseline. Furthermore, it was recommended to the subjects to completely exhale to residual volume to measure the end-tidal O_2 concentration ($PetO_2$). The same measurements were repeated in the 2 subjects after lung packing. Thereafter, the impact of lung packing on the cardiovascular alterations induced by a 4-minute duration apnea was investigated. For this purpose, the BHD were investigated in the supine position during 2 different apneas, 1 after a deep breath (ie, 100% VC) and 1 after lung packing. The divers underwent the 2 apneas in a randomized order. The apneas were separated by a rest period of normal breathing of 15 minutes. Pulse oximetry O_2 saturation was continuously monitored using a earlobe pulse oximeter Nellcor N-595 (Nellcor Inc, Pleasanton, CA).

CARDIAC FUNCTION TESTING

Heart rate (HR) was recorded using an analog 3-lead electrocardiogram (BIOPAC Systems, Goleta, CA). Continuous finger blood pressure (Finapres model 2300, Ohmeda Monitoring Systems, Englewood, CO) was sampled at 1 kHz (by an analog-to-digital converter, MP 150, BIOPAC Systems).

Stroke volume (SV) was assessed by echocardiography and Doppler (MyLab 30CV, Esaote, Genoa, Italy) from the combination of the aortic cross-sectional area (ACSA) and the aortic blood flow recorded on the same site. Aortic velocity envelope (AoVTI) was recorded

from the ascending aorta using a 2-MHz Doppler ultrasound probe positioned in the suprasternal notch.

The SV and cardiac output (CO) were calculated as follows: $SV = AoVTI \times ACSA$; $CO = SV \times HR$. Systemic vascular resistance (SVR) was calculated as mean blood pressure divided by cardiac output. Blood flow velocities of the right middle cerebral artery were continuously recorded using transcranial Doppler ultrasonography (Waki TCD, Atys Medical, Soucieu en Jarrest, France).

Results

BASELINE MEASUREMENTS

In diver 1, VC was measured at baseline to 6.85 L and after lung packing reached 8.37 L (+22%). In diver 2, VC was measured at baseline to 7.43 L and after lung packing reached 8.21 L (+10%). After lung packing, an increase in $PetO_2$ was found in the 2 BHD in comparison with baseline: in diver 1, $PetO_2$ was measured at 15.7 kPa at baseline (VC) and reached 17.6 kPa after lung packing (+12%); in diver 2, $PetO_2$ was measured at 16.1 kPa and reached 17.9 kPa after lung packing (+11%).

INVESTIGATIONS DURING APNEAS

The easy phase is the initial phase of apnea in which the subject feels no urge to breathe, in contrast with the second phase, called the “struggle” phase, during which involuntary breathing movements start. During apneas after lung packing, the duration of the easy phase was prolonged from 80 seconds to 120 seconds for diver 1 (+50%) and from 110 seconds to 130 seconds for diver 2 when compared with apnea performed at VC (+18%). Comparisons between the 2 apneas, preceded or not preceded by lung packing, are shown in [Figures 1 and 2](#).

HEART RATE

After an initial tachycardia, HR gradually decreased both at VC and after lung packing. For diver 1, who exhibited the greater increase in pulmonary volume by lung packing, tachycardia was more pronounced at the beginning of apnea after this maneuver. For diver 2, HR time courses were similar during the 2 apneas.

STROKE VOLUME

A decrease in SV was observed in the 2 subjects at the beginning of apneas. This decrease was more marked when the divers used lung packing, and reached –60% (from 90 mL to 37 mL) for diver 1 and –43% (from 114 mL to 66 mL) for diver 2 after these maneuvers.

Figure 1. Time course of (a) cardiac output, (b) mean blood pressure, and (c) systolic peak flow velocity of the mean cerebral artery during apnea performed after a deep breath (100% of vital capacity [solid line]) or after lung packing (100% of vital capacity plus lung packing [dashed line]) in diver 1. Vertical dotted lines indicate the beginning and end of apneas (0 to 240 s). Base, baseline; Rec, measurement performed during recovery 1 minute after the end of apnea.

CARDIAC OUTPUT

An initial increase in cardiac output secondary to the accelerated HR was found in the 2 subjects during the 4-minute apneas performed at VC (Figures 1A and 2A). This increase disappeared after 30 seconds of apnea; thereafter, a decrease was observed. In contrast, when divers performed lung packing, a decrease in cardiac output was found at the beginning of apneas. After 2

minutes of apnea, the time course of cardiac output was similar to apnea performed at VC.

SYSTEMIC VASCULAR RESISTANCE

When apnea was performed at VC, both divers exhibited an initial decrease in SVR (from 16 mm Hg to 12 mm Hg, percentage variation -24% in diver 1; and from

Figure 2. Time course of (a) cardiac output, (b), mean blood pressure, and (c) systolic peak flow velocity of the mean cerebral artery during apnea performed after a deep breath (100% of vital capacity [solid line]) or after lung packing (100% of vital capacity plus lung packing [dashed line]) in diver 2. Vertical dotted lines indicate the beginning and end of apneas (0 to 240 s). Base, baseline; Rec, measurement performed during recovery 1 minute after the end of apnea.

10 mm Hg to 8 mm Hg, -19% in diver 2). Thereafter, SVR increased continuously, reaching at the end of apnea $+412\%$ in diver 1 and $+389\%$ in diver 2. After lung packing, an initial decrease in SVR was recorded in the 2 divers (from 14 mm Hg to 8 mmHg, percentage variation -45% in diver 1; and from 13 mm Hg to 10 mm Hg, -18% in diver 2). Thereafter, SVR increased

continuously, reaching at the end of apnea $+367\%$ in diver 1 and $+328\%$ in diver 2.

