Cardiovascular changes during underwater static and dynamic breath-hold dives in trained divers

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1Department of Integrative Physiology, University of Split School of Medicine, Split, Croatia; 2Real-Time Systems Laboratory, Scuola Superiore Sant’Anna, Pisa, Italy; 3Department of Internal Medicine, University Hospital Split, Split, Croatia; and 4The Imego Institute, Gothenburg, Sweden

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Breskovic T, Uglesic L, Zubin P, Kuch B, Kraljevic J, Zanchi J, Ljubkovic M, Sieber A, Dujic Z. Cardiovascular changes during underwater static and dynamic breath-hold dives in trained divers. J Appl Physiol 111: 673–678, 2011. First published June 30, 2011; doi:10.1152/japplphysiol.00209.2011.—Limited information exists concerning arterial blood pressure (BP) changes in underwater breath-hold diving. Simulated chamber dives to 50 m of freshwater (mfw) reported very high levels of invasive BP in two divers during static apnea (SA), whereas a recent study using a noninvasive subaquatic sphygmomanometer reported unchanged or mildly increased values at 10 m SA dive. In this study we investigated underwater BP changes during not only SA but, for the first time, dynamic apnea (DA) and shortened (SHT) DA in 16 trained breath-hold divers. Measurements included BP (subaquatic sphygmomanometer), ECG, and pulse oxymetry (arterial oxygen saturation, SpO2, and heart rate). BP was measured during dry conditions, at surface fully immersed (SA), and at 2 mfw (DA and SHT DA), whereas ECG and pulse oxymetry were measured continuously. We have found significantly higher mean arterial pressure (MAP) values in SA (~40%) vs. SHT DA (~30%). Postapneic recovery of BP was slightly slower after SHT DA. Significantly higher BP gain (mmHg/duration of apnea in s) was found in SHT DA vs. SA. Furthermore, DA attempts resulted in faster desaturation vs. SA. In conclusion, we have found moderate increases in BP during SA, DA, and SHT DA. These cardiovascular changes during immersed SA and DA are in agreement with those reported for dry SA and DA.

arterial pressure; pulse oximetry; lactates; underwater measurement; human

CARDBOVASCULAR CHANGES during dry static breath-hold include bradycardia (3), reduced cardiac output, and increased arterial blood pressure (BP) caused by vasoconstriction of selected vascular beds (7, 14, 20) due to increased sympathetic efferent nerve activity toward skeletal muscle (11, 14). The purpose seems to be redistribution of the reduced cardiac output to heart and brain in conjunction with reduced overall oxygen consumption and work of heart. During dynamic cycle exercise, diving-induced bradycardia is powerful enough to override the exercise tachycardia for the period of apnea (3, 5, 26, 27). The cardiac output is reduced throughout apneas during exercise, largely due to the bradycardia, whereas the systemic vascular resistance increases (6).

BP changes during dry breath-hold diving have been recorded in the past with different measuring devices (for invasive measurement with arterial line, whereas for noninvasive a standard arm sphygmomanometer, a Korotkoff sound-based automatic sphygmomanometer, a photoplethysmograph, and an aneroid manometer) and under different experimental conditions (supine apnea at rest or during exercise, with or without the subject’s face immersed in cold or thermoneutral water). Under all of these circumstances, moderate increases in BP were found with an augmented response to face immersion. The only study that measured invasive BP during deep breath-hold dives was the study of Ferrigno et al. (8). Those authors have measured BP response in two elite divers that were compressed in the wet compartment of the chamber to 50 m of freshwater (mfw). The highest values of BP (systolic BP 280–300 mmHg and diastolic BP 150–200 mmHg) were measured during 10–20 mfw descent. Contrary to the study of Ferrigno et al. (8), Sieber et al. (25) have recently measured BP at 10 mfw with a novel noninvasive subaquatic sphygmomanometer in elite breath-hold divers (BHD) during the 2nd minute of SA. They have reported unchanged BP values when compared with values obtained at surface. However, one must note that, in contrast with the continuous invasive BP measurements in the Ferrigno et al. (8) study, the BP was measured only once directly after the subjects arrived at 10 mfw within approximately the 1st minute of apnea (diving method: variable weight, descent speed ~1 mfw/s). Recently, Perini et al. (22) reported continuous BP changes with photoplethysmography (finger BP with the transducer outside the water) during prolonged immersed SA below the water surface. They reported at the end of SA significantly increased systolic (193 mmHg) and diastolic (127 mmHg) BP. Thus reported BP changes during immersed SA are controversial, ranging from unchanged to very high values. No data exist for underwater BP measurement during DA. This state of affairs is disturbing because healthy people, including underwater hockey players, synchronized swimmers, and elite BHD, practice voluntary apnea on a regular basis. Divers are only an extreme example for voluntary apnea.

