CASE REPORT

Asystole and increased serum myoglobin levels associated with 'packing blackout' in a competitive breath-hold diver

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Summary

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Many competitive breath-hold divers use 'glossopharyngeal insufflation', also called 'lung packing', to overfill their lungs above normal total lung capacity. This increases intrathoracic pressure, decreases venous return, compromises cardiac pumping, and reduces arterial blood pressure, possibly resulting in a syncope breath-hold divers call 'packing blackout'. We report a case with a breath-hold diver who inadvertently experienced a packing blackout. During the incident, an electrocardiogram (ECG) and blood pressure were recorded, and blood samples for determinations of biomarkers of cardiac muscle perturbation (creatine kinase-MB isoenzyme (CK-MB), cardiac troponin-T (TnT), and myoglobin) were collected. The ECG revealed short periods of asystole during the period of 'packing blackout', simultaneous with pronounced reductions in systolic, diastolic, and pulse pressures. Serum myoglobin concentration was elevated 40 and 150 min after the incident, whereas there were no changes in CK-MB or TnT. The ultimate cause of syncope in this diver probably was a decrease in cerebral perfusion following glossopharyngeal insufflation. The asystolic periods recorded in this diver could possibly indicate that susceptible individuals may be put at risk of a serious cardiac incident if the lungs are excessively overinflated by glossopharyngeal insufflation. This concern is further substantiated by the observed increase in serum myoglobin concentration after the event.

Introduction

Many competitive breath-hold divers use a technique called 'glossopharyngeal insufflation', or 'lung packing', to increase the volume of air in the lungs above the normal total lung capacity before their performances. After a maximal inhalation, the diver takes a mouthful of air, then opens the glottis and forces this air into the lungs through a manoeuvre resembling swallowing. By repeating the manoeuvre, a volume of air up to 4 l above the normal total lung capacity can be added to the lungs (Overgaard et al., 2006; Seccombe et al., 2006; Loring et al., 2007). This overfilling of the lungs causes an increase in intrathoracic pressure (Overgaard et al., 2006; Seccombe et al., 2006), and as a consequence, cardiac pumping may be compromised and arterial blood pressure reduced to a great extent (Andersson et al., 1998; Novalija et al., 2007). Among breath-hold divers, a loss of consciousness called 'packing blackout' is known to be associated with excessive insufflation of the lungs. Such adverse

events have been conveyed to the scientific community, and the occurrence of 'packing blackout' is mentioned in some studies concerning glossopharyngeal insufflation (Andersson et al., 1998; Lindholm & Nyren, 2005; Novalija et al., 2007; Potkin et al., 2007). However, as research ethics naturally constrains such investigations, there is limited information about various physiological variables during an episode of 'packing blackout'.

Case report

The breath-hold diver in this case, a 22-year-old, healthy male (height 184 cm; weight 78 kg; blood pressure 110/62 mmHg), was a volunteer for a study concerning the cardiovascular and respiratory effects of maximal-duration apnoeas in competitive breath-hold divers. After receiving a description of the experimental protocol, which was approved by the research ethics committee at Lund University, he gave his informed consent. The protocol of the study was designed to resemble a

breath-hold training session or competition performance in the 'static apnoea' category, but in contrast to static apnoea competitions, no immersion was involved. Instead, the subject was resting in the supine position on a mattress during the entire protocol. The participants were instructed to use preparations according to their own preference in order to be able to perform one maximum-duration apnoea. Accordingly, the diver in this case intended to perform two 'warm-up apnoeas' of sub-maximal duration before attempting the maximal-duration apnoea. The diver's preparations included that all apnoeas were to be preceded by hyperventilation and glossopharyngeal insufflation. In control measurements before the apnoeas, the diver's vital capacity was 6·9 l in the supine position, which increased to 7·4 l with glossopharyngeal insufflation. After baseline recordings of various cardiovascular variables, the diver prepared for the first 'warm-up' apnoea and began hyperventilating. With a maximal inhalation followed by glossopharyngeal insufflation, the diver began the first apnoea. After approximately 10 s it was noted that he began exhaling and experienced a loss of consciousness, presumably because of a fall in blood pressure and cerebral perfusion following glossopharyngeal insufflation. The experimenter intervened and approximately 30 s after the beginning of apnoea the diver spontaneously regained consciousness. The diver reported no symptoms or residual effects from the event.

