Cardiac Magnetic Resonance Imaging during Pulmonary Hyperinflation in Apnea Divers

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ABSTRACT

BATINIC, T., W. UTZ, T. BRESKOVIC, J. JORDAN, J. SCHULZ-MENGER, S. JANKOVIC, Z. DUJIC, and J. TANK. Cardiac Magnetic Resonance Imaging during Pulmonary Hyperinflation in Apnea Divers. *Med. Sci. Sports Exerc.*, Vol. 43, No. 11, pp. 2095–2101, 2011. **Purpose**: Apnea divers hyperinflate the lung by taking a deep breath followed by glossopharyngeal insufflation. The maneuver can lead to symptomatic arterial hypotension. We tested the hypotheses that glossopharyngeal insufflation interferes with cardiac function further reducing cardiac output (CO) using cardiac magnetic resonance imaging (MRI) to fully sample both cardiac chambers. **Methods**: Eleven dive athletes (10 men, 1 woman; age = 26 ± 5 yr, body mass index = 23.5 ± 1.7 $kg \cdot m^{-2}$) underwent cardiac MRI during breath holding at functional residual capacity (baseline), at total lung capacity (apnea), and with submaximal glossopharyngeal insufflation. Lung volumes were estimated from anatomic images. Short-axis cine MR images were acquired to study biventricular function. Dynamic changes were followed by long-axis cine MRI. **Results**: Left and right ventricular end-diastolic volumes (LVEDV, RVEDV) decreased during apnea with and without glossopharyngeal insufflation (baseline: LVEDV = 198 ± 19 mL, RVEDV = 225 ± 30 mL; apnea: LVEDV = 125 ± 38 mL, RVEDV = 148 ± 37 mL, $P < 0.001$; glossopharyngeal insufflation: LVEDV = 108 ± 26 mL, RVEDV = 136 ± 29 mL, $P < 0.001$ vs baseline). CO decreased during apnea (left = −29 ± 4 %, right = −29 ± 4 %) decreasing further with glossopharyngeal insufflation (left = −38% ± 4%, right = −39% ± 4%, $P < 0.05$). HR increased 16 ± 4 bpm with apnea and 17 ± 5 bpm with glossopharyngeal insufflation ($P < 0.01$). Ejection fraction moderately decreased (apnea: left = −5% ± 2%, right = −7% ± 2%, glossopharyngeal insufflation: left = −6% ± 2%, right = −10% ± 2%, $P < 0.01$). With continued apnea with and without glossopharyngeal insufflation, LVEDV and CO increased over time by a similar but small amount ($P < 0.01$). **Conclusions**: The major finding of our study was that submaximal glossopharyngeal insufflation decreased CO further albeit by a small amount compared to maximal inspiratory apnea. The response was not associated with severe biventricular dysfunction. **Key Words:** DIVING PHYSIOLOGY, GLOSSOPHARYNGEAL INSUFFLATION, CARDIAC OUTPUT, SYSTOLIC FUNCTION

BASIC SCIENCES

Apnea divers practice voluntary lung hyperinflation. They hyperventilate, inspire deeply, and then perform glossopharyngeal insufflation ("lung packing") (27,40). Air is taken into the mouth and pharynx and "pumped" repeatedly into the lungs. On endoscopic examination, the soft palate is elevated during the maneuver, thus attenuating air leakage through the nose. The pharyngeal lumen is initially expanded and then contracted in a coordinated sequence including larynx elevation, glottis opening, and posterior tongue displacement, as air is injected into the trachea. Thus, apnea divers provide an excellent model for cardiac and hemodynamic studies on lung hyperinflation. Competitive breath-hold divers apply glossopharyngeal insufflation to increase lung gas content above baseline total lung capacity (TLC), thus improving performance.