Thus, the primary aim of this study was to measure BP changes BHD in fully immersed conditions during SA and for the first time during DA and shortened (SHT) DA in 16 trained BHD. SHT DA attempt was shortened for the duration of time needed to obtain a single underwater BP measurement (50–60 s). The second aim was to investigate continuous HR and pulse oximetry (SpO2) and pre- vs. post blood lactate changes during same dives. We also analyzed not only the cardiovascular changes during apneas but also the recovery phase, which was rarely investigated in the past, especially during maximal end-inspiratory long apneas.
HEMODYNAMICS DURING IMMERSED STATIC AND DYNAMIC APNEA

METHODS

Subjects. We recruited 16 elite BHD (14 men and 2 women), and two of the subjects were recent world champions in DA. All experimental procedures in the study were completed in accordance with the Declaration of Helsinki and approved by Ethics Committee of the University Of Split School Of Medicine. All subjects provided written consent to take part in this study. Anthropometric characteristics, pulmonary function data, and diving history of the subjects are shown in Table 1.

Study protocol. Protocol was conducted in 3 consecutive days. The subjects were instrumented with various underwater equipment, allowing us to measure arterial BP, electrocardiography (ECG), SpO2, and heart rate (HR). Furthermore, blood lactate concentration was measured before and after apnea attempts.

Assessment of arterial BP. Arterial BP was obtained using special subaqueous sphygmomanometer, already explained in detail previously (24). In brief, this instrument has an upper arm cuff that is automatically inflated through an electromagnetic valve with air supplied from a SCUBA tank. The device consists of a microcontroller, a differential pressure sensor, and a display. The electronic parts are situated in a waterproof housing that is mounted directly on the cuff. It can withstand pressures up to 10 bar. The calculation of systolic and diastolic pressure is based on the oscillometric method. The measurement of BP is manually initiated, and the time required for obtaining one BP measurement with subaqueous sphygmomanometer is ~50–60 s.

ECG/SpO2 underwater unit. ECG, SpO2, and HR were measured with a data logger specially designed for underwater applications (12). Two-channel ECG was sampled at 250 Hz. SpO2 and HR were measured with a reflective sensor probe. All measurement data plus additional water pressure and temperature were stored on a SD card in text file format (FAT16). All data were processed in real time. The electronics plus a battery pack were encapsulated in a lexan housing (waterproof and pressure proof up to 20 bar).

We used this device in 10 of 16 subjects. For the remaining subjects we used a custom-made device based on similar technology for SpO2, except for having ECG capability (13).

HR was derived from ECG in subjects who had ECG recordings; in the rest of the subjects, HR was derived from SpO2 plethysmography recordings.

Assessment of blood lactate concentration. Blood lactate concentration was obtained with a portable hand-held analyzer (Accutrend Lactate; Roche, Mannheim, Germany). Before and after each apnea attempt, divers’ fingers were cleaned and dried with a towel to avoid any influence of water on the results. Blood drop was taken from the fingertip and was put on a test strip (BM-Lactate; Roche).

On the 1st day of the study, upon arrival to the laboratory, subjects were informed about procedures and potential risks. After giving their written informed consent to participate in the study, anthropometric measurements and dynamic spirometry (Quark PFT; Cosmed, Rome, Italy) were obtained from each subject. Experiments took place in an indoor swimming pool over the following 2 days. The pool size was 25 m in length and 2 m in depth. Air temperature in the facility was 26.8 ± 1.5°C, and pool water temperature was 26.9 ± 1.9°C. BHD wore 5-mm-thick neoprene wetsuits without left sleeves, which enabled placement of the upper arm cuff for measurement of BP. The suit served to prevent hypothermia and to fixate the underwater ECG/SpO2 device housing beneath it. Additionally, subjects wore a diving mask, which was used for fixating pulse oximeter sensor on the temporal region of the head.

