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Supraventricular arrhythmias following breath-hold submersions in cold water

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Tipton MJ, Kelleher PC, Golden FStC. Supraventricular arrhythmias after breath-hold submersions in cold water. Undersea Hyperbaric Med 1994; 21(3):305–313.—Twelve subjects undertook one submersion into water at 5°C and two at 10°C wearing either a wet or dry suit. During the submersions the subjects held their breath for as long as they could and then breathed through respiratory tubing for a further 10 s before being removed from the water. Bradycardia (heart rate <60 beats/min) was observed during breath holding in 10 subjects in 28 of the 36 submersions. Ectopic arrhythmias were observed in 11 subjects in 29 of the 36 submersions, a much higher frequency than previously reported. These ectopic arrhythmias included premature atrial and junctional complexes, runs of supraventricular tachycardia, and premature ventricular complexes. They occurred predominantly in the 10-s period of submersion after the cessation of breath holding. The possible etiology of these arrhythmias and their significance are discussed and it is concluded that after breath-hold termination during cold-water submersion there is a short time during which the heart may be particularly susceptible to supraventricular ectopic arrhythmias.

diving, heart rate, electrocardiogram

The hazards of the initial cardiorespiratory responses to immersion in cold water have been well documented (1), as has the danger of prior hyperventilation to extend underwater breath-hold (BH) time (2). The potential for cardiac overload during immersion in water at thermoneutral temperatures is well understood (3), and ectopic eats have also been noted during the initial stages of head-out, cold-water immersions (4). Although less well documented, cardiac arrest is now recognized as a rare but definite cause of sudden incapacitation and death when water enters the nostrils (4).

There have been some excellent reviews of the "diving response" (5–7) including many accounts of bradycardia during face-only (8–11) and whole-body submersion (12–14). Arnold (10) reported heart rates (HRs) of less than 15 beats/min in 5 of 27

subjects while undertaking face immersion in iced water: the rate in 1 subject was as low as 5.6 beats/min. Daly et al. (7) have described the potential dangers of the vagal response associated with diving, which include temporary sinus arrest.

Incidents of arrhythmia, including abnormal P waves, nodal rhythms, idioventricular rhythms, and premature beats, have been reported during BH diving and swimming (13, 15–18). However, most of the literature on diving response suggests that normal cardiac rhythm is immediately restored on breaking breath hold in water (12). A few authors have noted electrocardiogram (ECG) abnormalities immediately after the termination of breath holding (13, 16, 17), but these have been infrequently reported and only during underwater activity such as diving and swimming.

In the present paper, previously unreported ECG abnormalities of subjects undertaking resting, seated, BH submersions in cold water are described. These abnormalities were observed during an experiment designed to determine the protection provided by immersion suits against cardiorespiratory responses to cold water.

MATERIALS AND METHODS

After ethical committee approval, informed consent in accordance with the code of ethics of the World Medical Association was obtained from 12 healthy male volunteers who were not cold habituated. A medical examination, including a 12-lead ECG, was performed on each subject. The following submersions were completed by each subject:

- 5°C wearing an immersion dry suit (DS5), designed to keep the body dry with the exception of the face and hands. This was worn over the following dry underclothing: swimming trunks, cotton underwear, woollen pullover, and a cotton coverall;
- 10°C wearing the same clothing (DS10); and
- 10°C wearing a trunk (5 mm) and arms (3 mm) neoprene wet suit (WS10) over the same underclothing.

Throughout the experiments the subjects assumed a standardized, upright-seated posture on a metal chair. The order in which the subjects performed the submersions was counterbalanced to minimize the effects of cold habituation.

Experimental procedure

Each subject completed all three of his submersions in 1 day with at least 2 h between successive submersions. They refrained from eating and drinking alcoholic or caffeinated beverages before the experiment.

Subjects wore a noseclip and breathed through a mouthpiece during each submersion. Before submersion the subjects rested for 10 min in air at thermoneutral temperatures while baseline measurements were taken. At the end of the presubmersion period the subjects were lowered into the water at 0.2 m/s, by means of an electric winch attached to the chair, until the top of the head was at a depth of no greater than 15 cm.