Cardiac output decreases within the first apnea minute without glossopharyngeal insufflation and partially recovers later on (16,33,34). Glossopharyngeal insufflation further increases TLC by approximately 24% accompanied by an increase in static lung compliance (7,30,32). Glossopharyngeal insufflation compresses intrathoracic vessels and profoundly changes the geometry of both cardiac ventricles. The maneuver decreases intrathoracic blood volume reducing end-diastolic volume and stroke volume (27,37). Symptomatic hypotension can occur (9,10,28). In an ECG study, apnea with glossopharyngeal insufflation induced biventricular systolic dysfunction with ventricular ejection fractions as low as 30% (37). A recent cardiac magnetic resonance imaging (MRI) study showed reduction in left ventricular (LV) contractile function with marked LV enlargement during prolonged apnea (36). ECG data may be less precise because cardiac volumes are calculated from two-dimensional measures applying geometric assumptions on cardiac chamber symmetry. It is not surprising that

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two-dimensional techniques have their limitations in after RV and LV volumes during physiological maneuvers altering chamber symmetry. Moreover, intraobserver and interobserver variability is relatively high (13,17,31). If voluntary lung hyperinflation would, indeed, elicit profound decreases in cardiac function in trained athletes, the observation could be clinically important for patients with "involuntary" lung hyperinflation, such as chronic obstructive pulmonary disease or positive pressure ventilation (26). However, reliable data on LV and RV geometry and systolic function during lung hyperinflation are limited. Therefore, we applied cardiac MRI, which is considered the standard of reference for cardiac morphology and function, to study the effect of lung hyperinflation on biventricular geometry and function in apnea divers (20,21). To our knowledge, this is the first cardiac MRI study measuring influences of glossopharyngeal insufflation on RV and LV size and function. We hypothesized that glossopharyngeal insufflation profoundly exacerbates changes in ventricular volumes and cardiac output elicited by apnea.

METHODS

We recruited 11 active apnea divers (10 men, 1 woman; age = 26 ± 5 yr, weight = 80 ± 8 kg, height = 185 ± 6 cm, body mass index = 23.5 ± 1.7 $kg \cdot m^{-2}$). In preceding months, they had participated in at least seven diving competitions and at least 70 training sessions each consisting of 30–40 maximal apneas. Divers were accustomed to control their inspired volume. All were healthy nonsmokers on no medications. The University of Split, School of Medicine, ethics committee approved the study and written informed consent was obtained.

Imaging protocol. Divers arrived at the laboratory at least 2 h after having a light breakfast and had abstained from caffeine for at least 12 h. To familiarize divers with the laboratory environment and study procedures, they practiced apnea with and without glossopharyngeal insufflation during a prescanning session. Finger blood pressure and arterial oxygen saturation levels were recorded during the prescanning session only because the equipment could not be placed in the scanner room. We obtained continuous ECG recordings during the prescanning session and in the scanner room. Divers performed apnea wearing a nose clip with closed lips to avoid air leaks. For safety reasons, we instructed divers to perform submaximal glossopharyngeal insufflation in the MR scanner by counting the numbers of glossopharyngeal insufflations. We obtained all MRI measurements with a clinical 1.5-T MR scanner (Avanto; Siemens Medical Solutions AG, Erlangen, Germany) in the radiology department of the university hospital in Split, Croatia, using a dedicated cardiac phased-array coil. All subjects underwent an imaging protocol consisting of three parts.

Part 1 (image acquisition during repeated breath holds at functional residual capacity [FRC]): First, we acquired a stack of sagittal slices (thickness = 8 mm, interslice gap = 18 mm) covering the whole thorax to assess total lung volume using an untriggered balanced steady-state free precession (b-SSFP) technique (TR = 338 ms, TE = 1.35 ms, matrix = 256 × 256, field of view = 400 × 400 mm, in-plane resolution = 1.5 × 1.5 mm^2, imaging time = ~30 s). For quantification of cardiac structure and function, we obtained high–temporal resolution cine images of LV horizontal and vertical long axes using an ECG-triggered b-SSFP sequence (TR = 22.4 ms, TE = 1.32 ms, 64 phases, matrix = 192 × 156, in-plane resolution = 1.7 × 1.7 mm^2, slice thickness = 6 mm, imaging time = 12–19 s per slice). Furthermore, we acquired a stack of contiguous short axis slices (slice thickness = 7 mm, interslice gap = 4 mm) by cine imaging (TR = 36.2 ms, TE = 1.39 ms, 30 phases, matrix = 256 × 184, in-plane resolution = 1.4 × 1.4 mm^2, about 13 slices with imaging time = 7–10 s per slice), covering both right and left ventricles. Image acquisition at FRC is the common way in cardiac MRI and is used as baseline for comparison with the image acquisition during apnea.