The subjects were instrumented next to the pool, and the equipment was tested for malfunction. Once the continuous measurement of ECG/SpO2 was switched on, the stopwatch was started as well. Thus we were able to enter arterial BP values and blood lactate concentration measured at the noted time points and afterward to time-align changes in these parameters with changes measured with ECG/SpO2 underwater unit.

On the 2nd day of the protocol, subjects performed two SA. They did it by floating in supine position on the surface of the water. Upon the pre-assumption for the apnea being finalized, they were allowed to take a couple of deep breaths prior inhalation to total lung capacity. Following that, they would turn in the prone position with their head submerged in the water and hold their breath for as long as possible. During SA protocol ECG/SpO2 measurements were continuously monitored, whereas BP measurements were manually initiated consecutively every minute. The measurements continued until normalization of these parameters. Blood lactate concentration was measured before SA and every 3 min during the recovery period until the increase in lactate levels would plateau.

After SA protocol, each subject would exit the water to rest and to enable downloading of recorded data from underwater devices. After 15-min resting period, they would enter the water again and do one maximal DA. The divers would undergo a short preparation for the DA, which was similar to the one prior the SA. After inhaling to total lung capacity, they would submerge at the bottom of the pool and start to swim the longest possible distance. Following the surfaceing they would rest by the edge of the pool, thus allowing BP and blood lactate measurements. As previously described, ECG and SpO2 were measured continuously, whereas BP measurements were done at the baseline and consecutively every minute during recovery phase until normalization. Blood lactate levels were measured similarly as in SA protocol.

On the 3rd day of the protocol, divers performed SHT DA. The maximal duration of the apnea was determined the day before and was shortened for the duration of time needed to obtain one underwater BP measurement (50–60 s). Subjects performed SHT DA similarly as maximal DA, except they would stop swimming at the previously determined spot and would remain submerged at the bottom of the pool while still holding their breath. At that point, they would meet with one of our investigators waiting to take underwater BP measurements using SCUBA gear. After the BP measurement was finalized the diver would surface by the edge of the pool, and further measurements of BP and lactates were done during the recovery period similarly as the day before.

Data analysis. BP measurements were taken before the beginning of the apnea attempt, continuously every 50–60 s during SA, at the end of SHT DA, and every minute through the 5-min recovery period after all trainings of HR and SpO2 were averaged during the 1-min baseline period, during each 20% interval of the relative duration of the apneas, and during last 30-s period in the 1st min of recovery.

Statistical analysis. Results were expressed as means ± SD. To compare changes of variables before, during, and after each apnea, we used ANOVA for repeated measurements. In case of significant difference, Bonferroni test was applied as post hoc test for specific comparison. Values at the same time points in SA and DA were

<table>
<thead>
<tr>
<th>Table 1. Anthropometric characteristics of subjects</th>
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<tbody>
<tr>
<td>Divers (n = 15)</td>
</tr>
<tr>
<td>Age, yr</td>
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<tr>
<td>Height, cm</td>
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<tr>
<td>Weight, kg</td>
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<tr>
<td>BMI</td>
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<td>FVC, %predicted</td>
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<tr>
<td>FEV1, % predicted</td>
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<tr>
<td>FEV1/FVC, %</td>
</tr>
<tr>
<td>Years practicing apnea, yr</td>
</tr>
<tr>
<td>Personal best static apnea, s</td>
</tr>
<tr>
<td>Personal best dynamic apnea, m</td>
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<tr>
<td>Time since last training, days</td>
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</table>

Values are means ± SD. BMI: body mass index; FVC: forced vital capacity; FEV1, forced expiratory volume in the 1st second.
compared using a paired Student *t*-test. The slopes representing change in BP and SpO$_2$ over time were calculated using linear regression. Statistical significance was set at *P* = 0.05. All analyses were performed with Statistica 7.0 software (Statsoft, Tulsa, OK).

