Effects of glossopharyngeal insufflation on cardiac function: an echocardiographic study in elite breath-hold divers

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Potkin R, Cheng V, Siegel R. Effects of glossopharyngeal insufflation on cardiac function: an echocardiographic study in elite breath-hold divers. J Appl Physiol 103: 823–827, 2007. First published June 7, 2007; doi:10.1152/japplphysiol.00125.2007.—Glossopharyngeal insufflation (GI), a technique used by breath-hold divers to increase lung volume and augment diving depth and duration, is associated with untoward hemodynamic consequences. To study the cardiac effects of GI, we performed transthoracic echocardiography, using the subcostal window, in five elite breath-hold divers at rest and during GI. During GI, heart rate increased in all divers (mean of 53 beats/min to a mean of 100 beats/min), and blood pressure fell dramatically (mean systolic, 112 to 52 mmHg; mean diastolic, 75 mmHg to nondetectable). GI induced a 46% decrease in mean left ventricular end-diastolic area, 70% decrease in left ventricular end-diastolic volume, 49% increase in mean right ventricular end-diastolic area, and 160% increase in mean right ventricular end-diastolic volume. GI also induced biventricular systolic dysfunction: left ventricular ejection fraction decreased from 0.60 to a mean of 0.30 (P = 0.012); right ventricular ejection fraction, from 0.75 to a mean of 0.39 (P < 0.001). Wall motion of both ventricles became significantly abnormal during GI; the most prominent left ventricular abnormalities involved hypokinesis or dyskinesis of the interventricular septum, while right ventricular wall motion abnormalities involved all visible segments. In two divers, the inferior vena cava dilated with the appearance of spontaneous contrast during GI, signaling increased right atrial pressure and central venous stasis. Hypotension during GI is associated with acute biventricular systolic dysfunction. The echocardiographic pattern of right ventricular systolic dysfunction is consistent with acute pressure overload, whereas concurrent left ventricular systolic dysfunction is likely due to ventricular interdependence.

RECREATIONAL AND COMPETITIVE breath-hold divers have achieved depth records exceeding maximal diving depths predicted from the physiological ratio of total lung capacity (TLC) to residual lung volume (6, 13). Increasing lung volume beyond TLC in breath-hold diving is desirable to augment oxygen stores, increase intrathoracic gas for better pressure equalization, and reduce chest compression, potentially allowing for free diving to greater depths (7, 13, 14). One technique used to achieve such high lung volumes is called “glossopharyngeal insufflation,” also known as “buccal pumping” or “lung packing.” After maximal inspiration to TLC, the diver fills the mouth with air, while the glottis remains closed, then opens the glottis and forces this air into the lung. The increase in lung volume above TLC from glossopharyngeal insufflation may be due to an increase in vital capacity with little or no change in residual volume (2, 9, 13, 14, 17). This technique has reportedly been utilized by post-polio and quadriplegic patients as a temporary alternative to mechanical ventilation (3).

Multiple reports have shown that significant hemodynamic abnormalities occur during glossopharyngeal insufflation. Arterial blood pressure falls, and heart rate increases during this maneuver (2, 7–9, 13, 14). Decrease in intrathoracic blood volume and cardiac size have been noted on magnetic resonance imaging (12). Glossopharyngeal insufflation has been associated with light-headedness and even loss of consciousness (Ref. 2 and R. Potkin, unpublished observations). Whether these untoward changes are due to impaired cardiac function, however, has not previously been elucidated. We conducted an echocardiographic study to better define the effects of glossopharyngeal insufflation on cardiac function.

METHODS

Five elite breath-hold divers (identified by having a personal best breath-hold time ≥6 min or dive depth ≥65 m) first underwent standard pulmonary function testing, using the Puritan Bennett Renaissance Spirometry system (Pleasanton, CA) and Knudson predicted values. Vital capacity measurement was performed four times for each diver pre- and postglossopharyngeal insufflation, and the highest recorded volume was reported. Transthoracic echocardiography (TTE), arterial blood pressure, and heart rate measurements were then performed on all divers at rest and during glossopharyngeal insufflation.

