

FACE IMMERSION BRADYCARDIA: COMPARISON OF SWIMMERS AND NONSWIMMERS<sup>1</sup>

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**Abstract.** A profound bradycardia may be exhibited by waterfowl and aquatic mammals when they dive underwater in search of food. A similar response occurs in humans diving underwater or simply wetting the face while breathholding. This bradycardia is mediated by the parasympathetic nervous system (vagus). We investigated whether the extent of this apneic face immersion bradycardia, or bradycardia during dry apnea, is greater in actively training competitive swimmers than in nonswimmers. Eight competitive swimmers and eight age/sex matched nonswimmers each performed apneic face immersion and dry apneic maneuvers while prone. Resting cardiac cycle (interval) duration was not significantly different between these groups, but the swimmers had a significantly longer interval duration (lower heart rate),  $P < 0.05$ , and a greater percentage decrease of interval duration during both apneic face immersion and dry apnea. Swimmers—but not nonswimmers—demonstrated cardiac arrhythmias considered to be vagally mediated during face immersion. These results indicated that competitive swimmers in active training may have a greater range of parasympathetic nervous system (vagal) control of the heart as well as the well-accepted greater resting level of vagal tone.

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Certain waterfowl and aquatic mammals exhibit a profound bradycardia upon diving underwater, involving heart rates as low as 6 beats per minute (bpm) and persisting despite the increased physical activity as the animal searches for food. The bradycardia is accompanied by intense peripheral vasoconstriction preserving the body's oxygen stores for the vital organs. This response pattern, called the "diving reflex," has also been observed in humans (Irving 1963, Elsner *et al* 1966, Olsen *et al* 1962). Irving (1963) observed that diving bradycardia was more pronounced and appeared more regularly in persons accustomed to swimming than in nonswimmers. Craig (1963), however, reported that diving bradycardia was "as prominent in poor swimmers as in those subjects who were familiar with the water." Craig's diving data were determined when the subjects, dressed in wet

suits, were submerged to the bottom of a pool 2.3 m deep, and control measurements were made when the subjects were prone on the surface of the water breathing through a snorkle tube. Thus, the variable was submersion and not face wetting. This is an important distinction because the reflex has been demonstrated in response to merely wetting the nasal area of the face, or "face immersion," especially if the face immersion is accompanied by apnea (Elsner *et al* 1966, Wayne and Killip 1967, Hong *et al* 1970, Strømme *et al* 1970, Bove *et al* 1968, Hutinger 1971, Frey and Kenney 1977).

Wayne and Killip (1967) compared face-immersion bradycardia in sedentary subjects with that in oarsmen who were actively training. The resting heart rate of the oarsmen was lower, but the percentage change with apneic face immersion or breathhold was no greater than the percentage change in the sedentary subjects. Strømme *et al* (1970)

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also observed significant bradycardia with apneic face immersion in healthy subjects 15 to 61 years of age, but found no relationship between level of physical fitness, as measured from the Harvard step test, and degree of bradycardia.

Other researchers have observed a significantly greater degree of bradycardia with apneic face immersion in individuals who are physically trained. Hong *et al* (1970) reported that experienced divers demonstrated greater face-immersion bradycardia than did non-divers. Bove *et al* (1968) observed an augmentation of the bradycardic response to apneic face immersion after a non-swimming training program, and Hutinger (1971) reported that the bradycardic response to face immersion was exhibited by competitive swimmers, 12 to 22 years of age.

The purpose of our study was to determine whether the degree of bradycardia elicited by face immersion is greater in competitive swimmers who are regularly exposed to the water than in nonswimmer control subjects.

#### METHODS

Two groups of students matched for age and sex were studied. Group I, the swimmers, consisted of 6 males and 2 females, 15-20 years of age, all of whom had been swimming competitively 4 or more years, and who at the time of the study regularly spent large periods of time in the water. The swimming program for these subjects involved two 45 min workouts each day. A 400 m warm-up swim was followed by a period of interval training during which the subjects swam 100 m sprints with a recovery

period between sprints. Training distance totaled approximately 2000 m each day. This program had been in effect approximately 3 months when the experiments were performed. The subjects spent many additional hours a day in the water, and most participated in additional year-round swimming programs. Group II, the nonswimmers, also consisted of 6 males and 2 females between 15 and 22 years of age. Their physical activity ranged from "no regular activity" to occasional jogging or social basketball.