Part 2 (image acquisition during three subsequent apnea periods at TLC): During the first apnea, we planned LV long- and short-axes imaging in a standardized fashion. The second apnea started with anatomic thorax imaging. After HR had stabilized, we began continuous short axis cine image acquisition. During the third apnea, we performed alternating vertical and horizontal long-axes continuous cine imaging. Imaging was aborted by a signal from the diver or when incipient involuntary diaphragm contractions began causing motion artifacts.

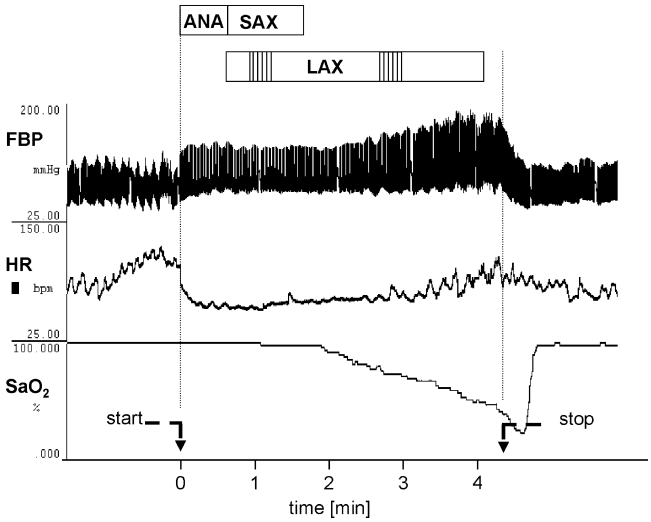

FIGURE 1—Finger blood pressure (FBP), HR, and arterial oxygen saturation level (Sao_2) recorded during apnea with glossopharyngeal insufflation in one diver during the prescanning training session. Apnea started at time point 0. The timing for anatomic imaging (ANA), long-axis (LAX), and short-axis (SAX) cine MRI is illustrated. *Shaded areas* represent the time windows chosen for the LAX image analysis of dynamic changes during apnea, i.e., minutes 1 and 3.

Part 3 (image acquisition during three subsequent periods of apnea with glossopharyngeal insufflation) was performed in the same way like part 2. We performed imaging during apnea without (part 2) and with glossopharyngeal insufflation (part 3) in randomized order. Figure 1 illustrates the timing of MRI during apnea with and without glossopharyngeal insufflation. Because rapid initial HR and BP changes and involuntary breathing movements later on can interfere with image acquisition during apnea, we analyzed images obtained 1 and 3 min into apnea.

Image analysis. We estimated lung volumes after manually contouring pulmonary borders excluding heart and mediastinal structures in contiguous thorax images. We assessed RV and LV structure and function in short-axis images by manually drawing endocardial contours in end-diastole and end-systole using dedicated software (MASS7.1; Medis AG, Leiden, The Netherlands) (19). In both ventricles, we quantified end-diastolic volume, end-systolic volume, stroke volume, ejection fraction, and cardiac output. For the assessment of dynamic changes throughout apnea, we evaluated end-diastolic and end-systolic images in horizontal and vertical long axes. We calculated LV parameters from subsequent long axes using a biplane area–length model (5). Two experienced observers analyzed images according to consented guidelines for image analysis.

Statistics. All values are given as mean ± SD unless noted otherwise. We tested absolute differences in ventricular volumes by one-way ANOVA for repeated measurements followed by *post hoc* Bonferroni tests. We used paired *t*-tests to compare relative changes in ventricular volumes and cardiac output calculated from short-axis cine MR images during apnea with and without glossopharyngeal insufflation. When baseline measures were not normally distributed, we applied Mann–Whitney rank sum tests. We assessed normal Gaussian distribution using Kolmogorov–Smirnov tests, with natural logarithmic transformations completed, as required. Furthermore, we applied two-way repeated-measures ANOVA to assess differences in biplane LV volumes between apnea with and without glossopharyngeal insufflation over time (minute 1 vs minute 3). Statistical significance was set at $P < 0.05$. For all these tests, we applied Prism 5 for Windows (GraphPad Software, Inc., San Diego, CA).

