Detection of venous gas emboli after repetitive breath-hold dives: case report

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ABSTRACT

Introduction: Neurological symptoms after breath-hold (BH) diving are often referred to as "Taravana" and considered a form of decompression sickness. However, the presence of "high" gas embolism after BH diving has never been clearly shown. This study showed high bubble formation after BH diving

Materials and methods: We performed transthoracic echocardiography on a 53-year-old male spearfishing diver (180 cm; 80 kg; BMI 24.7) 15 minutes before diving and at 15-minute intervals for 90 minutes after diving in a 42-meter-deep pool. Number of dives, bottom time and surface intervals were freely determined by the diver. Dive profiles were digitally recorded for depth, time and surface interval, using a freediving computer. Relative surface interval (surface interval/diving time) and gradient factor were calculated.

Results: High bubble grades were found in all the recorded echocardiograms. From the first to third recording (45 minutes), Grade 4 Eftedal-Brubakk (EB) bubbles were observed. The 60-, 75- and 90-minute recordings showed a reduction to Grades 3, 2 and 1 EB. Mean calculated GF for every BH dive was 0.22; maximum GF after the last dive was 0.33.

Conclusions: High bubble grades can occur in BH diving, as confirmed by echocardiographic investigation. Ordinary methods to predict inert gas supersaturation may not able to predict Taravana cases.

INTRODUCTION

Acute manifestations of neurological symptoms after breath-hold (BH) diving have been described since 1965 by Cross in pearl divers of the Tuamotus [1] and by Paulev in Danish military personnel previously exposed to pressure during submarine escape training [2]. In the same year, using the U.S. Navy no-decompression limits Lanphier calculated the likelihood of developing decompression sickness after repetitive breath-hold dives (BH dives) using specific parameters such as average diving time, depth and surface intervals [3]. More recently an alternative theoretical table was developed and proposed for BH diving [4,5].

The syndrome of neurological symptoms is usually defined as a decompression sickness of breath-hold divers. Often called "Taravana," it presents as a complex clinical picture characterized by different combinations of complete or partial hemiparesis, visual problems, hearing or speech impairment, sensory loss, and minor symptoms such as nausea, dizziness and headache. [6] In severe cases it can lead to loss of consciousness and death. [7]

Many Taravana cases have been described since 1965, and multiple cerebral injuries were described in two case reports by Kohshi [8-10].

Many possible pathogenic mechanisms have been

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considered as causes for Taravana, including arterial gas embolism (AGE), [11,12] cerebral in situ bubble formation and inert gas accumulation [7,13] similar to decompression sickness (DCS) in scuba diving.

AGE triggered by lung over-expansion is considered the most likely cause of Taravana, occurring after a single deep dive, especially if the diver used glosso-pharyngeal insufflation manoeuvres. Even if the genesis of these particular Taravana cases (only one deep dive) is still doubtful, inert gas accumulation cannot be excluded [14].

Cerebral in situ bubble formation is considered questionable because of the high cerebral blood flow, while the hypothesis of inert gas tissue saturation is believed possible [11]. According to this hypothesis, tissue supersaturation could be possible in cases of repetitive deep BH diving with a long bottom time and short surface interval.

This hypothesis is supported by Schaefer [15], who showed a ”foamy” aspect of peripheral blood in Ama divers immediately after repetitive BH diving. This is considered as a sign of bubbling due to nitrogen supersaturation by Radermacher [16], who demonstrated nitrogen accumulation in venous blood, and by Spencer, who reported detecting bubbles in BH divers by precordial Doppler monitoring [17].

Recently low-grade intravascular bubbles (Spencer’s Grade 1) was also detected in an Ama diver after long dive times with short surface recovery [18]. Bubbles were also observed in the ocular tear film after both dry-chamber and in-water recreational diving [19], and DCS was investigated in sea turtles [20] and in whales [21].

On the other hand, Boussuges and colleagues did not find any circulating bubbles in breath-hold divers after underwater fishing dives [22].

This case report shows “high” bubble formation after BH diving, recorded by an echocardiography protocol and agrees with papers that reported bubbles recorded by precordial Doppler techniques

MATERIALS AND METHODS

Deep pool test

One male spearfishing diver, age 53; height 180 cm; weight 80 kg; body mass index (BMI) 24.7 was investigated by transthoracic echocardiography before and after a training session of consecutive breath-hold dives in a swimming pool that allowed diving to a depth of 40 meters (air temperature 36°C; water temperature 33°C).