Echocardiographic procedure. Each diver had resting supine blood pressure (by manual sphygmomanometry) and heart rate measured. Standard TTE was then performed using the Philips IE33 system (Philips, Bothell, WA), with the diver in the left lateral decubitus position for parasternal and apical imaging and the supine position for subcostal images. The diver was then asked to inspire to TLC and perform the glossopharyngeal insufflation maneuver. The number of buccal pumping maneuvers ranged from 15 to 30, lasting a total of 30 s. During glossopharyngeal insufflation, blood pressure and heart rate measurements were repeated, and subcostal echocardiographic views were obtained (as the maneuver created lung artifact that severely degraded echocardiographic images from other ultrasound transducer positions). The diver then resumed normal respiration.

Echocardiographic measures. Because only subcostal views provided adequate windowing during glossopharyngeal insufflation, we limited all comparison measurements to this view. Images were stored using the Camtronic digital imaging system (Emageon, Birmingham, AL) for off-line analysis. Quantitative measures included the following: end-systolic and end-diastolic area of the left and right ventricles (obtained by manual tracing of ventricular endocardium), end-systolic and end-diastolic volume of both ventricular cavities (calculated by quantitative imaging (12)). Glossopharyngeal insufflation has been associated with light-headedness and even loss of consciousness (Ref. 2 and R. Potkin, unpublished observations). Whether these untoward changes are due to impaired cardiac function, however, has not previously been elucidated. We conducted an echocardiographic study to better define the effects of glossopharyngeal insufflation on cardiac function.

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Simpson’s rule, Ref. 21), ejection fraction (EF) of both ventricles (calculated using end-diastolic and end-systolic volumes), and diameter of the inferior vena cava (IVC). All quantitative measurements were obtained by two separate, trained readers blinded to each other’s results. Qualitative measures included left and right ventricular wall motion and development of spontaneous contrast in the IVC. Because visualization was limited to subcostal images, wall motion could be evaluated only for the following ventricular segments: basal, mid-, and distal lateral left ventricular wall; basal, mid-, and distal interventricular septum; and basal and distal right ventricular free wall. Each ventricular segment was categorized as normal, moderately hypokinetic, severely hypokinetic, akinetic, or dyskinetic.

Statistical analyses. For each quantitative variable, the average of the two reader measurements was used as the diver data point in the statistical analyses. Interreader variability of the measurements was assessed by the intraclass correlation coefficient and the mean reader difference (expressed as a percentage of the average of the reader measurements). Results for quantitative variables were summarized as means ± SD. Comparisons of quantitative variables at rest and during glossopharyngeal insufflation were performed by the paired t-test or the Wilcoxon signed-rank test, as appropriate.

This study was reviewed and approved by the institutional review board at the Cedars-Sinai Medical Center.

RESULTS

The five divers included four men and one woman, with a mean age of 37.2 yr (range, 28–54 yr). No diver had any significant past or current medical problems, and none took any prescription medication. All divers exhibited above-normal vital capacity at rest and increased vital capacity on pulmonary function testing after glossopharyngeal insufflation. Other demographic data of the divers are shown in Table 1.

Table 2 summarizes mean quantitative hemodynamic and echocardiographic findings before and during glossopharyngeal insufflation for all five divers. During glossopharyngeal insufflation, all divers experienced a significant increase in heart rate (from a mean of 53 to 100 beats/min) and became significantly hypotensive (mean systolic blood pressure decreased from 112 to 75 mmHg, and diastolic blood pressure decreased from 82 to 70 mmHg). Lindholm and Nyren (13) reported a mean increase in heart rate from 78 to 97 beats/min and mean systolic blood pressure drop from 122 to 70 mmHg. Lindholm and Nyren (13)