BLOOD PRESSURE

At the beginning of apnea, when the subjects performed lung packing, a decrease in blood pressure was observed (Figures 1B and 2B).

TRANSCRANIAL DOPPLER ULTRASONOGRAPHY

A decrease in mean cerebral artery blood velocities was observed at the beginning of the apnea (Figures 1C and 2C), when the divers performed lung packing (from 46 to 18 cm/s, percentage variation -61% in diver 1; and from 33 to 21 cm/s, -36% in diver 2). This decrease was particularly pronounced in diver 1. After 30 seconds to 1 minute, velocities gradually increased, reaching a maximum at 4 minutes. This time course was similar in the 2 subjects during the 2 sessions (lung packing and VC apneas). At the end of apnea, oxygen saturation decreased to 79% in diver 1 and to 75% in diver 2 after apneas performed at VC. When the diver used lung packing, the decrease was measured to 81% in diver 1 and to 72% in diver 2.

Discussion

The glossopharyngeal insufflation maneuvers performed by the 2 divers allowed increasing pulmonary volume to $+22\%$ in diver 1 ($+1.52$ L) and to $+10\%$ in diver 2 ($+0.78$ L). Lung packing was associated with an increase in PetO_2 in both divers. Furthermore, during apneas, the use of lung packing allowed a prolongation of the easy phase. These findings agree with previous works⁴ and suggest that an increase in performance should be expected by the use of lung packing.

In our study, the data recorded from the 2 competitive divers demonstrated substantial changes of hemodynamic status induced by lung packing that seemed to be related to the amount of gas insufflated. At the beginning of the apneas, a decrease in blood pressure was recorded in both divers. This decrease was more marked when they performed lung packing. We observed a major decrease in blood pressure in diver 1, who exhibited the greater increase in pulmonary volume by lung packing in comparison with diver 2.

The decrease in blood pressure could be attributed to greater lowering of cardiac output secondary to the use of lung packing. Indeed, when the divers performed apnea after a deep breath, an increase in cardiac output was observed at the beginning of the apnea. This increase was secondary to an accelerated HR. After 30 seconds, HR slowed and cardiac output decreased. In contrast, although the HR remained accelerated at the beginning of apnea, the marked lowering in SV led to a substantial decrease in both cardiac output and cerebral blood flow when divers performed lung packing. These changes can explain loss of consciousness in the first seconds after lung packing. The decrease in cardiac output has been attributed to changes in cardiac load and ventricular interdependence.²

After the first seconds of apnea, the time course of hemodynamic status was comparable, regardless of using lung packing or not. Two minutes after the beginning of the apneas, cardiac output returned to a similar level in the 2 sessions. Thereafter, the divers experienced a progressive decrease of cardiac output secondary to the decrement in both SV and HR. At the end of the 4-minute apnea, the decrease in HR was responsible for a major decrease in cardiac output in both conditions. The increase in cerebral blood velocities, recorded by transcranial Doppler ultrasonography, expressed the brain protection under hypoxemic condition. Lastly, the recovery seemed to be similar for the 2 divers, regardless of whether they used lung packing.

These findings in the 2 world-class divers suggested that the greater the pulmonary volume increased by lung packing, the greater the hemodynamic disorder. Previous works have demonstrated that lung packing can provoke other injuries such as asystole,³ arterial gas embolism,⁵ or pneumothorax.⁶ Consequently, the question whether to forbid this maneuver in competition should be seriously considered.

Furthermore, this maneuver is also used by BHD during their training and by other elite athletes, such as swimmers.⁷ As this maneuver is most frequently learned on land, it is recommended that such maneuvers be performed in the supine position, because impairment in cardiac load induced by lung packing can result in a major decrease in cardiac output in the erect position, exposing the subjects to syncope and injuries from falls. Although risks remain in the supine position, the optimization of the cardiac preload may limit the decrease in cardiac output.

In water immersion conditions, the redistribution of blood flow from the caudal portions of the body to the intrathoracic circulation⁸ can attenuate the impairment in cardiac output secondary to lung packing. Nevertheless, a loss of consciousness exposes the diver to drowning.

Lastly, at depth, the high pulmonary volume obtained by the use of lung packing might protect against immersion pulmonary edema and alveolar hemorrhage observed in BHD.⁹ Indeed, the reduction in pulmonary volume at depth leads to a major increase in thoracic blood volume associated with a restrictive left ventricle filling pattern. Pulmonary vascular engorgement can induce capillary stress failure and edema. Using submersible Doppler 2-dimensional echocardiography, Marabotti et al¹⁰ recently reported that these changes were reversed after lung volume re-expansion using scuba apparatus at depth. Consequently, when the diver begins the apnea at higher pulmonary volume using lung packing, the pulmonary vascular engorgement might be attenuated at depth.

Further studies are needed to better assess the benefit and risk ratio of lung packing. If lung packing remains authorized, it is important to alert the competitors to the risks induced by this maneuver, and to recommend that breath-hold divers determine the minimal lung packing useful to improve their equilibration maneuvers and chest comfort sensation at depth. Indeed, the amount of gas insufflated should be limited to avoid the risk of hemodynamic disorder and pulmonary barotrauma. Furthermore, it is important to recommend a strict surveillance of breath-hold divers at the beginning of apnea.

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