Clin Auton Res (2009) 19:363–366
DOI 10.1007/s10286-009-0025-2

RESEARCH LETTER

Cardiovascular changes during maximal breath-holding in elite divers

Pietro Guaraldi · Maria Serra · Giorgio Barletta · Giulia Pierangeli · Rossana Terlizzi · Giovanna Calandra-Buonaura · Danilo Cialoni · Pietro Cortelli

Received: 21 April 2009 / Accepted: 28 June 2009 / Published online: 5 August 2009

Abstract During maximal breath-holding six healthy elite breath-hold divers, after an initial "easy-going" phase in which cardiovascular changes resembled the so-called "diving response", exhibited a sudden and severe rise in blood pressure during the "struggle" phase of the maneuver. These changes may represent the first tangible expression of a defense reaction, which overrides the classic diving reflex, aiming to reduce the hypoxic damage and to break the apnea before the loss of consciousness.

Keywords Apnea · Diving · Sympathetic nervous system · Parasympathetic nervous system · Hypoxemia

Introduction

During voluntary apnea after an initial "easy-going" phase, in which the subject feels no urge to breathe, involuntary diaphragmatic contractions occur, signaling the physiological need to restart breathing ("physiological breaking point") [9]. Despite that, the apnea can be prolonged into a "struggle" phase, until when a powerful involuntary defense reaction breaks the apnea before the subject becomes unconscious.

The major physiological adaptation to endure the lack of oxygen during apnea is the so-called "diving reflex", characterized by peripheral vasoconstriction and bradycardia. Although several studies assessed this reflex in humans and animals, most of the studies adopted different techniques, protocols and experimental settings and focused on the "easy-going" phase of the apnea only [2, 3, 7].

While the "diving bradycardia" is a consistent finding in all these studies, most of the authors reported no or only a modest and gradual increase in blood pressure (BP) in humans. Only recently Heusser et al. [4] demonstrated that elite divers, during maximal end-inspiratory dry apnea, exhibit an increased sympathetic vasoconstrictor activity, with a massive increase in BP, which does not reach a ceiling.

Aim of the current study was to investigate the time course of the hemodynamic changes occurring during maximal AFI in normobaric conditions, considering separately the "easy-going" and the "struggle" phases of the maneuver.

Methods

Subjects

Six healthy male breath-hold divers from the Apnea Academy Research volunteered for the study and signed informed consent prior to the enrolment in the protocol (Table 1). All subjects were drug-free and abstained from alcohol or caffeine containing beverages from the day before the study. Before enrolment abnormalities of the autonomic control of cardiovascular system were excluded.

P. Guaraldi (✉) · G. Barletta · G. Pierangeli · R. Terlizzi · G. Calandra-Buonaura · P. Cortelli
Dipartimento di Scienze Neurologiche,
Alma Mater Studiorum-Università di Bologna,
Via Ugo Foscolo, 7, 40123 Bologna, Italy
e-mail: pietro.guaraldi@unibo.it

M. Serra
Dipartimento di Chirurgia Generale e dei Trapianti d'Organo,
Ospedale S.Orsola-Malpighi, Via Massarenti 9,
40123 Bologna, Italy

D. Cialoni
Apnea Academy Research, Alessandria, Italy

Table 1 Subjects characteristics and apneic time

Subject no.	Age (years)	Weight (kg)	Height (cm)	BMI	Apneic time (s)	Static apnea record (s)	Individual delay (s)
1	27	72	182	21.7	240	285″	17″
2	38	74	176	23.9	208	285″	23″
3	34	70	168	24.8	223	246″	20″
4	32	60	173	20	207	330″	17″
5	48	83	177	26.5	237	345″	19″
6	44	75	183	22.4	267	326″	12″
Mean ± SD	37 ± 8	72 ± 8	177 ± 6	22 ± 3	230 ± 23	303 ± 37″	18 ± 4″

BMI body mass index; *static apnea record* individual static apnea record; *individual delay* individual time delay from the end of apnea to the minimum of sO_2 and pO_2 and to the maximum of pCO_2

Table 2 Cardiovascular and blood gases changes during baseline, end of "easy-going" and end of "struggle" phases of breath-holding with face immersion in cold water