**RESULTS**

Although all 16 divers successfully completed 3 days of the experimental procedures, we acquired good quality recordings from 15 of 16 divers. On average the divers endured 244 ± 63 s of SA. SHT DA lasted on average 105 ± 21 s. Out of total apnea time for SHT DA 40 ± 25 s (36 ± 14%) accounted for underwater swimming, during which the divers swam an average length of 37 ± 18 m. The divers spent the rest of the total apnea time resting on the bottom of the pool for BP measurement. Duration of maximal DA was 82 ± 27 s, during which the divers swam an average length of 74 ± 20 m. The average swimming speed was 0.9 ± 0.2 m/s, ranging from 0.6 to 1.4 m/s.

**Arterial BP.** Baseline mean BP [mean arterial pressure (MAP)] was 94 ± 10 mmHg; systolic blood pressure (SBP) was 129 ± 15 mmHg, and diastolic blood pressure (DBP) was 78 ± 9 mmHg. Figure 1 shows changes in MAP, SBP, and DBP at the baseline, during SA, SHT DA, and DA, and during 5 min of recovery. Throughout SA there was a continuous increase in SBP, DBP, and MAP, reaching their peaks toward the end of the apnea. The observed maximal increase in these variables ranged between 30 and 42% and was significantly higher compared with baseline values (*P* = 0.001, *P* = 0.001, and *P* = 0.002, respectively). Within 3 min of recovery, BP values returned to baseline.

At the end of SHT DA, SBP, DBP, and MAP significantly increased to ~30% compared with baseline and were normalized in the 4th min of recovery. The maximal reached value in arterial BP was lower compared with a maximal increase observed in SA (SBP 166 vs. 157 mmHg, *P* = 0.051; DBP 110 vs. 100 mmHg, *P* = 0.016; MAP 129 vs. 119 mmHg, *P* = 0.014).

In the 1st min of recovery after maximal DA, the increase in SBP was ~44% and in DBP was ~22%, whereas MAP was increased ~32% compared with baseline. MAP normalized in the 10th min of recovery. SBP in the 6th min of recovery, and DBP in the 4th min of recovery. Values of SBP and MAP after DA compared with corresponding time points in recovery after SA were significantly higher during the 4-min recovery period. DBP in 4th and 5th min of recovery after DA was higher compared with those values measured in analogous recovery time points after SA.

The slopes representing gain in SBP, DBP, and MAP during SA were significantly lower compared with corresponding slopes for SHT DA (Fig. 2). The increase in these parameters was 63–100% faster in SHT DA.

**HR and SpO$_2$.** Changes in HR and SpO$_2$ during SA, SHT DA, and DA are presented in Table 2. At the beginning of the SHT DA and DA, there was a significant increase in HR that was not present in SA. During SHT DA and DA, HR was significantly higher compared with the same time points in SA. However, in all three types of apnea a reduction in HR toward the end of apnea was observed. The most profound reduction in SpO$_2$, as expected, was seen in SA and DA. The level of desaturation was similar between these two apnea disciplines. Nevertheless, the average slope representing the speed of reduction in SpO$_2$ was ~2.5 times steeper in DA compared with SA (Fig. 3). The SpO$_2$ began to decrease toward the end of apnea, and it reached a minimum value ~15–20 s after the end of apnea in all conditions.

**Blood lactate concentration.** Average baseline blood lactate concentration was 1.7 ± 0.5 mmol/l. Lactate concentration increased significantly after all three types of apnea (Table 3). However, the increase was highest after DA (*P* < 0.001). In addition, a significant positive correlation (*r* = 0.702, *P* = 0.003) was observed between swimming speed and level of the blood lactate concentration. Furthermore, swimming speed during DA and SHT DA inversely correlated with the duration of static apnea (*r* = −0.6, *P* = 0.016). On average, lactate concentration reached its maximal value within 6 min after apnea. There was no difference in time needed to achieve maximal lactate concentration among various types of apnea.