RESULTS

We included 10 divers (9 men, 1 woman) in the statistical analysis. One diver had to be excluded based on insufficient glossopharyngeal insufflation and an apnea time of <3 min. Figure 2 shows end-diastolic MR images in the horizontal long axis and midventricular short axis at baseline (FRC), at 1 min during apnea, and at 1 min during apnea with glossopharyngeal insufflation. Lung volume increased during glossopharyngeal insufflation by 1.16 L in this diver. Shape and diameters of both ventricles changed profoundly after lung hyperinflation with and without glossopharyngeal insufflation. Lung volume was 3.7 ± 0.8 L at baseline, 8.7 ± 1.1 L at TLC, and 9.4 ± 1.4 L after glossopharyngeal insufflation ($P < 0.05$, apnea vs glossopharyngeal insufflation). Mean lung volume increased by 0.7 ± 0.4 L during submaximal glossopharyngeal insufflation. HR increased from 67 ± 13 bpm at baseline to 83 ± 14 bpm at minute 1 of apnea and to 84 ± 21 bpm at minute 1 during glossopharyngeal insufflation.

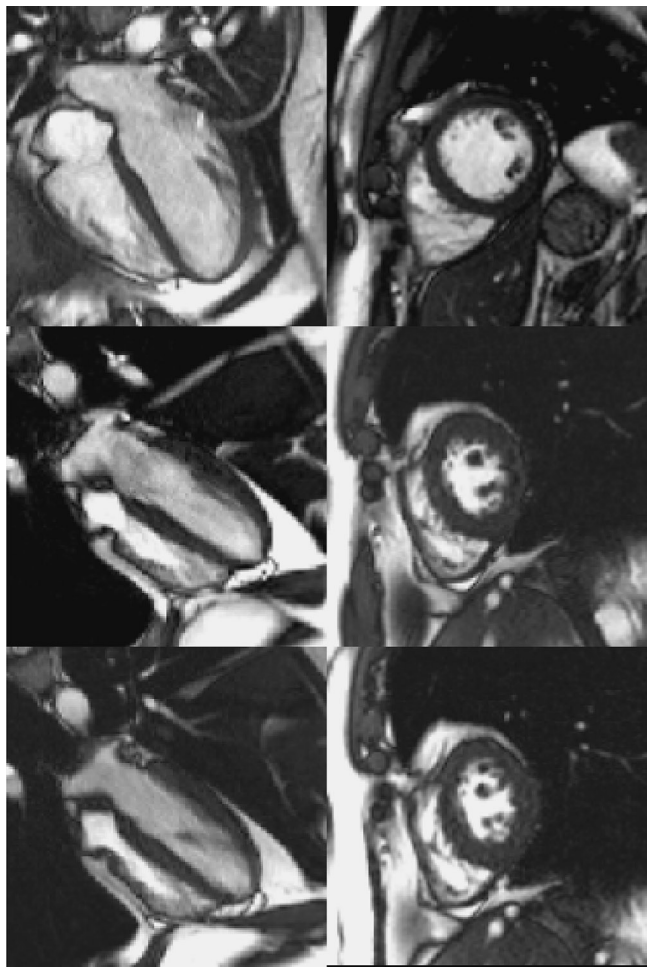

FIGURE 2—Representative end-diastolic MR images in the LV horizontal long axis (*left*) and midventricular short axis (*right*) during baseline (FRC, *top*), 1 min of apnea (TLC, *middle*), and 1 min of apnea with glossopharyngeal insufflation (*bottom*). Lung volume increased during glossopharyngeal insufflation by 1.16 L in this diver. Note the profound change of shape and diameters of both ventricles after lung inflation with and without glossopharyngeal insufflation.

Apnea influences on ventricular size and function. LV and RV cardiac output were 8.2 ± 1.4 and 8.0 ± 1.4 $L \cdot min^{-1}$ at baseline (FRC). For both ventricles, cardiac output decreased 29% ± 12% with apnea. Ejection fraction responded similarly for both ventricles (left = −5% ± 5%, right = −7% ± 5%, $P < 0.01$) during apnea. The findings suggest that the major effect on the heart seen during maximum inspiratory apnea is due to decreased venous

TABLE 1. LV and RV volumes, cardiac output, and ejection fraction during breath hold with and without glossopharyngeal insufflation ("lung packing") derived from ECG-triggered cine MR short-axis images.