During the incident, an electrocardiogram (ECG) was recorded with an amplifier connected to an A/D-conversion system (lead II, ECG100, BIOPAC Systems Inc., Goleta, CA, USA) used for recordings of the various variables, and arterial blood pressure was recorded non-invasively with a photoplethysmometer (Finapres 2300, Ohmeda, Madison, WI, USA) with the cuff on the right middle finger (Fig. 1). About 4 s into the apnoea, without any preceding sinus bradycardia, a ventricular extrasystole occurred (Fig. 2). Immediately following this, the normal sinus rhythm changed to sinus arrest with nodal escape, including two periods of asystole with R–R intervals of 6·6 and 7·3 s. The arterial blood pressure recording confirms the periods of asystole. Blood samples for determination of serum levels of biomarkers of cardiac muscle perturbation, i.e. creatine kinase-MB isoenzyme (CK-MB), cardiac troponin-T (TnT), and myoglobin had been collected at the beginning of recordings (control) and were again collected 40 and 150 min after the event. Myoglobin increased after the event, while there were no changes in the other markers (Table 1).

Discussion

The systolic, diastolic, and pulse pressures were markedly reduced during the period when the diver was performing the glossopharyngeal insufflation technique and began holding his breath, in accordance with earlier observations (Andersson et al., 1998; Novalija et al., 2007). The reductions in arterial pressures follow the impediment of venous return and consequent interference with cardiac pumping that results from the high
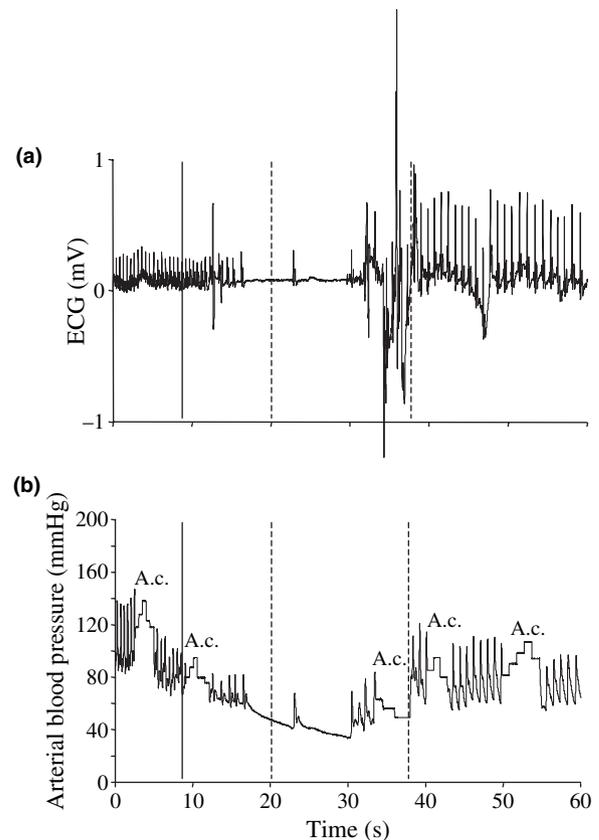
Figure 1 (a) ECG and (b) non-invasive arterial blood pressure recordings during the 'packing blackout'. The vertical, solid line (9 s) indicates the beginning of apnoea. The vertical, dashed lines (20 and 37 s) indicate the period of unconsciousness, as judged by reviewing a video recording of the event. 'A.c.' indicates autocalibration periods of the blood pressure monitor.
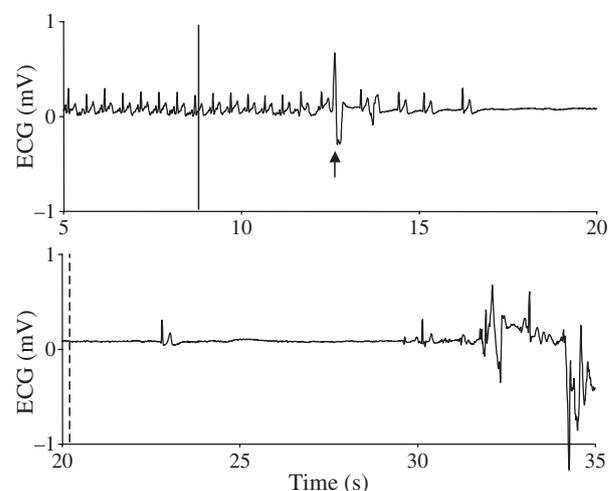
Figure 2 ECG during the 'packing blackout'. The vertical solid line (9 s) indicates the beginning of apnoea. The vertical, dashed line (20 s) indicates beginning of syncope. A ventricular extrasystole is identified (arrow). The QRS-complexes occurring between 13–23 s represent nodal escape beats (absent P waves). The recording is influenced by motion artifacts at 32 s and onwards.

