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Cardiorespiratory responses and reduced apneic time to cold-water face immersion after high intensity exercise

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ABSTRACT

Apnea after exercise may evoke a neurally mediated conflict that may affect apneic time and create a cardiovascular strain. The physiological responses, induced by apnea with face immersion in cold water (10 °C), after a 3-min exercise bout, at 85% of $VO_{2max,}$ were examined in 10 swimmers. A pre-selected 40-s apnea, completed after rest (AAR), could not be met after exercise (AAE), and was terminated with an agonal gasp reflex, and a reduction of apneic time, by 75%. Bradycardia was evident with immersion after both, 40-s of AAR and after AAE ($P < 0.05$). The dramatic elevation of, systolic pressure and pulse pressure, after AAE, were indicative of cardiovascular stress. Blood pressure after exercise without apnea was not equally elevated. The activation of neurally opposing functions as those elicited by the diving reflex after high intensity exercise may create an autonomic conflict possibly related to oxygen-conserving reflexes stimulated by the trigeminal nerve, and those elicited by exercise.

1. Introduction

A condition that has lately drawn attention due to the increased number of reported unexplained sudden deaths is, when athletes enter the often very cold and turbulent water in the swimming segment of triathlon events. In that segment they either run a short distance prior to entering the water until they get to an adequate depth to dive, or immediately begin with a high intensity swimming effort so that they can gain their space in the event. In triathlon events in the USA between 2006–2008 there were 14 deaths, 13 of which during the water segment (Harris, 2010). Shattock and Tipton (2012), have recently introduced the term "autonomic conflict" to describe a mechanism underlying the increased number of sudden deaths that appeared even in strong healthy swimmers, in many cases 3-m from entry point in cold water. This autonomic conflict is thought to be the result of the co-activation of the sympathetic and vagal system, inducing electrical disturbances in the heart and arrhythmias. We could speculate that high intensity exercise such as that which the triathletes are exposed to, prior or immediately at entry in cold water can induce a strong autonomic response which along with the increased metabolic demands might be expected to exacerbate this autonomic conflict.

Cold-water immersion apnea in water is a condition that can elicit two different mechanisms of physiological responses that can occur either consecutively or concurrently depending on the temperature of the water and the condition of the individual (Paton et al., 2005). Those are on one hand the responses induced by the diving response (DR) and on the other hand the responses induced by the cold shock response (CSR). The DR causes a parasympathetically driven bradycardia via an excitation of cardiac vagal motor-neurons and a drop in heart rate, a concomitant rise in blood pressure, and peripheral vasoconstriction with a decrease in stroke volume and cardiac output, and is known to be exaggerated by face immersion in cold water. The CSR with immersion in cold water below 15 °C, induces a sympathetically driven stimulus via the activation of cutaneous thermoreceptors, many times a rise in heart rate, hyperventilation, a respiratory gasp, and an increase in blood pressure (Geladas, 2008). Additionally, it has been demonstrated that the cold shock response when activated could reduce maximal apneic time and stimulate ventilation with cold water face immersion. Jay et al. (2007) suggest that face immersion at temperatures of 10 °C and below create a rapid cold-temperature-sensitive neuronal drive that has an immediate effect on reducing apnoeic times. In the above study maximum apneic time had dropped almost 50% from 30 to 0 °C.

The last few years the DR has been considered a subset of the trigemino-cardiac reflex (TCR), both of which belong to the

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so called 'oxygen conserving reflexes' (OCR), that are activated by stimulation of the trigeminal nerve than innervates the face (Schaller et al., 2009; Lemaitre et al., 2015). The TCR manifests itself after mechanical stimulation of the trigeminal nerve, as bradycardia, hypotension, and gastric hypermobility while the DR leads to decreased ventilation, bradycardia, intense peripheral vasoconstriction, and increased mean arterial blood pressure (MABP). All these responses collectively prevent drowning and maintain oxygenation to the heart and brain at the expense of less oxygen demanding organs (Lemaître et al., 2007; Lemaitre et al., 2015). As mechanisms, they have not yet been fully elucidated, but there are assumptions that the DR could even be the peripheral expression of the TCR. The difference between DR and TCR is that in TCR there is a MABP decrease, whereas in DR the MABP gradually increases. These protective and oxygen conserving reflex systems, mediate from the rostral ventrolateral medulla oblongata to the upper brainstem (Amirjamshidi et al., 2013). Both the TCR, and the DR are believed to be modifications to allow economizing of the oxygen stores, acting as protective OCR, and aiming at keeping the body alive during cold water immersion or other types of stimuli (Lemaitre, 2015). There is a clinical importance in understanding both reflexes because they have both been associated with detrimental to survival outcomes, although the exact pathophysiology of these systems still needs to be determined. The TCR is important as it has been noted -through mechanical stimulation- to induce hypotension in neurosurgery, while the DR is known to induce severe to fatal apneas to breath hold divers and infants (Amirjamshidi et al., 2013; Matturri et al., 2005). The DR is highly pronounced in infants and if exaggerated could be detrimental, and is frequently considered the cause of sudden infant death syndrome (SIDS).