Parameter (Unit)	Baseline (FRC)	Apnea without Packing	Apnea with Packing
HR (bpm)	67 ± 13	83 ± 14*	84 ± 21*
LVEDV (mL)	198 ± 19	125 ± 38*	108 ± 26*
LVESV (mL)	75 ± 11	55 ± 23*	47 ± 11*
LVSV (mL)	123 ± 16	70 ± 17*	62 ± 19*
LVCO ($L \cdot min^{-1}$)	8.2 ± 1.4	5.6 ± 0.8*	4.9 ± 0.8*
LVEF (%)	62 ± 4	57 ± 6*	56 ± 7*
RVEDV (mL)	225 ± 30	148 ± 37*	136 ± 29*
RVESV (mL)	104 ± 23	79 ± 25*	76 ± 20*
RVSV (mL)	121 ± 17	69 ± 16*	60 ± 19*
RVCO ($L \cdot min^{-1}$)	8.0 ± 1.4	5.6 ± 0.8*	4.7 ± 0.8*
RVEF (%)	54 ± 6	47 ± 7*	44 ± 10*

Baseline images were obtained at the level of FRC. Images during apnea were obtained after about 1 min of apnea.

* $P < 0.001$ compared to baseline, mean ± SD, one-way ANOVA for repeated measurements, Bonferroni *post hoc* test.

EDV, end-diastolic volume; ESV, end-systolic volume; SV, stroke volume; CO, cardiac output; EF, ejection fraction.

return, decreased end-diastolic volume, and decreased stroke volume.

Glossopharyngeal insufflation influences on ventricular size and function. Cardiac output further decreased with glossopharyngeal insufflation (left = −39% ± 13%, right = −40% ± 14%, $P < 0.05$). The decrease in ejection fraction was more pronounced for the right ventricle during apnea with glossopharyngeal insufflation compared to that for the left ventricle (left = −6% ± 5%, right = −10% ± 6%, $P < 0.05$ vs apnea). The additional decrease in end-diastolic volume and stroke volume with glossopharyngeal insufflation was rather small and did not reach significance compared with apnea without glossopharyngeal insufflation. Absolute ventricular volume, cardiac output, and ejection fraction values at baseline and during apnea with and without glossopharyngeal insufflation are given in Table 1. Figure 3 summarizes relative changes in ventricular volumes, cardiac output, and ejection fractions in comparison to baseline values obtained at FRC.

Cardiac performance during apnea (long-axis MRI). We compared HR, LV volumes, ejection fraction, and cardiac output at two time points (minute 1 and minute 3) during apnea with and without glossopharyngeal insufflation (Table 2). Two-way ANOVA for repeated measures showed a highly significant effect for timing ($P < 0.001$), indicating partial end-diastolic volume and cardiac output recovery with continued apnea. No effect for glossopharyngeal insufflation and no interaction between timing and glossopharyngeal insufflation were found. During apnea without glossopharyngeal insufflation, mean HR tended to be higher at 3 min of apnea compared with 1 min of apnea. Mean stroke volumes were similar at both time points as was ejection fraction.

DISCUSSION

The major finding of our MRI study in diving athletes was a substantial decrease in RV and LV volumes and cardiac output with maximal inspiratory apnea. Submaximal glossopharyngeal insufflation decreased ventricular volumes and cardiac output further albeit by a small amount. RV end-diastolic volume remained reduced under these conditions. In contrast to earlier findings (36,37), LV or RV enlargement did not occur. However, RV ejection fraction decreased more than LV ejection fraction, emphasizing the well-known higher sensitivity of the RV to acute after-load increases (14). Cardiac MRI late into apnea was limited by motion artifacts elicited by involuntary breathing movements, as well as by HR changes and premature beats. Nevertheless, comparing LV volumes and cardiac output measurements 1 and 3 min into apnea showed an extremely significant time effect with and without glossopharyngeal insufflation supporting the hypothesis of a partial recovery. Together, our findings challenge the idea that lung hyperinflation induces severe reduction of ejection fraction. Instead, submaximal glossopharyngeal insufflation seems to decrease systemic venous return more than apnea alone and slightly reduces RV filling. Furthermore, the sudden pulmonary venous resistance increase during glossopharyngeal insufflation further augments RV after-load subsequently, decreasing RV stroke volume through the Frank–Starling mechanism (12,38). Changes in ventricular geometry may also contribute to the cardiac output reduction.

