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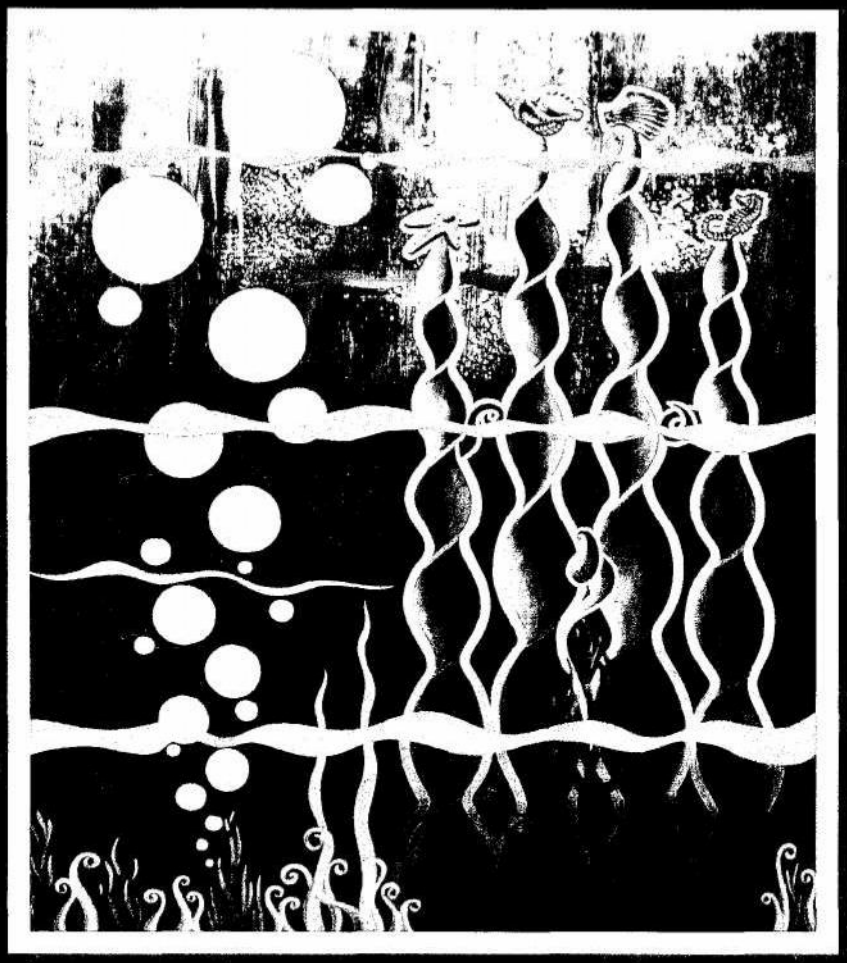

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PROCEEDINGS

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014 CARDIOVASCULAR RESPONSES TO APNEA DURING DYNAMIC EXERCISE AND BREATH-HOLD UNDERWATER SWIMMING

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INTRODUCTION

Breath-hold maneuvers induce hypoxia, hypercapnia, and various cardiovascular responses typically including increases in total peripheral resistance, mean arterial pressure (MAP) and decreases in heart rate (HR). The cardiovascular effects are commonly interpreted as an oxygen saving mechanism similar to the mammalian dive response [1, 2]. Exercise provokes opposite cardiovascular responses which support oxygen spending: An increase in HR and, under some circumstances, also stroke volume increase. Changes in total peripheral resistance (TPR) during exercise are the result of a vasoconstriction in the non-exercising tissues and a vasodilatation in the exercising muscles.
In a recent study it was shown that the combination of apnea and intensive dynamic exercise leads to a marked increase in MAP and a HR depression 15 s to 20 s after the onset of the stimulus. These effects were shown to be primarily due to breath-holding, not to the resulting hypoxia and/or hypercapnia [3]. The increase of MAP in these combinations is obviously mediated by a vasoconstriction. The combination of exercise and apnea is a typically combination in underwater swimming. Moreover effects might be assumed by increased ambient pressure and the swimming speed. Aim of this study was, to investigate the applicability of the results from dry lab experiments to a field situation in open water.

METHODS

4 male and 2 female experienced divers performed 2x2 runs of apnea swimming with two different intensities on the surface, in 5 m and 10 m depth (12 runs in total). The runs for the different depths were performed on different days. Due to technical problems data of one male subject were only available for 10 m depth. Water temperature was 24 °C in all experiments.The task for the subject was to swim as long as possible in apnea with a constant speed. After 2 min resting the subject removed the mask and rested for further 2 min. All experiments were performed without a mask to avoid different influence from receptors in the face. In the under water experiments the subjects breathed before and after the swimming from the SCUBA of a saftety diver. The subjects should choose alternating moderate and high speed in the runs (Fig. 1). After each run the subjects recovered for 2 min.

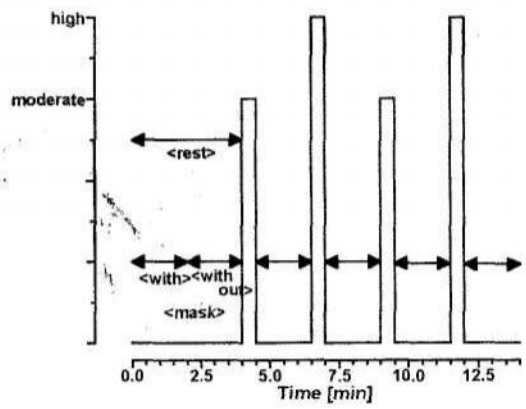

Fig. 1: Complete protocol performed on the surface, in 5 m, and 10 m depth.