	Basal period	End of "easy-going" phase	End of "struggle" phase
Length (s)	NA	108 ± 59	122 ± 48
SBP (mmHg)	142 ± 9	173 ± 10	275 ± 48*§
DBP (mmHg)	89 ± 8	102 ± 9	131 ± 22*§
HR (b/m)	77 ± 13	65 ± 12	54 ± 8§
CO (L/min)	5.86 ± 1.05	5.06 ± 1.63	5.02 ± 1.83
TPR (mmHg s/ml)	1.19 ± 0.32	1.71 ± 0.58	2.35 ± 0.76§
pO_2 (mmHg)	86 ± 16	61 ± 26	20 ± 8*§
pCO_2 (mmHg)	33 ± 6	36 ± 7	47 ± 7*§
sO_2 (%)	100 ± 1	93 ± 11	72 ± 13*§

Data are presented as means ± SD in the three phases of the maneuver

SBP systolic blood pressure; *HR* heart rate; *CO* cardiac output; *TPR* total peripheral resistance; *sO_2* oxygen saturation; *pO_2* partial tension of oxygen; *pCO_2* partial tension of carbon dioxide

§ End of "struggle" phase versus basal: $P < 0.05$

* End of "struggle" phase versus end of "easy-going" phase: $P < 0.05$

Measurements

We measured non-invasively systolic and diastolic BP (SBP, DBP) by photoelectric plethysmography (Portapres, TNO-TPD Biomedical Instrumentation, Amsterdam, the Netherlands); cardiac output (CO) and total peripheral resistances (TPR) by model flow analysis; heart rate (HR) by electrocardiogram (Grass 7P511). We recorded oxygen saturation (sO_2) with a finger pulse oxymeter (Ohmeda 3740 Pulse Oxymeter, Louisville, CO, USA; 3 s averaging) and partial tension of oxygen (pO_2) and carbon dioxide (pCO_2) by the transcutaneous pO_2/pCO_2 monitoring system (Radiometer, Copenhagen). Breathing movements were recorded by an abdominal strain gauge (Grass DC preamplifier 7P1). All parameters were monitored continuously with a polygraph (Grass Model 78). A/D conversion at 1,000 Hz per channel was obtained for the ECG, BP and breathing signals for subsequent calculation. Apneic time was measured by a chronometer and recorded with an event marker connected to the computer.

Experimental protocol

The study was conducted in conformity with the principles of the Declaration of Helsinki and was approved by the Independent Ethics Committee of the University of Bologna. The study was performed in a temperature-controlled (23 ± 1°C) clinical investigation room. Subjects were asked to perform the longest apnea in the sitting position, with their face totally immersed in a container set on a table filled with cold water (6–8°C), keeping their arms on both sides of the container. Volunteers had 5 min to prepare themselves to the apnea in the way they were used to do in their training sessions.

Data analysis

The cardiovascular changes induced by AFI were evaluated in three phases. We considered the mean BP, HR, CO and TPR from the minute before the preparation to apnea as the basal period (phase I). The apneic time was divided

in 10-s periods and we calculated the mean of BP, HR, CO and TPR for each period. The first diaphragmatic contraction during apnea, identified from the artefacts on the respiratory signal was considered as the beginning of the "struggle" phase. The values obtained 10 s before the first diaphragmatic contraction were considered as the end of "easy-going" phase (phase II) and the values obtained 10 s before the end of apnea as the end of "struggle" phase (phase III). Since finger probe pulse oximetry [6] and the transcutaneous pO_2/pCO_2 monitoring system exhibited a significant time delay in recording apnea-induced hypoxia and hypercapnia (E5280 probe had a response time of approximately 20 s for 90% response in pO_2 and approximately 50 s for 90% response in pCO_2), we synchronized the delayed values of pO_2, pCO_2 and sO_2 with the proper corresponding cardiovascular values, calculating, for each subject, the time delay from the end of apnea to the minimum of sO_2 and pO_2 and to the maximum of pCO_2 (Table 1).

Statistics

Data obtained in phase I, II and III were reported as means ± SD and compared using a repeated measures analysis of variance (ANOVA) with Bonferroni and Tukey post hoc test for multiple comparisons (SPSS Statistics 17.0, Chicago, IL, USA; $P < 0.05$).

Results

Apneic times are reported in Table 1.