Table 1. Demographic data of the five divers studied

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Diver 1</th>
<th>Diver 2</th>
<th>Diver 3</th>
<th>Diver 4</th>
<th>Diver 5</th>
</tr>
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<tbody>
<tr>
<td>Sex</td>
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<td>Male</td>
<td>Male</td>
<td>Male</td>
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<tr>
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<td>32</td>
<td>35</td>
<td>37</td>
<td>54</td>
</tr>
<tr>
<td>Height, m</td>
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<td>1.70</td>
<td>1.91</td>
<td>1.83</td>
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<tr>
<td>Weight, kg</td>
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<td>88.6</td>
<td>84.1</td>
<td>83.2</td>
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<tr>
<td>BMI</td>
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<td>20.4</td>
<td>24.3</td>
<td>25.1</td>
<td>27.2</td>
</tr>
<tr>
<td>Comorbidities</td>
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<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Resting vital capacity, liters</td>
<td>7.56</td>
<td>4.56</td>
<td>6.17</td>
<td>6.49</td>
<td>6.26</td>
</tr>
<tr>
<td>% predicted*</td>
<td>155</td>
<td>119</td>
<td>115</td>
<td>117</td>
<td>149</td>
</tr>
<tr>
<td>Insufflation vital capacity, liters</td>
<td>9.59</td>
<td>5.65</td>
<td>6.45</td>
<td>8.42</td>
<td>7.47</td>
</tr>
<tr>
<td>% predicted*</td>
<td>197</td>
<td>147</td>
<td>121</td>
<td>152</td>
<td>178</td>
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<tr>
<td>Insufflation volume, liters</td>
<td>2.03</td>
<td>1.09</td>
<td>0.28</td>
<td>1.93</td>
<td>1.21</td>
</tr>
</tbody>
</table>

BMI, body mass index. *Predicted vital capacities are from Knudson.

during glossopharyngeal insufflation. Mean right ventricular EF decreased by 48% to 0.39 (P < 0.001), and mean left ventricular EF decreased by 50% to 0.30 (P = 0.012).

Figures 1 and 2 depict changes in each diver’s quantitative hemodynamic and echocardiographic measures during glossopharyngeal insufflation, respectively. All divers exhibited a decrease in measured left ventricular area, left ventricular volume, and left and right ventricular EFs. With the exception of diver 5, all divers exhibited an increase in right ventricular area and volume.

Glossopharyngeal insufflation induced abnormal wall motion in both ventricles, as shown in Table 3. The most prominent left ventricular wall motion abnormalities involved the interventricular septum, which became either severely hypokinetic or dyskinetic. Right ventricular wall motion abnormalities were more diffuse, with hypokinesis or dyskinesis of nearly all visible segments.

Diver 3 and diver 5 had adequate echocardiographic images to allow evaluation of the IVC before and during glossopharyngeal insufflation. In both cases, the IVC size was normal (diameter was 19 mm in both), with normal inspiratory collapse at rest, and then became dilated (diver 3 to 23 mm and diver 5 to 27 mm) with appearance of spontaneous contrast during glossopharyngeal insufflation.

Reader agreement. Overall, there was good intraclass correlation of quantitative echocardiographic measures between readers. Mean interrater differences on quantitative variables were generally small (<5%) in relation to the actual variable magnitude.

DISCUSSION

Some competitive breath-hold divers perform glossopharyngeal insufflation to acutely increase lung volume, exceeding physiological TLC to achieve greater diving depths or longer breath-hold times. While the exact mechanism of how glossopharyngeal insufflation assists the diver is unknown, the maneuver itself has been associated with unidirectional hemodynamic effects. In three subjects performing glossopharyngeal insufflation, Andersson et al. (2) reported a mean increase in heart rate from 78 to 97 beats/min and mean systolic blood pressure drop from 122 to 70.6 mmHg. Lindholm and Nyren (13)
observed pronounced reductions in heart and intrathoracic vessel size in elite breath-hold divers performing this maneuver. Transpulmonary pressure elevation has been reported with glossopharyngeal insufflation in a similar cohort (13, 15, 17).

Cardiac performance during breath holding without glossopharyngeal insufflation has been evaluated by a variety of methods (9, 14). Using impedance cardiography, Ferrigno et al. (9) found that left ventricular performance, expressed as a combination of a shortening of the left ventricular ejection time and a lengthening of the pre-ejection period, decreased during breath holding. The authors attributed the fall in stroke volume to decreased venous return.