The face immersion/breathhold procedures used with both groups were described previously (Frey and Kenney 1977). Breathholding with or without immersion was performed with subjects prone on a special table which incorporated a water-filled sink at one end. Subjects were instructed to hold their breath after a slightly larger-than-normal inspiration and to maintain the breathhold as long as they could with comfort, *i.e.*, to each subject's individual endpoint. For face immersion maneuvers, subjects lowered their faces into the water to a depth sufficient to wet the nasal and mouth areas while keeping the ears dry. A sling across the sink supported the forehead. Water temperature was maintained at 27 °C.

An electrocardiogram was recorded for measurement of cardiac cycle duration on a beat-by-beat basis; it was examined also for evidence of cardiac arrhythmias. The single longest beat interval and the longest 5 consecutive-beat period for which the mean interval length was determined were identified for each participant during each experiment. Group I and Group II means for these indicators of apneic face immersion or dry apneic bradycardia were compared by t test, with  $P \leq 0.05$  used as the criterion for statistical significance.

#### RESULTS

A typical response of a subject from Group I to apneic face immersion is shown in figure 1. We observed a tachy-

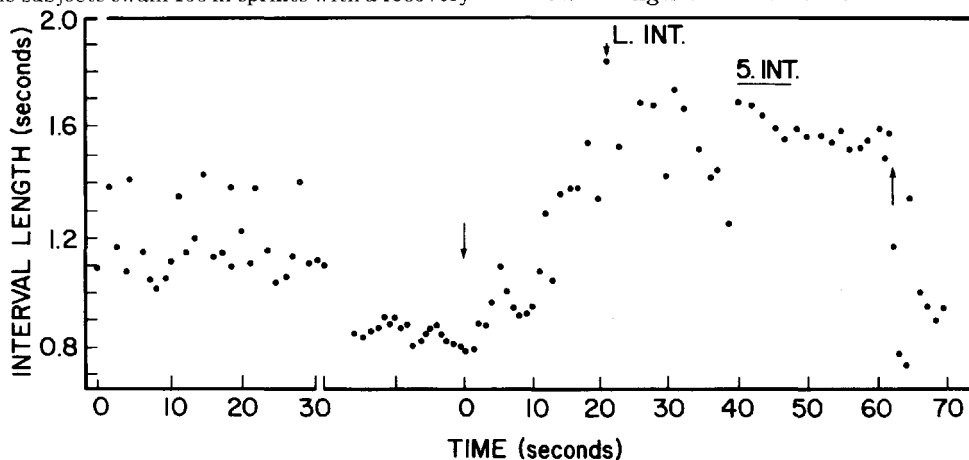


FIGURE 1. A typical response to face immersion by a trained subject. Shown on left is a beat-by-beat plot (dots) during a resting control period. Immersion and initiation of breathhold is indicated by first arrow. Emergence is indicated by final arrow. Longest single beat (L. INT.) and longest consecutive five beats (5. INT.) are indicated.

cardia immediately preceding breathhold and face immersion (indicated by the first arrow) and a prompt increase in interval length thereafter. The longest interval indicated in figure 1 (L.INT) was 1844 milliseconds (equivalent to a heart rate of 32 bpm) and was reached 21 seconds after the initiation of apneic face immersion. Bradycardia was maintained until emersion when breathing was resumed (indicated by the second arrow in fig. 1).

Mean resting control (prone) interval durations for Groups I and II respectively were 1051 milliseconds (heart rate 57 bpm) and 942 milliseconds (64 bpm), which were not statistically different (table 1). Responses to apneic face immersion, however, showed significant differences between groups. Mean longest interval for the swimmers was 1536 milliseconds (46% longer than control) as compared with 1140 milliseconds (21% longer than control) for the non-swimmers. The mean longest 5-beat interval duration was also greater for swimmers 1394 vs 1088 milliseconds) and represented a greater percentage decrease (33% vs 16%). Breathhold durations during face immersion did not show significant differences between groups, averaging 56 sec for Group I (range 30 sec to 72 sec) and 43 sec for Group II (range 16 sec to 74 sec).

We also compared interval lengths for

breathhold without facial wetting (table 1). The trained subjects reached significantly longer beat intervals (lower heart rates) for both a single beat (1371 vs 1128 milliseconds) and for 5 consecutive beats (1274 vs 1068 milliseconds). These increases in interval represented a greater percentage change for the swimmers.

Of the 8 swimmers tested, 4 exhibited arrhythmias such as wandering pacemaker and junctional rhythm during apneic face immersion and dry apnea. One swimmer who repeatedly displayed such arrhythmias during breathhold and face immersion experiments showed no arrhythmias during an experimental session conducted several months later when he was no longer involved in the rigorous swimming program (although he remained very active). The arrhythmias of this subject occurred early in the breathhold or face immersion and did not appear to be a function of breathhold duration. No arrhythmias were observed in any of the subjects in the nonswimmer control group.