Fig. 2: Swimming situation under water. The subject held the board with electronics during the apnea swimming and is accompanied by a safety diver.

HR and swimming speed were monitored simultaneously with a programmable data logger system designed for under water applications. A Polar(r) sensor was applied to detect heart beats. The signal was received by a Polar Smart Receiver(r) and registered with a time resolution of 200 Hz. HR was evaluated beat by beat and averaged for 2 s intervals. The whole electronics are integrated into a board which was held by the subject (Fig. 2).

Data analysis was performed with SPSS. Two-way-ANOVA's were performed for repeated measurements with factors 'depth' and 'intensity' for swimming and recovery data and with factors 'depth' and 'mask' for the resting data, respectively. Due to the small number of subjects multiple comparisons were not performed. Significance was accepted for P<5%.

RESULTS

Tab. 1: Mean (± std.dev.) HR (min-1) and speed (m s-1) for the different depths and intensities (n=6).

	Surface		5 m depth		10 m depth	
	HR	Speed	HR	Speed	HR	Speed
Rest with mask	104±11		86±25		82±12	
Rest without mask	90±13		88±27		83±11	
Moderate speed	101±24	0,53±0,05	98±25	0,55±0,04	90±21	0,63±0,09
Recovery	98±18		90±20		87±16	
High speed	118±34	0,63±0,09	114±28	0,78±0,08	104±20	0,79±0,10
Recovery	108±25		97±23		94±16	

Tab. 1 gives an overview of the averages with regard to intensity and depth. Non-significant changes were seen for resting with and without mask. In general HR is decreasing with depth during the swimming and during recovery, although the speed is increasing with depth or even in the same range. For HR both factors 'depth' and 'intensity' had a significant influence, whereas for speed only the factor 'intensity' was proven significant. No difference was seen in speed between the two repetitions. Moreover, the speed was seen relatively constant for each run.

For both intensities HR started to increase after the onset of swimming and showed stagnation after approximately 10 s. There was seen a suspicious delay for the increase in 10m depth (see Fig. 3a). With exception of the 10 m depth/high intensity trial, to begin of recovery HR increases for about 5 s and then declined (Fig. 3b).

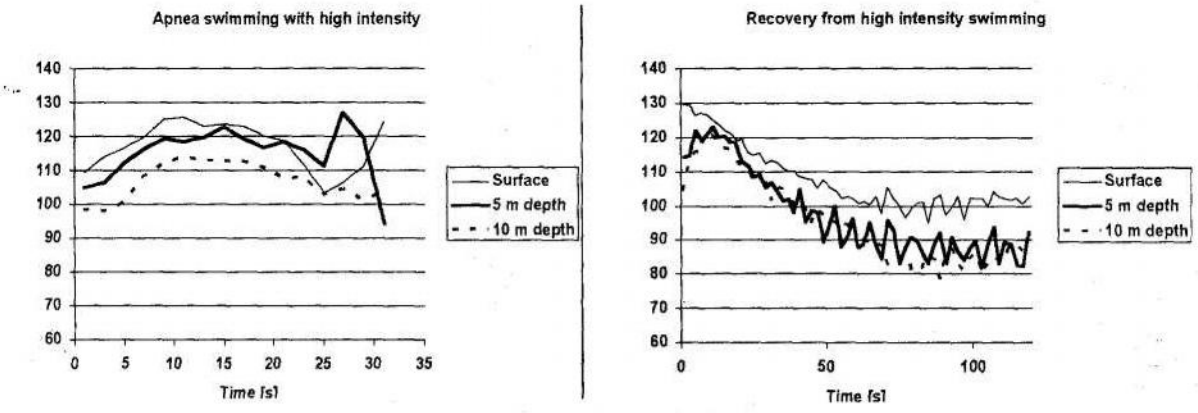

Fig. 3: Mean HR during (left panel) and after swimming (right panel) for 5 (surface/5 m) and 6 (10 m) subjects. SEs ranged typically between 8 and 12 min-1.

DISCUSSION

Differences in HR during swimming on surface versus depth may be explained by the change in efficiency. Swimming at the surface creates more draft than underwater swimming with the same speed.

After the onset of exercise it is expected to see an increase in HR. In low or moderate intensities this may stabilize after about 60 s, for high intensities a continuous increase is expected. However, in these experiments an increase in HR was only seen in the first seconds of activity followed by a stagnation or bradycardia. The HR results are in line with the results from experiments in the laboratory when apnea and heavy exercise were combined [3]. This confirms also results from other studies. The diverging drives of oxygen saving mechanisms due to the apnea and sympathic drives as response to exercise lead to this unusual time course in HR. Chemo-reflexes like hypoxia or hypercapnia can be excluded to have significant influence in this time frame [3].

Even if MAP was not measured it can be assumed that this is accompanied by an increase in MAP. This may endanger divers with hypertension problems. It is suggested to recommend even older divers to avoid the combination of apnea and strenuous underwater swimming. The increase in HR observed at beginning of recovery phase is probably due to intra-thoracic pressure during *apnea.*

The phenomenon of delayed HR increase in 10 m depth might be related to higher partial O2 pressures but also to a deeper inspiration before apnea. This should be subject of further investigations.

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