**DISCUSSION**

This study shows that, during fully immersed SA, SHT DA, and DA, MAP is increased to a similar magnitude as that reported previously by us and others in dry conditions (~25–45%). After resumption of breathing after SA, BP returns to
control values in the 1st min, suggesting fast cardiovascular recovery, even when SA lasted for ~4 min. During SHT DA (~35–40 s DA, followed by ~50–60 s SA), a slightly steeper increase in BP was found when expressing data compared with same time point during SA, suggesting exaggerated hypertensive response. Arterial desaturation occurred faster during maximal DA, and the largest postdive increase in lactate was observed as expected due to the simultaneous combination of apnea and dynamic exercise (underwater fin swimming).

Elite BHD are exposed to extreme chemoreflex stimulation (hypoxia/hypercapnia) during maximal SA and DA. Following maximal apneas, alveolar oxygen partial pressure can be reduced to 20–30 mmHg, with arterial oxygen saturation around 50% (17, 19). These perturbations in arterial blood gases cause massive chemoreflex stress, which presents a major drive force for the increase in arterial BP. At the end of the dry maximal end-inspiratory apnea, muscle sympathetic nerve activity is increased by about 2,000%, predominantly because of this extreme chemoreflex stimulation (11).

BP values measured during immersed breath-hold diving are scarce and controversial and range from extremely high (8) to unchanged or mildly increased (25). Large increases of BP reported by Ferrigno et al. (8) could have potentially deleterious effects and result in serious acute events such as pulmonary edema, myocardial infarction, retinal hemorrhage, and stroke. Except for the potentially negative effects of increased arterial BP during apnea on the human organism, recent studies have shown that pulmonary squeeze due to increased hydrostatic pressure and profound hypoxia in trained BHD may result with postapneic pulmonary edema and hemoptysis (16), frequent loss of motor control, or loss of consciousness during competition (10) and increased serum levels of brain damage marker S100B protein (2), suggesting disturbed blood-brain barrier. This suggests that, together with previously described acute complications of extreme breath-holding, an abrupt high increase in arterial BP may represent an additional acute risk factor during apnea.

However, we have found only moderate increases in SBP, DBP, and MAP during fully immersed SA of a similar magnitude to previously collected data during dry conditions (20, 21). We did not record any alarming levels of BP as shown by Ferrigno et al. (8), although in some subjects during SA SBP increased up to 207 mmHg and DBP increased up to 112 mmHg. Perini et al. (22) have recorded, by using continuous a finger BP device (Portapres; Finapres Medical Systems, Amsterdam, The Netherlands), slightly higher average BP values (SBP 193 and DBP 127 mmHg) in SA than we did (SBP 166 and DBP 110 mmHg). However, since their data were obtained from the peripheral artery, SBP and MAP may have been slightly overestimated.

The slopes representing gain in SBP, DBP, and MAP during SHT DA were significantly greater compared with corresponding slopes for SA, indicating exaggerated hypertensive response during dynamic apnea disciplines. SHT DA was comprised of rather short DA lasting around 30 s and short SA lasting around 50–60 s for a single BP measurement, suggesting that measured BP increase would be even greater during maximal DA. Additionally, even 1 min after the end of maximal DA, average SBP was still 185 mmHg, and thus values close to 200 mmHg were most probably present at the end of DA.

BP recovery after SA was very fast, returning to the baseline values within 3 min, whereas much slower recovery was found after maximal DA. MAP was still increased after 5 min postmaximal DA, normalizing in the 10th min of the recovery.

As expected, much faster arterial desaturation and larger postdive lactate increase was noted after maximal DA compared with SA due to concurrent apnea and dynamic exercise.

Fig. 2. Bar graphs comparing gain over apnea duration in SBP, DBP, and MAP during SA and SHT DA. Values are presented as means ± SD. *P < 0.05.
However, at the end of both disciplines, arterial oxygen saturation reduced to similar levels of SpO2. This suggests that the divers may have reached similar levels of chemoreflex stress (at least hypoxic) in both apnea disciplines. There was a continuous lag of 15–20 s between the end of apnea and the time point in which the minimal value in SpO2 was achieved. The nadir SpO2 value corresponds to the end of apnea but was delayed probably because of the circulation time between the lungs and the forehead.