The subjects were instructed to take a slightly larger breath than normal and begin breath holding as the water crossed their chin. The subjects held their breath for as

long as they could while submerged and were lifted from the water 10 s after breaking their breath hold. During this 10-s period the subjects were able to breathe through a mouthpiece open to room air.

Experimental measures

The physical characteristics of the subjects were measured. This included the estimation of percentage body fat from skinfold thickness obtained at four sites (19). Maximum BH time and the volume of last inspiration before breath holding were measured using a pneumotachograph (Morgan Instruments Ltd, England) attached to the inspiratory side of the mouthpiece.

A 3-lead (lead II) ECG was obtained from all subjects throughout the experiments (408 monitor, Tektronix, Oregon). This was recorded continuously on a pen recorder. HR was calculated from the two longest consecutive R–R intervals. This approach was considered to be the most appropriate indicator of vagal drive in the circumstances of the present experiment in which BH times were short and HRs were declining.

The ectopic arrhythmias were divided into the following categories: single supraventricular extrasystoles [SV(S)], including atrial and junctional premature complexes; multiple supraventricular extrasystoles [SV(M)], including pairs of premature atrial or junctional complexes and short runs of supraventricular tachycardia; and premature ventricular complexes (PVC).

Statistics

An analysis of variance (ANOVA) was performed on the BH time data obtained from the repeated-measure design. The Sheffe method of multiple comparisons was used to investigate contrasts which were significant in the ANOVA.

RESULTS

The average (SD) physical characteristics of the subjects were: age (yr) 26 (5); height (cm) 182 (7); weight (kg) 75 (9); %fat 13.1 (4.1); $\dot{V}O_{2_{max}}$ (liter/min, $n = 10$) 3.8 (0.5). The mean (SD, $n = 10$) inspiratory volumes (liters) immediately before breath holding were: DS5, 2.08 (0.94); DS10, 2.54 (0.88); WS10, 2.3 (0.88). The volumes did not differ significantly between conditions and were not related to the subsequent occurrence of arrhythmias. No significant difference was found among conditions in the heart rates recorded in air at rest. No arrhythmia was observed during breath holding in air in any of the subjects. The average heart rates recorded during breath holding in water were less than those recorded during rest in air and just before submersion (Table 1).

Table 1: Average HRs (beats/min) Recorded in Air and During Breath Holding Underwater ($n = 12$)

	Dry Suit, 5°C	Dry Suit, 10°C	Wet Suit, 10°C
Air, rest	83	75	83
Air, presubmersion	120	112	127
Water, during breath hold	58	54	58

The BH times of each of the subjects in each of the conditions and in air are presented in Table 2. During submersion, the mean BH time seen in DS5 tended to be shorter ($P < 0.1$) than that seen in either the DS10 or WS10 conditions. The BH times in DS10 and WS10 conditions were not significantly different.

When breath holding underwater the majority of subjects had HRs that were below 60 beats/min, and eight subjects developed a HR of less than 50 beats/min in at least one of the conditions. Eleven of the 12 subjects, in 29 of the 36 submersions, demonstrated ectopic arrhythmias during at least one of the conditions. These arrhythmias occurred predominantly just before the termination of breath holding and within 10 s after the end of breath holding. In 8 of the submersions they occurred both before and immediately after the termination of breath hold, and in 21 submersions they only occurred immediately after the termination of breath holding.

Details of the arrhythmias observed for each subject are presented in Table 3. Due to the chaotic nature of the rhythms observed immediately after the termination of breath holding (Figs. 1–4), only those rates recorded during breath holding are presented in Table 3.

Single supraventricular extrasystoles, including premature atrial and junctional complexes, were the most prevalent arrhythmias and were observed in 10 subjects in a total of 23 breath holds during submersion (Figs. 1, 2, and 4). Pairs of supraventricular extrasystoles or runs of supraventricular tachycardia [SV(M)] were observed at the end of breath holding in 10 of the subjects in a total of 14 submersions (Figs. 2 and 3). On a 1-lead ECG it is difficult to determine the origin of supraventricular ectopic arrhythmias; however, some of these arrhythmias demonstrated upright P waves with a non-sinus morphology and a shortened P–R interval consistent with an atrial origin (Fig 1, 4th complex), whereas others had no evidence of a P wave suggestive of a nodal origin. Some of the arrhythmias seemed to be related to respiratory rhythm (Fig. 4); this could, however, represent atrial bigeminy with a heart rate coincidental with respiration.