Exercise at its initiation also triggers sympathetic activity with a parallel parasympathetic withdrawal, resulting to increases in heart rate, which immediately after the cessation of exercise need to be both reversed, through parasympathetic reactivation and sympathetic deactivation. Cold water face immersion has been suggested as an efficient way in the acceleration of post-exercise parasympathetic reactivation (Al Haddad et al., 2010a). The continuation of sympathetic hyperactivity with the postponement of the parasympathetic reactivation after exercise has been associated with a poor cardio protective effect, with ventricular fibrillation and an increased risk of sudden cardiac death (Billman 2002; Smith et al., 2005; as in the Al Haddad et al., 2010a,b). Lindholm et al. (1999) noted that apnea during steady state exercise, without water immersion had elicited bradycardia and a slowing of oxygen uptake in the lungs of exercising humans and that bradycardia and hypertension develop gradually and not immediately. Andersson (2004), on the other hand, compared cardiovascular responses to apnea with and without cold-face immersion (10 °C), in exercising humans, during steady state exercise, and noticed an overriding of the diving response with a concomitant oxygen-conserving effect. In the above study the diving response and oxygen storage was augmented with apnea with face immersion probably caused by the observed reduced cardiac output. So far the studies related to apnea and/or cold face immersion with exercise have dealt with steady state bicycle ergometer conditions and there are no reported studies of the acute immediate responses of sudden exposure to multiple stimuli such as immersion in cold water immediately after high intensity exercise. Tsuchimochi et al. (2009), suggest that both static and dynamic exercise are known to increase cardiac pump function as well as arterial blood pressure via a feedforward control neural mechanism of the exercise pressor reflex (EPR), by central command and feedback control. It is suggested that stimulation of the mesencephalic locomotor region may play a role in cardiac sympathetic activity at the onset of exercise (Tsuchimochi et al., 2009).

The activation of neurally opposing functions as those elicited by face immersion in cold water after high intensity exercise may create an autonomic conflict, possibly related to oxygen-conserving reflexes stimulated by the diving reflex, the cold shock response and/or the trigemino-cardiac reflex, and those elicited by exercise, such as the exercise pressor reflex. All these need to be addressed and elucidated. We hypothesized that the impact of preceding high intensity exercise followed by immediate cold-water face immersion would create a neurally mediated impact on the acute and immediate sympathetic-parasympathetic responses, augmenting the responses of either the diving response and/or the cold shock response, possibly triggering an antagonistic effect and eliciting an "autonomic conflict", with immediate and acute cardio-respiratory responses.

2. Materials and methods

2.1. Participants

Participants were instructed to read and sign the protocol that was in accordance with 'The Code of Ethics of the World Medical Association (Declaration of Helsinki), and was approved by the Ethics Committee of the University of Athens (No 46121, 17/01/2014). An initial health screening questionnaire (PAR-Q Validation Report) and a blood pressure screening test ensured their health status (Chisholm et al., 1975). A total of ten ($N = 10$) male swimmers aged 21.30 ± 2.5 years, with, a height of 1.84 ± 0.08 m, a weight of 84.61 ± 10.07 kg, and a % body fat of 9 ± 0.03%, participated in this study. They were all active athletes with a VO_{2max} of 48.20 ± 7.24 ml min^{-1} kg^{-1}. All were non-smokers and were asked to refrain from alcohol and exercise 24 h prior to all testing.

2.2. Protocol

Anthropometric measurements were determined after participants completed an informed consent form, and their health screening assessment. An incremental treadmill test was then administered, with increases in both speed and % grade, until the criteria for attaining a VO_{2max}, were achieved (Bruce protocol). On the same day following the determination of their VO_{2max}, participants were acquainted with the face immersion and the experimental procedures that took place a week later. The day of the experimental trials, on arrival, the participants rested, connected to a spirometer, a Polar heart rate monitor, and a pulse oximeter for 10 min in a sitting position, and data were continuously sampled. Face immersion apneas took place after rest, followed by a 10-min break and repeated again immediately after a 3-min high intensity trial. Systolic and diastolic blood pressure, venous blood gases and respiratory parameters were measured, at rest, after the resting facial immersion, following a 10-min break before exercise and post-exercise immediately after facial immersion. Four subjects repeated the protocol above without apnea to distinguish the effect of exercise recovery alone from exercise recovery with facial cold-water immersion. In this experiment, the post-exercise period measurements of heart rate and blood pressure were matched to the terminal apnea times of each individual subject.

2.3. Face immersion procedure

The main experimental procedure consisted of two face immersions with apnea in cold water (10 °C ± 1.05), one after resting for 10 min, and a second one after a 3-min progressively increasing in intensity exercise trial on a treadmill. During cold-water face immersion participants lay in a prone position on a mattress with an attached tank of cold water, and submerged their faces until their