Table 1 Serum concentrations of creatine kinase-MB isoenzyme (CK-MB), troponin-T (TnT), and myoglobin before and after the 'packing blackout'.

	CK-MB (μg l^{-1})	TnT (μg l^{-1})	Myoglobin (μg l^{-1})
Control	1·7	<0·05	44
+40 min	1·7	<0·05	119
+150 min	2·0	<0·05	117

intrathoracic pressure (Potkin et al., 2007). This probably causes a decrease in cerebral perfusion, which most likely was the ultimate cause of syncope in this diver. The glossopharyngeal insufflation technique has previously been ascribed to be associated with a risk of syncope due to compromised cardiac pumping and reduced arterial blood pressure (Andersson et al., 1998; Lindholm & Nyren, 2005; Novalija et al., 2007; Potkin et al., 2007). However, there have been no previous reports actually showing the blood pressure changes during such an adverse event.

Whereas it was already known that the glossopharyngeal insufflation technique could be associated with the risk of syncope and leads to right and left ventricular dysfunction (Potkin et al., 2007), there have been no previous reports indicating that the consequences of this technique may be dysrhythmogenic. The short asystolic periods recorded in this diver could possibly indicate that susceptible individuals may be put at risk of a serious cardiac incident if the lungs are excessively overinflated by the use of the glossopharyngeal insufflation technique. This concern is further substantiated by the observed increase in serum myoglobin concentration after the event. The increase in myoglobin, even without increases in CK-MB and troponin-T, may indicate that the glossopharyngeal insufflation in this case resulted in poor perfusion of the myocardium, with consequent myocardial ischemia. Also in patients with significant obstructive coronary artery disease and normal left ventricular function, severe short-lasting myocardial ischemia caused a transient slight cardiac release of myoglobin, without changes in troponin-T or creatine phosphokinase concentrations (Krüger et al., 2005). It was concluded that the observations could be explained by a rapid reperfusion effect on ischemic myocardium or minor damaged single myocardial cells (Krüger et al., 2005). The increase in myoglobin, with unchanged CK-MB and troponin-T levels, may be explained by the relatively smaller molecular size of myoglobin, i.e. the smaller and cytosolic dissolved myoglobin might more easily pass the cell membrane in ischemic and injured cells (Krüger et al., 2005). We observed no increases in myoglobin, CK-MB, or troponin-T in six other competitive breath-hold divers performing glossopharyngeal insufflation and maximal-duration apneas leading to profound asphyxia without experiencing loss of consciousness in the study for which the diver in this case volunteered.

It is widely accepted that serum elevations of CK-MB, troponin-T and myoglobin indicate myocardial damage (Hetland & Dickstein, 1996; Christenson & Azzazy, 1998; Krüger et al., 2005), but a rise in myoglobin may also be found in conditions related to skeletal muscle damage. However, we doubt that skeletal muscle could have been adversely affected by the incident described here, i.e. skeletal muscle is unlikely to be the origin of the myoglobin release.

The risk for cardiac perturbation from a technique practiced by competitive breath-hold divers suggested by the observations in the present case represents a new risk not described earlier. In addition, competitive breath-hold divers are subjected to several other risks. Besides the obvious risk for a hypoxic loss of consciousness naturally associated with prolonged apneas (Lindholm, 2007), e.g., pulmonary hypertension with right ventricular hypertrophy (Scherhag et al., 2005), pneumomediastinum or cerebral arterial gas embolism resulting from glossopharyngeal insufflation (Jacobson et al., 2006; Lindholm et al., 2007), and pulmonary edema and hemoptysis following lung squeeze during diving (Fitz-Clarke, 2006; Lindholm et al., 2008; Linér & Andersson, 2008) are risks that recently have been either suggested or observed in competitive breath-hold divers. In addition, the long-term effects from repetitive exposures to hypoxia on the brain need further evaluation (Andersson et al., 2006; Potkin & Uszler, 2006; Ridgway & McFarland, 2006). Whereas we can understand the incentives for individuals participating in competitive breath-hold diving, we do not endorse this activity as being medically safe.

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