Lack of effect of 24 atm abs environment on face immersion bradycardia in human subjects

To the Editor

Two earlier studies have reported on testing the effects of hyperbaric exposure on the well-recognized bradycardia that accompanies apneic face immersion in animals and humans. Both were performed during the decompression stages of saturated dives, one to 12.5 atm abs (1), the other to 27 atm abs (2). The results of the two reported studies are contradictory, one showing enhanced bradycardia (1), the other showing no influence of hyperbaria on face immersion bradycardia (2).

Our participation in the November 1995, 24 atm abs heliox saturation dive at the Japan Marine Science and Technology Center (JAMSTEC) at Yokosuka, Japan, provided an opportunity to examine this issue again and to ask the following questions: Does hyperbaric exposure alter face immersion bradycardia? If so, is there a consistent relationship with increased pressure? Answers to these questions might be of some practical importance relating to the physiologic consequences of apneic dives at great depth, as might occur with breath-holding during brief removal, accidental or otherwise, of face mask and mouthpiece.

The phenomenon of hyperbaric bradycardia, a sustained lowering of heart rate observed without immersion during sojourn in hyperbaric environments, has been well documented (3). The response is sometimes seen to gradually disappear with time at high pressure. Hyperbaric bradycardia is unlikely to be related to the topic of the present investigation, but we were nevertheless alert to its possibility.

Four male subjects, members of the JAMSTEC staff, participated in the dive. The dive consisted of 4 days (Days 1–4) for pre-dive control at 1 atm abs in air, 1 day (Day 5) for compression, 7 days (Days 6–12) for saturation at 24 atm abs in He–O₂, 8 days (Days 13–20) for decompression, and 4 days (Days 21–24) for post-dive control at 1 atm abs in air. Preparations for the tests and their performance adhered to a pre-established schedule by which they were conducted between 1500 and 1600 h on each of the test days. Control tests were performed by breath-holding in air alternating in a random order with face immersions.

The subjects rested, sitting quietly, for 10 min before each test. During this time and throughout the tests subjects were comfortably seated in front of a table on which a basin, approximately 30 cm in diameter, was placed. The test consisted of a 15 s of normal breathing period and 45 s of breath holding or face immersion. Each test was performed in triplicate, allowing 2 min between tests. Subjects were instructed to inhale in an approximation of inspiratory reserve volume. They were cautioned to avoid increased intrathoracic pressure as in a Valsalva maneuver. Water temperature was 30° ± 1°C, the same temperature used in the previously mentioned studies (1,2).

The experiments were performed in the pre-dive control period on Days 2 and 4, on Days 6, 8, 10, and 12 during saturation at 24 atm abs, on Days 14 (21 atm abs) and 19 (6 atm abs) during decompression; and on Days 21 and 23 during the post-dive control period. Heart rates were recorded by echocardiogram leads attached in the lead II position. The data of Days 2 and 4 were averaged as the pre-dive control value, Days 6, 8, 10, and 12 as the 24 atm abs value, and Days 21 and 23 as the post-dive value, and two individual values of Days 14 and 19 were taken as decompression values. Statistical difference of values among various pressures was determined by one-way analysis of variance and values between corresponding control and face immersion or breath-hold in air were compared by paired t test. Differences were considered significant at P < 0.05. Heart rate responses to face immersion or breath hold in air are shown in Table 1.

Moderate face immersion-induced bradycardia (average low rate 13% below resting heart rate) was recorded throughout. There were no significant differences in the test results comparing face immersions with the same procedure performed throughout the entire period: pre-dive control, compression, decompression, and post-dive control.

Our results differ from those of Hong et al. (1) who reported an enhanced bradycardia during hyperbaric exposure; in turn, they agree with those of Kerem and Salzano (2) who reported no differences among the face immersion breath-hold procedures during hyperbaric exposure.
Table 1: Responses of Heart Rate to Face Immersion or Breath Holding During Exposure at Different Atmospheric Pressures\(^a\)

<table>
<thead>
<tr>
<th>Condition Pressure</th>
<th>Heart Rate, beats/min</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
</tr>
<tr>
<td>Predice control, 1 atm abs</td>
<td>68 ± 5</td>
</tr>
<tr>
<td>Saturation, 24 atm abs</td>
<td>66 ± 3</td>
</tr>
<tr>
<td>Decompression, 21 atm abs</td>
<td>68 ± 4</td>
</tr>
<tr>
<td>Decompression, 6 atm abs</td>
<td>66 ± 3</td>
</tr>
<tr>
<td>Postive control, 1 atm abs</td>
<td>77 ± 5</td>
</tr>
</tbody>
</table>

\(^a\)Values are means ± SE. Values for control were averages for the first 10 s (1–10 s) of test period and those for breath hold and face immersion were average for the last 40 s (15–60 s) of corresponding breath-hold and face-immersion test. Effect of transient heart rate changes immediately before and after breath holding was thus eliminated from the data. \(^b\)\(p<0.05\) vs. corresponding control value.

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REFERENCES