The numbers of dives, warm-up times, bottom times and surface intervals were freely determined by the diver following his usual training methods. The subject was an active, experienced spearfishing diver often involved in international championships. He did not perform any compressed-gas diving prior to the breath-hold exposures and during the 30 days after the tests.

The subject had no historic or clinical evidence of arterial hypertension, cardiac, pulmonary or any other significant disease. He was non-smoker and had previously suffered a Taravana episode.

Information about age, gender, height and weight was recorded and BMI was calculated. Heart rate, diastolic and systolic arterial pressure were monitored and recorded 15 minutes before and 15 minutes post diving.

The diving profiles were recorded (measurements every two seconds) for each dive, including depth, diving time, bottom time and surface interval using a freediving computer (UP-X1 Omersub Spa Monza Brianza Italy). The relative surface interval (surface interval/diving time) and the gradient factor (GF) were calculated.

Calculating the GF is a way to measure nitrogen supersaturation in the “leading” tissue at the end of each dive – i.e., the compartment showing the higher calculated inert gas supersaturation at any given time during ascent or post surfacing, a.k.a. “leading tissue.” This approach theoretically predicts the calculated maximum value (M-value) allowed for all 16 tissues considered in the Bühlmann ZH-16 Model C. Calculations of GF were performed for all 16 tissues, and the maximum GF value in the leading tissue at the end of the dive was recorded.

Table 1: Summary of anthropometric data

anthropometric data		
age	53	
height	180	
weight	80	
BMI	24.7	
heart rate	before 68	after 76
diastolic pressure	before 70	after 85
systolic pressure	before 130	after 140

The theoretical possible accumulation of inert gas during repeated breath-hold dives was investigated using DAN Europe's original Diver Safety Guardian dive simulator, normally used for scuba diving, and modified to simulate the specific characteristics of BH diving.

This simulator allowed us to study the BH diving profiles using the Bühlmann ZH-L16 model C algorithm to estimate nitrogen uptake in fast compartments and ultimately to analyze the GF.

Echocardiography protocol

Echocardiography images were recorded 15 minutes before the start of the dive training session and every 15 minutes after until 90 minutes after using a commercially available instrument (MyLab 5, Esaote SPA, Florence, Italy) with cardiac probe (2.5-3.5 MHz). All echocardiograms were performed with the subject lying on his left side and breathing normally. Recording time was 20 seconds, both in a resting condition and after three knee bends. All frames were saved in the hard drive for subsequent analysis.

Ultrasound recordings were made by a technician with more than 500 hours in recording experience in transthoracic echocardiography, bubble grade count, and evaluation. Bubbles were graded according to the EB scale as follows [23]:

0 – no bubbles;
1 – occasional bubbles;
2 – at least one bubble per four heart cycles;
3 – at least one bubble per cycle;
4 – continuous bubbling, at the least one bubble cm^2 in all frames;
5 – "whiteout" – impossible to see individual bubbles.

RESULTS

One male spearfishing diver was studied, with heart rate, diastolic and systolic arterial pressure reported respectively: 68 bpm and 70/130 mm/Hg 15 minutes before the dive session; 76 bpm and 85/140 mm/Hg 15 minutes after the training session (Table 1).

The diver did not perform any pre-dive exercise or training, did not use a wetsuit (for a comfortable temperature in the water) and made his dives using constant weight without the use of any device to simplify the dives.

We found bubbles in all the echocardiograms recorded at 15-minute intervals over a 90-minute period post-dive. From the first to the third recording (a span of 45 minutes) we observed Grade 4 bubbles according to the EB scale (i.e., continuous bubbling, with at least one bubble per cm^2) (Figure 1). The last three recordings (at 60, 75 and 90 minutes) showed a reduction in bubbles (Grades 3, 2 and 1 EB). Recordings were analyzed and graded separately by two technicians with experience in transthoracic echocardiography, bubble evaluation; analyses were performed frame by frame; and no disagreement occurred.

The subject performed four shallow dives before starting the deep dive series, then made eight deep dives (to the bottom of the pool at 42 meters' depth) and one additional dive at 15 minutes (the approximate half-time mark [14.8 meters] of the session) and a final dive to 16.9 meters after the last deep dive (Figure 2).