Additionally, we have found different strategies used by divers attempting to dive the longest possible distance. Those divers that had ability to endure the longest SA duration used slower swimming speed, thus taking advantage of their better SA performance. On the other hand, the divers that used faster swimming speed, thus taking advantage of their better endurance, caused increased resistance for movement through the water. The cause for that discrepancy may be caused by factors already mentioned for SA. Moreover, in our study we used a 5-mm-thick neoprene diving suit, which is not fully suitable for the suit, together with scientific equipment, divers wore 3–4 kg of additional weights to keep neutral buoyancy. These mentioned for SA. Moreover, in our study we used a 5-mm-thick neoprene diving suit, which is not fully suitable for fin swimming. Because of increased buoyancy caused by the suit, together with scientific equipment, divers wore 3–4 kg of additional weights to keep neutral buoyancy. These caused increased resistance for movement through the water, causing significant reduction in distance.

In conclusion, our current results with underwater BP measurement at 2-m depth in trained BHD confirms previous reports of moderate increases during SA (supine rest) and DA (cycle ergometry with apnea) under dry conditions. Future studies should investigate BP changes at greater depths (measured with subaqueous sphygmomanometer) as well as continuously measure BP at depths using either invasive or photoplethysmography techniques.

Table 2. Changes in HR and SpO2 during various phases of SA, SHT DA, and DA

<table>
<thead>
<tr>
<th>Apnea</th>
<th>Baseline</th>
<th>0–19%</th>
<th>20–39%</th>
<th>40–59%</th>
<th>60–79%</th>
<th>80–100%</th>
<th>Recovery 1st min</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>HR</td>
<td>SpO2</td>
<td>HR</td>
<td>SpO2</td>
<td>HR</td>
<td>SpO2</td>
<td>HR</td>
</tr>
<tr>
<td>SA</td>
<td>74 ± 12</td>
<td>99 ± 1</td>
<td>75 ± 18</td>
<td>99 ± 1</td>
<td>64 ± 18</td>
<td>98 ± 2</td>
<td>65 ± 17</td>
</tr>
<tr>
<td>SHT DA</td>
<td>70 ± 9</td>
<td>99 ± 1</td>
<td>101 ± 21†</td>
<td>98 ± 1</td>
<td>100 ± 22†</td>
<td>98 ± 2</td>
<td>95 ± 23†</td>
</tr>
<tr>
<td>DA</td>
<td>73 ± 12</td>
<td>99 ± 1</td>
<td>117 ± 21†</td>
<td>99 ± 2</td>
<td>104 ± 25†</td>
<td>97 ± 3</td>
<td>93 ± 27†</td>
</tr>
</tbody>
</table>

Values are presented as means ± SD. HR, heart rate; SpO2, pulse oximetry; SA, static apnea; SHT DA, shortened dynamic apnea; DA, dynamic apnea. *P < 0.05 compared with baseline; †P < 0.05 compared with corresponding data point in SA.

Table 3. Baseline, maximal, overall change, and time needed to achieve maximum in blood lactate concentration after SA, SHT DA, and DA

<table>
<thead>
<tr>
<th>Blood Lactate Concentration</th>
<th>Baseline, mmol/l</th>
<th>Maximum, mmol/l</th>
<th>Change, mmol/l</th>
<th>Time to Max, min</th>
</tr>
</thead>
<tbody>
<tr>
<td>SA</td>
<td>1.7 ± 0.7</td>
<td>2.7 ± 0.8*</td>
<td>1.1 ± 0.5</td>
<td>5.4 ± 3</td>
</tr>
<tr>
<td>SHTDA</td>
<td>1.6 ± 0.4</td>
<td>2.3 ± 0.4*</td>
<td>0.8 ± 0.3</td>
<td>4.6 ± 1.9</td>
</tr>
<tr>
<td>DA</td>
<td>1.9 ± 0.4</td>
<td>3.8 ± 1.0†</td>
<td>2.0 ± 1.0†</td>
<td>3.8 ± 1.8</td>
</tr>
</tbody>
</table>

Values are presented as means ± SD. *P < 0.05 compared with baseline; †P < 0.05 compared with SA.

Fig. 3. Change in pulse oximetry (SpO2) during SA and DA. Values are presented as means ± SD. Level of desaturation was similar at the end of SA and DA. P = NS (nonsignificant).
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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

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