Our data are in contrast to earlier findings during apnea with and without glossopharyngeal insufflation obtained by ECG or MRI (36,37). In one study, subcostal single-plane echo images were obtained during a 30-s glossopharyngeal insufflation maneuver. The investigators quantified biventricular volumes and function using Simpson's rule. They

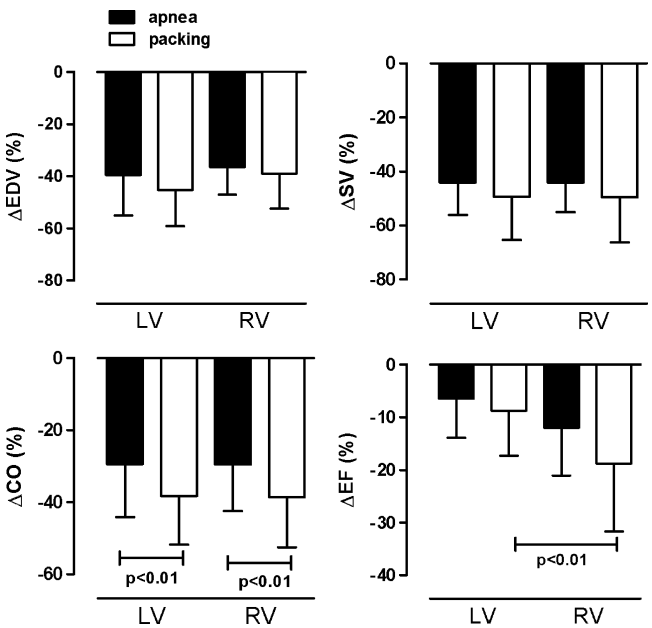

FIGURE 3—Relative ventricular volume, cardiac output, and ejection fraction changes at about 1 min of apnea with (packing) and without glossopharyngeal insufflation (apnea) versus baseline at FRC calculated from short-axis cine MRI.

TABLE 2. HR, LV volumes, cardiac output, and ejection fraction during breath hold with and without glossopharyngeal insufflation ("lung packing") derived from ECG-triggered cine MR long-axes images.

	Apnea without Packing		Apnea with Packing	
Parameter (Unit)	**1 min**	**3 min**	**1 min**	**3 min**
HR (bpm)	80 ± 15	87 ± 16	87 ± 23	88 ± 18
LVEDV (mL)	112 ± 15	125 ± 15*	108 ± 26	120 ± 20*
LVSV (mL)	69 ± 12	69 ± 8	63 ± 22	68 ± 14
LVCO ($L \cdot min^{-1}$)	5.4 ± 1.9	6.0 ± 1.2*	5.0 ± 1.2	5.8 ± 1.2*
LVEF (%)	61 ± 7	56 ± 6	57 ± 8	56 ± 6

Images were obtained at 1 and at 3 min of apnea.
* $P < 0.001$ compared to baseline, mean ± SD, one-way ANOVA for repeated measurements, Bonferroni *post hoc* test.

observed severe biventricular dysfunction and described a 160% increase in RV end-diastolic volume during apnea (37). However, RV volumes were severely underestimated already at rest such that calculated RV cardiac output was below 1 $L \cdot min^{-1}$. Rotational asymmetry particularly of the right ventricle and limited acoustic access during lung inflation underline the need for an imaging technique with free access to any imaging plane and model free quantification. Furthermore, the timing of their measurements in the phase of unstable hemodynamics, i.e., in the first 30 s after packing, may also have contributed to the discrepancies with our findings. Another study applied MRI (32) to explore the cardiovascular response to prolonged breath hold. The authors describe a steady increase in LV size and decrease in LV ejection fraction during apnea. However, the initial image acquisition was conducted early during apnea, which is characterized by rapid changes in HR and blood pressure (Fig. 1). Comparisons between measurements during this dynamic phase and measurements obtained later during apnea may overestimate the increase in LV size. Our data consistently show that the size of both ventricles is, indeed, reduced during apnea. However, the subsequent cardiac output recovery with sustained apnea is small compared with the initial cardiac output decrease. We did not observe RV enlargement in cine images up to 3 min of breath holding, thus excluding acute failure of the RV over time. Moreover, abnormal septal movements toward the left ventricle or other features of ventricular interdependence were absent.

Prolonged apnea with glossopharyngeal insufflation may have deleterious effects on the lung (4,22), the heart (1,23), and the brain (2,29) and may cause frank syncope (1,6). Therefore, the exact knowledge about the right dosing of glossopharyngeal insufflation, which is safe in individual divers, is essential. Our study showed that submaximal glossopharyngeal insufflation does not profoundly decrease ventricular volumes and cardiac output compared with apnea without glossopharyngeal insufflation. The absolute changes in ejection fractions versus baseline were only 6% for the left ventricle and 10% for the right ventricle. A recent study used velocity-encoded MRI and measured pulmonary artery blood flow in divers during glossopharyngeal insufflation (8). The authors observed a 40% decrease in RV output compared with baseline, which is in accordance with our findings. All hemodynamic changes during repeated glossopharyngeal insufflation were completely reversible in this velocity-encoded MRI study.