The median depth was 40.2 meters (range 6.2 to 41.7 meters), mean diving time was 140.9 +/- 42.1 seconds; mean of surface interval was 593.8 +/- 540 seconds and the relative surface interval (ratio between the time spent on the surface/ underwater was 3.4 (Table 2).

The diver spent a long time at the maximum depth in every dive and especially on the dives reaching the bottom of the swimming pool. The mean bottom time in the 14 dives was 43.4 +/- 27.2 seconds, while the mean bottom time in the eight dives at the bottom (to 42 meters) was 53.6 +/- 19.1 seconds.

The analysis of the diving profile shows that the GF at the end of the training session was 0.33 and the mean was 0.22 +/- 0.13.

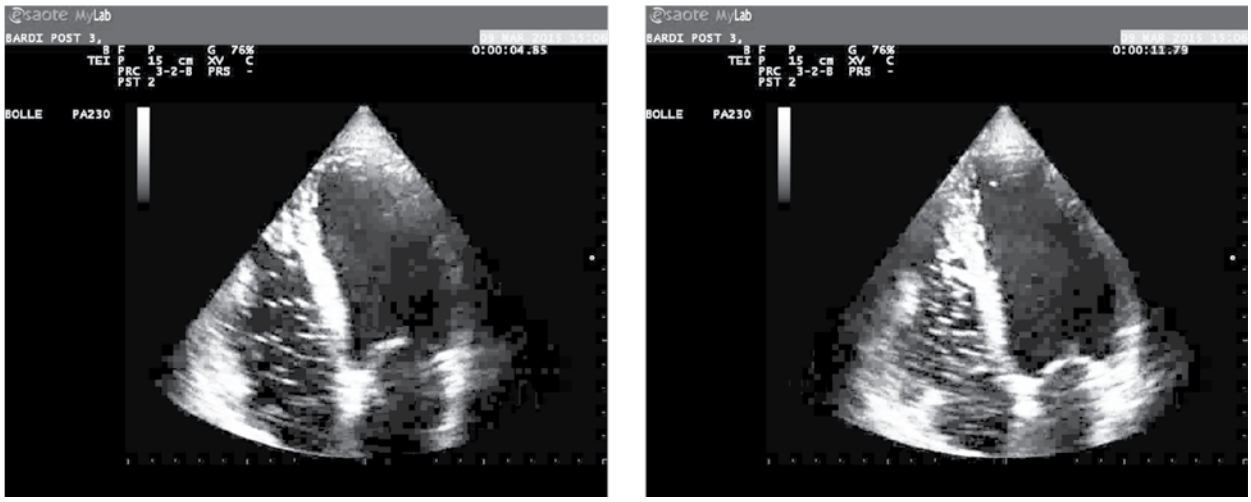

Figure 1: Case of high-grade bubbles after repetitive breath-hold dives.
Left: bubbles at rest. *Right:* bubbles after standard exercise (leg bending).

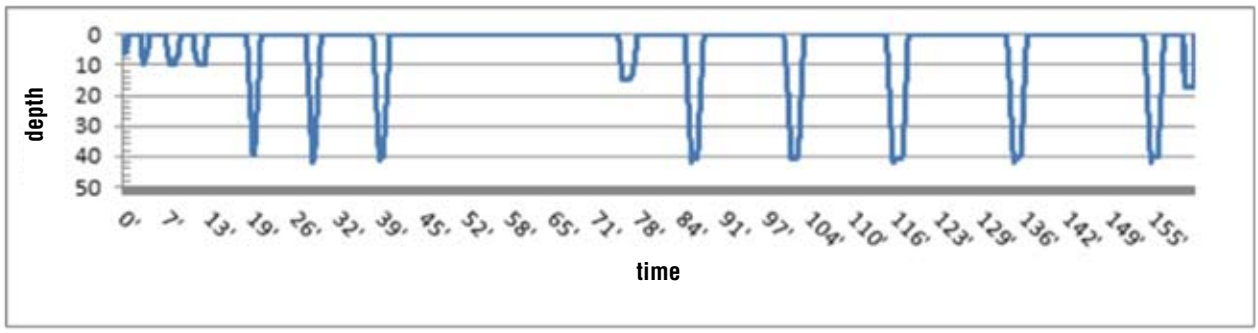

Figure 2: Diver made eight deep dives, one dive at 15 m and a final dive at 16.9 meters.