By performing TTE during glossopharyngeal insufflation, we found that glossopharyngeal insufflation induced acute right ventricular systolic dysfunction, with a pattern—cavity enlargement, diffuse wall motion abnormalities, and dilation of the IVC—typical of changes seen in acute right ventricular pressure overload. Lung volume increase during glossopharyngeal insufflation has been shown to increase intrathoracic and transpulmonary pressures (9, 13, 14, 17, 20, 25) and pulmonary vascular resistance (18–20, 23). It is likely that acute pulmonary pressure elevation during glossopharyngeal insufflation causes right ventricular systolic dysfunction from pressure overload, contributing to systemic hypo-

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**Fig. 1.** Quantitative hemodynamic measures of each diver at rest and during glossopharyngeal insufflation. bpm, Beats/min.

**Fig. 2.** Quantitative echocardiographic measures of each diver at rest and during glossopharyngeal insufflation.

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We found that glossopharyngeal insufflation also induced significant left ventricular dysfunction. Acute right ventricular dilatation can impair left ventricular filling by ventricular interdependence (10, 12, 16, 18–20, 22, 23, 27), and a marked increase in pulmonary vascular impedance from transmitted intrathoracic pressure can reduce pulmonary venous flow into the left atrium. Furthermore, in animal models of pulmonary hypertension, acute right ventricular dilatation has been shown to cause left ventricular contractile dysfunction (1, 4, 24). Alteration in left ventricular geometry is thought to play a major role (24).

A decrease in venous return has been theorized to reduce cardiac output in breath-hold divers (2, 9, 12). In our study, the appearance of spontaneous contrast in the IVC during glossopharyngeal insufflation indicated that the maneuver induced central venous stasis (11), likely as a consequence of acute pulmonary pressure elevation. This echocardiographic phenomenon is consistent with states of reduced venous return.

Adverse hemodynamic consequences of spontaneous and mechanical ventilation have been shown to be dependent on changes in lung volume and intrathoracic pressure. During positive pressure ventilation, the increase in intrathoracic pressure and lung volume reduces venous return and left ventricular end-diastolic volume (17–19, 21–23). The increase in pulmonary vascular resistance can cause right ventricular dilatation, right ventricular dysfunction, and abnormal septal movement into the left ventricle, decreasing left ventricular compliance. In addition, the heart may be compressed in the cardiac fossa, further decreasing left ventricular compliance while altering left ventricular configuration and volume (17, 18, 21–24). In our study, glossopharyngeal insufflation produced echocardiographic changes associated with many of the aforementioned findings, suggesting that glossopharyngeal insufflation affects cardiac function in a manner similar to positive-pressure ventilation.

Our study was performed with the divers in ambient air. While breath-hold divers do perform glossopharyngeal insufflation in air as part of training, they perform the maneuver more frequently in the immersed state, during both training and competition. We expect the echocardiographic and hemodynamic changes observed in our study to be ameliorated by immersion for two reasons. Compared with ambient air, the amount of lung expansion is limited by immersion and further reduced during descent due to the increase in ambient pressure, attenuating the cardiodepressive effects associated with very high lung volumes (7, 9, 14, 17). In addition, during immersion, there is redistribution of blood from the periphery to the intrapulmonary vessels, increasing intrathoracic blood volume (9, 14, 17), cardiac preload, and cardiac output.

In conclusion, we found that glossopharyngeal insufflation causes hypotension by inducing biventricular systolic dysfunction. The echocardiographic pattern of right ventricular systolic dysfunction is quite similar to that seen in acute pressure overload and is likely due to the transmission of acutely increased intrathoracic pressure to the pulmonary vasculature. The concomitant development of left ventricular systolic dysfunction likely reflects a combination of impaired left ventricular filling and decreased contractility from ventricular interdependence. Due to its marked depressive effect on cardiac function, glossopharyngeal insufflation is associated with serious hemodynamic consequences for divers in the nonimmersed state; therefore, the safety of this practice warrants further investigation.

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REFERENCES