Previously, we and others followed cardiac output changes during maximum apnea with and without glossopharyngeal insufflation (10,15,16) using impedance cardiography. All methods struggle with the extreme changes in thorax geometry, the tremendous amount of air in the lungs, and the dramatic HR and blood pressure changes during prolonged apnea. Each method has specific limitations and advantages. ECG images are taken during FRC breath holding to avoid motion artifacts and echo-dense air. The equations applied for stroke volume calculations from subcostal or four-chamber view images rely on the preserved geometry of the ventricle, which is not the case during apnea. MRI is the only method providing the possibility to cover both ventricles completely during maximum apnea with the highest accuracy and the lowest interobserver and intraobserver variability (20,21,31). All methods are disturbed by involuntary breathing movements during late apnea phases. New imaging technologies obtaining images faster and possibly independent from breathing movements might overcome some of these limitations (18,24,25,35,41).

The more pronounced RV ejection fraction decrease suggests that maximum glossopharyngeal insufflation may indeed attenuate ventricular performance. We studied submaximal glossopharyngeal insufflation and can only speculate on maximum glossopharyngeal insufflation volumes. Recent findings suggest that syncope during glossopharyngeal insufflation is commonly initiated through bradycardia and asystole with subsequent cardiac output reduction and not *vice versa* (1,6).

The initial decreases in ventricular volumes and cardiac output are likely explained by the increased intrathoracic pressure, leading to decreased intrathoracic blood volume, intrathoracic vessel compression, and reduced venous return. We speculate that diving response–induced bradycardia, massive sympathetic activation, and hypoxia-driven chemoreflex activation could contribute to the partial cardiac output recovery from minute 1 to minute 3 during apnea. In some divers with subsequent HR decreases and increased end-diastolic volume and end-systolic volume, partial cardiac output recovery may result from increased venous return caused by involuntary breathing movements. In others, partial cardiac output recovery may result from an HR increase. Therefore, our results on the increase in LV cardiac output

support the findings from earlier studies (36), but the increase is small. Because cardiac MRI is not a real-time imaging technique, motion artifacts elicited by involuntary breathing movements (Fig. 1), rapid HR changes, and premature beats made it impossible to cover phases beyond 3 min of apnea with appropriate image quality. This so-called struggling phase is physiologically interesting because divers become increasingly hypoxemic and hypercapnic. The response is associated with massive sympathetic activation (15,16).

One limitation of our study is that we did not control glossopharyngeal insufflation volume and tested only submaximal glossopharyngeal insufflation. We used the sagittal scout images of the chest to estimate lung volumes. The measured lung volume may not completely reflect the real glossopharyngeal insufflation volume. At higher glossopharyngeal insufflation volumes, the transpulmonary pressure may increase without further changes in volume simply by compression and decreasing chest compliance (39). We did not measure transpulmonary pressure in our study. Therefore, the amount of gas in the lungs after glossopharyngeal insufflation may be underestimated. Divers usually train glossopharyngeal insufflation during water immersion. Water immersion augments venous return, increases cardiac output (3,11), and the hemodynamic responses are affected by partial or total water immersion and by water temperature (9). Therefore, our results cannot be extrapolated to water immersion.

We conclude that lung hyperinflation through submaximal glossopharyngeal insufflation further reduces cardiac output by a small amount compared with the profound reduction during apnea without glossopharyngeal insufflation while biventricular ejection fraction is maintained. Our findings in divers indicate that from a hemodynamic point of view, submaximal glossopharyngeal insufflation is safe perhaps until an individual safety limit is attained. We suggest that individual hemodynamic tolerance to lung hyperinflation should be taken into account in divers practicing glossopharyngeal insufflation. Cardiovascular monitoring might be used to avoid hypotension and syncope during glossopharyngeal insufflation, particularly in inexperienced individuals.

The first two authors contributed equally to the study.
The authors are not aware of conflicting interests.
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The results of the present study do not constitute endorsement by the American College of Sports Medicine.

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