Cardiovascular parameters and pO_2, pCO_2 and sO_2 values are reported in Table 2.

No significant changes were found comparing the end of "easy-going" phase with baseline.

During the end of the "struggle" phase a significant rise in SBP ($P < 0.001$), DBP ($P < 0.001$), TPR ($P < 0.01$) and pCO_2 ($P < 0.01$), and a significant decrease in HR ($P < 0.01$), pO_2 ($P < 0.001$) and sO_2 ($P < 0.001$) were observed with respect to baseline.

During the end of the "struggle" phase SBP ($P < 0.001$), DBP ($P < 0.03$) and pCO_2 ($P < 0.05$) were significantly higher compared to the end of "easy-going" phase, while pO_2 ($P < 0.005$) and sO_2 ($P < 0.005$) were significantly lower.

Discussion

While the cardiovascular modifications during the "easy-going" phase were similar in our study to those previously reported in the literature, the large rise in BP during the "struggle" phase of AFI was different from the modest or transient BP increase demonstrated in most of the preceding studies [8]. Our results support recent findings by Heusser et al. [4] and provide information on the blood gases modifications in course of AFI, strengthening previously reported data [8].

The cardiovascular changes described in the current study during the "easy-going" phase of AFI resemble those described in diving mammals. However, while diving mammals are claimed to be able to maintain stable their mean arterial pressure for several minutes throughout the dive [5], humans cannot and exhibit a sudden and significantly severe rise in BP after the physiological breaking point. Therefore, it is tempting to speculate that these changes may represent the first tangible expression of a defense reaction, which overrides the classic diving reflex during the "struggle" phase of AFI, aiming to reduce the hypoxic damage and to break the apnea before the loss of consciousness.

This powerful defense reaction might represent the best response to acute extreme hypoxia/hypercapnia, but constitutes also the limit of our adaptation to prolonged apnea.

Recent studies [1, 4] showed that repeated episodes of hypoxemia in elite breath-hold divers do not lead to sustained sympathetic activity and arterial hypertension in absence of additional comorbidities. Similarly in the current study no clinical signs of circulatory failure or cardiac arrhythmias were observed, and the subjects reported no symptoms.

In conclusion, the current study confirms that elite divers exhibit a massive rise in BP during the "struggle" phase of AFI, but the small number of subjects analyzed, the specifics of our experimental setting and the inter-individual variability of CO values should be taken in account.

References

1. Dujic Z, Ivancev V, Heusser K, Dzamonja G, Palada I, Valic Z, Tank J, Obad A, Bakovic D, Diedrich A, Joyner MJ, Jordan J (2008) Central chemoreflex sensitivity and sympathetic neural outflow in elite breath-hold divers. J Appl Physiol 104:205–211
2. Ferretti G (2001) Extreme human breath-hold diving. Eur J Appl Physiol 84:254–271
3. Foster GE, Sheel AW (2005) The human diving response, its function, and its control. Scand J Med Sci Sports 15:3–12
4. Heusser K, Dzamonja G, Tank J, Palada I, Valic Z, Bakovic D, Obad A, Ivancev V, Breskovic T, Diedrich A, Joyner MJ, Luft FC, Jordan J, Dujic Z (2009) Cardiovascular regulation during apnea in elite divers. Hypertension 53:719–724
5. Hochachka PW (1981) Brain, lung, and heart functions during diving and recovery. Science 212:509–514
6. Lindholm P, Blogg SL, Gennser M (2007) Pulse oximetry to detect hypoxemia during apnea: comparison of finger and ear probes. Aviat Space Environ Med 78:770–773

7. Muth CM, Ehrmann U, Radermacher P (2005) Physiological and clinical aspects of apnea diving. Clin Chest Med 26:381–394
8. Pingitore A, Gemignani A, Menicucci D, Di Bella G, De Marchi D, Passera M, Bedini R, Ghelarducci B, L'Abbate A (2008) Cardiovascular response to acute hypoxemia induced by prolonged breath holding in air. Am J Physiol Heart Circ Physiol 294:H449–H455
9. Schagatay E, van Kampen M, Emanuelsson S, Holm B (2000) Effects of physical and apnea training on apneic time and the diving response in humans. Eur J Appl Physiol 82:161–169