Table 2: Maximum BH times (seconds) in Air and During Submersion

Subject	Air	Dry Suit, 5°C	Dry Suit, 10°C	Wet Suit, 10°C
1	25.8	17.3	26.8	25.4
2	22.8	5.3	9.2	18.1
3	55.8	27.1	26.6	20.9
4	35.4	21.5	34.3	20.9
5	28.2	27.4	32.5	79.9
6	36.3	11.4	22.1	12.8
7	41.3	21.8	44.6	49.3
8	37.0	17.8	20.4	13.7
9	37.4	22.6	26.4	17.5
10	27.8	17.6	36.1	16.8
11	80.5	96.1	105.0	95.8
12	47.0	19.6	18.3	20.8
Mean	39.6	25.6	33.8	32.7
SD	15.9	23.0	24.2	27.7

Table 3: Arrhythmias (AR) Associated with Submersion During Breath Hold and Immediately Post-Breath Hold[a]

	Dry Suit, 5°C		Dry Suit, 10°C		Wet Suit, 10°C	
n	BH, HR/AR	PostBH, AR	BH, HR/AR	PostBH, AR	BH, HR/AR	PostBH, AR
1	50/J SV (S)	SV (S)	45/1°, J	SV (S&M)	40/J	SV (S&M)
2	130/–	–	98/–	–	87/–	–
3	54/J	SV (M) PVCs	49/J	–	70/J	SV (M)
4	30/–	SV (S)	37/–	SV (S)	54/– SV (S)	SV (M)
5	52/– SV (S)	SV (M)	68/–	–	51/– SV (S)	SV (S)
6	100/–	SV (S)	74/–	SV (S&M)	94/–	–
7	53/J	SV (S)	46/J SV (S)	SV (S)	46/J SV (S)	SV (S)
8	43/1°C	SV (S)	45/1°	–	53/–	SV (M)
9	45/J SV (S)	SV (S&M) PVCs	48/J SV (S)	SV (S&M)	55/J SV (S)	SV (S)
10	40/J	SV (S)	37/J	SV (M)	38/J	SV (S&M)
11	43/–	SV (S) PVCs	38/–	SV (S&M)	55/–	SV (S)
12	55/–	SV (S)	57/–	SV (S)	53/–	SV (S&M)

Key: 1° = first degree block; J = junctional rhythm; SV (S) = single; (M) = multiple (pairs or short runs of supraventricular tachycardia); PVC = premature ventricular complex.

[a]HR (beats/min) calculated from longest two consecutive R–R intervals during breath holds.

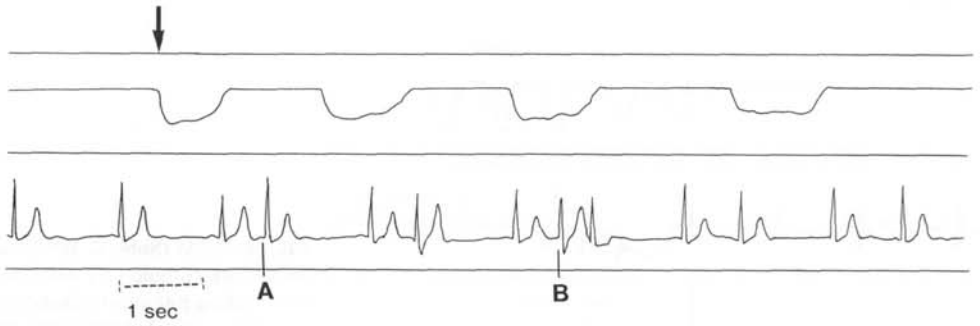

FIG. 1—ECG (Subj. 11, DS10) at end breath hold (↓) and corresponding pneumotachograph tracing (*top*). *Note* the supraventricular extrasytoles occurring both singly (*A*) and in a pair (*B*).