DISCUSSION

Our data show clearly that high bubble grades can occur in BH diving. Our study was made in the special environment of a very deep swimming pool; in this context it was very easy to conduct testing immediately after the dives and continue recording for all the time necessary – without technical problems, such as exhausting the echocardiograph's battery pack.

These results agree with Spencer's paper [15] and, more recently, the Lemaître manuscript [16], where the authors declared they had heard bubbles at precordial Doppler recording.

Our results are even more important if we consider that real spearfishing sessions frequently involve more severe exposure than those studied in our pool tests. In fact, the majority of spearfishing BH divers, when preparing for a competition, usually train intensively for six or seven consecutive hours over several days.

We found high bubble grades in one subject who performed only 14 dives (albeit very deep and long dives) and with a recovery at surface three times longer than the diving time. It is easy to imagine that in real spearfishing conditions the possibility of producing bubbles can increase dramatically.

Moreover, analysis of the diving profile does not show any particular increase of GF: The only data that capture our attention was the long time spent at the bottom, a typical characteristic in spearfishing divers who are the BH divers apparently more affected by Taravana.

This, even if it is still speculation, could indicate that longer time spent at the bottom is an important factor to develop Taravana and agrees with the evidence that

Table 2: Summary of anthropometric data

diving conditions	
warm-up (numbers of dives in shallow water)	4
number of dives	114
deep dives (m)	40.25 Range 6.2 41.7
diving time (sec)	140.9 +/- 42.1
mean of surface time (sec)	593.8 +/- 540
relative surface interval	3.4
mean of GF	0.22 +/- 0.12
maximum GF at the end	0.33

this disease appears usually in spearfishing BH divers. The confirmation that high bubble grades can occur after BH diving makes it reasonable to believe that Taravana can be caused, at least in some cases, by mechanisms similar to those involved in scuba diving decompression illness.

On the other hand, the very low GFs found at the end of dive sessions (0.33), at least in this case, seem to indicate that the approach based on theoretical M-value calculation cannot be used to evaluate BH diving-induced decompression stress

These results agree with Egi's and Thorsen's papers [4,5] that suggest the need to use continuous compartment half-lives in BH diving, and further suggest caution in the use of "classic" modalities to predict Taravana and also with the paper that reported bubbles recorded by Precordial Doppler techniques [16,17].

Considering that Boussuges, et al. did not find any circulating bubbles in breath-hold diving after underwater fishing dives, [22] it will be interesting to investigate the different risk factors that caused different results between Boussuges' paper and our investigation.

Limitations and speculative hypothesis

This work has some limitations: the first is that we investigated one case only; second, the special environmental conditions were helpful for the test but quite different when compared to normal BHD conditions.

However, confirmation of high bubble grades in BH diving is interesting new data, even in the presence of the above-cited limitations. Considering that the only data that captured our attention included the long bottom times, we postulate a way to explain the phenomena, even if absolutely speculative at this time. We hypothesize that during the gas uptake phase the mass of blood pooling in the lung as a result of the so-called blood-shift effect, may facilitate a higher transfer of nitrogen (from lungs to blood), as compared to what happens during the recovery time when the blood has shifted back from the lungs to the periphery, reducing the amount of blood available for gas exchange (from blood to lungs).

The increased flow in the lungs and the consequent increased diffusion surface made available by the blood-shift during the bottom phase could explain why inert gas uptake can significantly exceed its release. The extraordinary increase in depth reached by today's BH divers suggests that the possibility of significant inert gas supersaturation could become more frequent. The study of the mechanism that regulates Taravana could be essential to prevent its possible adverse consequences.

The possibility that external factors such as naval sonar could interfere with the cause of DCS in whales [21,24] confirms the many and variegated factors that may be involved in the pathogenetic mechanism of dysbaric bubble-generated injuries as well as Taravana. Thom's hypothesis, postulating that scuba-related DCS could also be involved in Taravana also needs consideration and opens new avenues for investigation [25].

Given the results of this case report a new study about Taravana – with a larger number of subjects and standardized conditions – is currently in progress, aiming at investigating whether individual predisposition to develop bubbles, such as specific genetic traits or other variants in endothelial function, for instance, could be involved with mechanisms similar to those reported in scuba diving and in the development of other BH diving-related injuries, [26,27,28].

CONCLUSIONS

High inert gas bubble grades can occur in breath-hold diving, as confirmed by our echocardiography data, making it reasonable to believe that Taravana can be caused, at least in some cases, by mechanisms linked with venous gas embolism.

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