DISCUSSION

Our research indicates that face immersion bradycardia is demonstrated in competitive swimmers to a greater degree than in age/sex matched nonswimmer controls. This finding confirms the speculation of Hutinger (1971) who

TABLE 1  
*Cardiac interval duration for swimmers and nonswimmers.*

	Swimmers*	Nonswimmers*	Group Diff.
<b>Control Prone</b>			
Interval msec (bpm)	1051 ± 102 (57)	942 ± 175 (64)	109
<b>Apneic Face Immersion</b>			
L.INT** msec (bpm)	1536 ± 234 (39)	1140 ± 206 (53)	396 <sup>+</sup>
difference from control	486 <sup>+</sup>	198 <sup>+</sup>	
5.INT/5† msec (bpm)	1394 ± 193 (43)	1088 ± 202 (55)	306 <sup>+</sup>
difference from control	343 <sup>+</sup>	146 <sup>++</sup>	
<b>Dry Apnea</b>			
L.INT** msec (bpm)	1371 ± 214 (44)	1128 ± 207 (53)	243 <sup>++</sup>
difference from control	320 <sup>+</sup>	187 <sup>+</sup>	
5.INT/5† msec (bpm)	1274 ± 174 (47)	1068 ± 208	206 <sup>++</sup>
difference from control	223 <sup>+</sup>	114 <sup>+</sup>	

\*Mean ± S.D. (No. Determinations).  
 \*\*Longest interval duration.  
 †Longest mean interval for 5 consecutive beats.  
<sup>+</sup>P < 0.01 to 0.001.  
<sup>++</sup>P < 0.05.

observed significant bradycardia with face immersion in competitive swimmers but did not study nonswimmers. Our study is consistent with previous observations by Irving (1963), who observed a stronger bradycardic response to diving in experienced swimmers than in nonswimmers and by Hong *et al* (1970), who reported greater bradycardia during face immersion in their experienced divers than in nondivers. Craig (1963) observed no differences in bradycardia between swimmers and nonswimmers, but his study monitored the effects of body submersion, not face wetting.

Strømme *et al* (1970) did not find a relationship between physical fitness as measured by the Harvard step test and face immersion bradycardia, but their subjects were not at a high level of physical fitness and were not reported to be actively training. Whayne and Killip (1967) reported on subjects who were oarsmen in active training and presumed to be at a high level of physical fitness, yet they did not exhibit greater face immersion bradycardia than a control group, suggesting that regular exposure and training in the water may be the significant factor. Bove *et al* (1968), observed in a longitudinal study, however, an increased degree of face-immersion bradycardia after a nonaquatic training program, which accompanied an increase in physical fitness. Thus, it may be that the face-immersion bradycardia depends not only on the water but is enhanced at high levels of regular physical activity greater than that performed by subjects in these other studies.

Resting bradycardia is usually observed in subjects who regularly perform physical exercise (Scheur and Tipton 1977). The absence of a statistically significant difference between swimmers and nonswimmers in our study probably results from the relatively small sample size. The lower resting heart rate of the physically conditioned individual relative to the unconditioned has been attributed to an increased vagal discharge, a reduced sympathetic tone, or a combination of both (Ekblom *et al* 1973, Frick *et al* 1967, Hall 1963, Raab

*et al* 1960). Face-immersion-induced bradycardia is also mediated by a vagal outflow, as demonstrated by its abolition after the administration of the parasympathetic nervous system blocking drug atropine-sulfate (Elsner *et al* 1966) and by the occurrence of arrhythmias, reported to be vagally mediated, exhibited in the present study as well as in other investigations (Olsen *et al* 1962, Whayne and Killip 1967). Our results indicating a greater percentage increase in interval length with both apneic face immersion and dry apnea in swimmers suggest that the magnitude of vagally-mediated change in heart rate is increased with swimming training as well as the greater level of tonic vagal control indicated by a lower resting heart rate.

It appears that extent of bradycardia is not a function of length of breathhold since we did not observe significant differences between duration of breathhold between groups and since the subject who consistently exhibited the most profound bradycardia had a relatively short breathhold time of about 30 sec. Extensive swimming training and/or regular exposure to water by swimming enhanced the extent of bradycardia experienced during apnea both in air and with face immersion. This finding suggests two alternative explanations which remain to be examined: face-immersion bradycardia is selectively enhanced as a result of training in the water, or it is enhanced only at very high or rigorous levels of physical training.

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