Premature ventricular contractions were observed in three subjects immediately after BH termination in the DS5 condition (Fig. 3). No PVC or SV(M) were noted during breath holding. No complex (multiformed or repetitive) ventricular arrhythmia was observed.

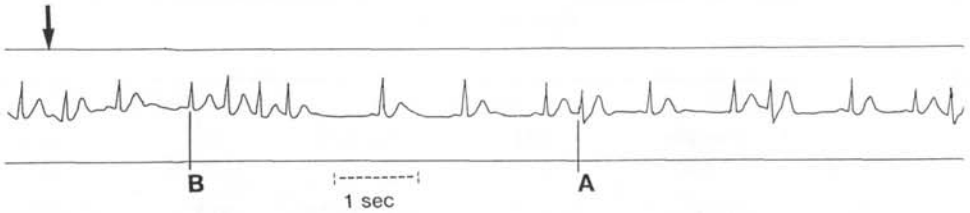

FIG. 2—ECG (Subj. 6, DS10) at end breath hold (↓). *Note* the supraventricular extrasystoles occurring singly (*A*) and as a short burst of supraventricular tachycardia (*B*) following cessation of breath holding.

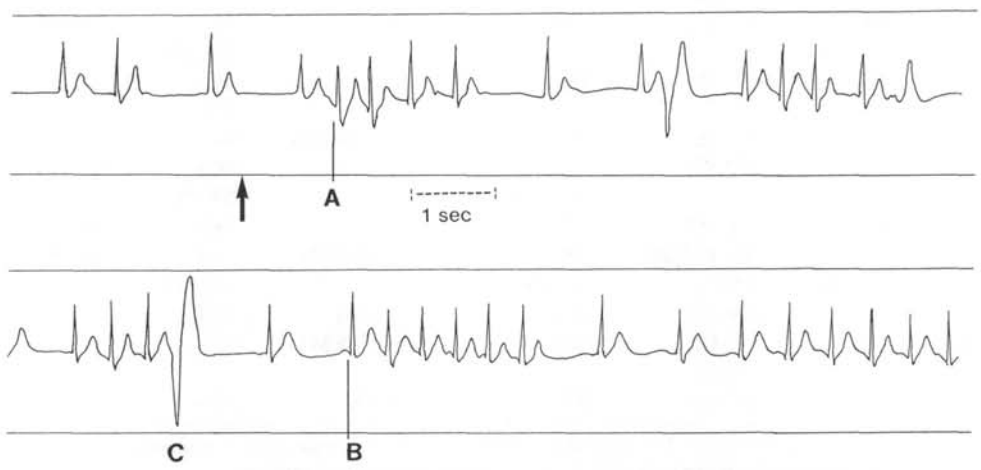

FIG. 3—ECG (Subj. 3, DS5) at end breath hold (↑). *Note* the supraventricular extrasystoles (*A*), a short run of supraventricular tachycardia (*B*), and premature ventricular complexes (*C*) after cessation of breath holding.

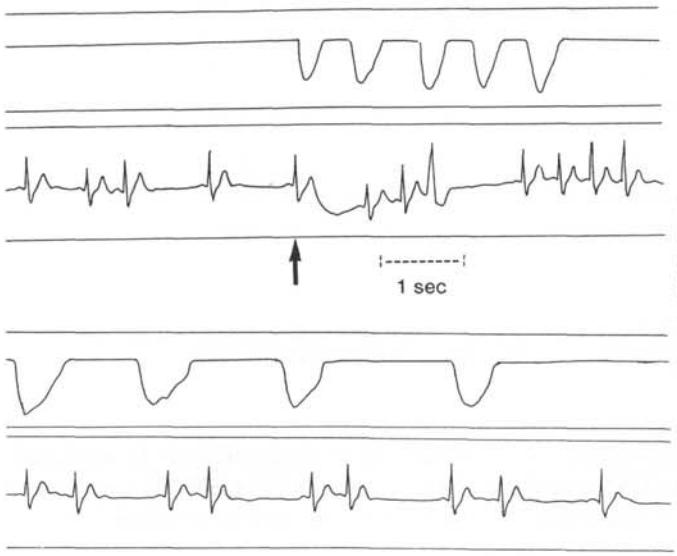

FIG. 4—ECG (Subj. 4, WS10) at end of breath hold (↑) with corresponding pneumotachograph recording (*top*); continued on lower two tracings. *Note* the temporal association between respiration and atrial bigeminy.

DISCUSSION

One of the unique observations of the present study is the high incidence of supraventricular ectopic arrhythmias immediately after breath holding (11 of 12 subjects in 29 of 36 submersions). Although the occurrence of ectopic arrhythmias have been noted in both air (20) and water after breath holding (13, 15–18, 21), they have not been described in detail. Hong et al. (13) reported premature atrial beats in two of five Korean Ama divers immediately after breath holding but did not elaborate further. Both Scholander et al. (15) and Olsen et al. (21) noted ectopic arrhythmias during recovery from breath holding but did not describe their frequency or character.

The comparatively high incidence of supraventricular ectopic arrhythmias observed in the present study immediately after breath holding may have been due, at least in part, to the experimental condition, viz. upright resting submersions with unacclimatized subjects wearing protective clothing. This is somewhat different from the methodology employed in the earlier study by Hong et al. (13) in which five lightly clothed, acclimatized subjects were investigated. These differences may be relevant to the etiology of the arrhythmias observed. For example, both the amount of clothing protection and degree of cold acclimatization can affect the magnitude of the initial cardiorespiratory responses to cold-water immersion (1). Furthermore, both body orientation and breath holding can influence venous return and thus cardiac function during immersion.

In attempting to understand the underlying mechanisms, it is important to distinguish between the ectopic arrhythmias and the frequently documented vagally induced rhythms. It is noteworthy that the majority of the ectopic arrhythmias observed immediately after breath holding occurred within 10 s of restoration of breathing. It is postulated that these arrhythmias may have been due to conflicting chronotropic inputs to the heart. With continued submersion, both positive and negative chronotropic inputs to the heart are most likely to be occurring simultaneously just after the cessation of breath holding. The negative chronotropic effect will result from a vagal drive which, although smaller than that seen during apneic face immersion, has been reported to be present in snorkel-breathing, face-immersed humans (14). A positive chronotropic effect may result from continued sympathetic stimulation from the peripheral cold receptors in areas other than the face, the cyclical reduction of vagal drive with the restoration of respiratory movement, and postvagal tachycardia (22).

It is suggested that the ectopic supraventricular arrhythmias occurring immediately after breath holding are most likely when the positive and negative chronotropic influences on the heart, consequent to sympathetic and parasympathetic stimulation, are elevated to a corresponding intensity. Such a situation may have been created in the present experiments; the similar maximum BH times observed in air and cold water (Table 2) suggest that the provision of protective clothing attenuated the normally predominant sympathetic drive, whereas the lower HRs recorded during submersion, compared to at rest in air (Table 1), suggest that a vagal drive was also present.

The suggestion that an augmented sympathetic and parasympathetic influence on the heart may provoke these arrhythmias is supported by the absence or reduced incidence of arrhythmias in situations where the sympathetic or parasympathetic response predominates. For example, ectopic arrhythmias have only rarely been

reported during both head-out, naked immersions in cold water (sympathetic predominance) or face-only immersion (parasympathetic predominance). It has been suggested (17) that hypoxia may also be a contributing factor, but this is unlikely to have been present in the current experiments given the relative inactivity of the subjects and the average duration of submersion.

Although the cause of the arrhythmias immediately after breath holding is of physiologic interest, from a practical viewpoint their clinical implications are unclear. The evidence from the present study suggests that although the arrhythmias occur in cold water in certain conditions, the majority have little clinical importance, being of short duration, supraventricular in origin, and producing no symptoms. Alternatively, their occurrence may be significant in susceptible individuals, such as those with underlying conduction defects. It is concluded that after breath-hold termination during cold-water submersion there may be a short time in which the heart is particularly susceptible to ectopic arrhythmias in individuals who remain submerged. The significance of this for individuals undertaking practical pursuits such as snorkeling remains to be determined.

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