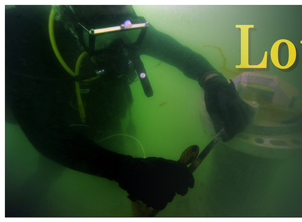


Lowering The "Bar" On Pulmonary Decompression Illness – A Case Of Tender "Chokes" After Serial Hyperbaric Chamber Exposures To 33 FSW

Hart, BB

Background

Pulmonary Decompression Illness ("chokes") is a severe form of decompression illness (DCI) thought to be due to the build-up of venous gas emboli in the pulmonary circulation and associated vasoactive response. It is a relatively rare condition usually manifesting only in divers incurring severe decompression stress (e.g. after ascents from deep dives of short duration, decompression of saturated compressed-air workers or where significant omission of required decompression has occurred) and aviators. The incidence of pulmonary DCI is estimated to be about 2% in U.S. Navy divers(2, 3), 6% in compressed-air workers (i.e. tunneling and caisson work)(4), and 6-10% for altitude exposures.(5, 6) As might be expected in cases of severe decompression stress, the majority of patients manifesting pulmonary symptoms have concurrent involvement of the musculoskeletal (51%) or central nervous (91%) systems.(7) Notably, however, significant declines in pulmonary function have been found in divers with no other DCI symptoms.(8-10)

Regardless of exposure etiology, pulmonary DCI symptoms characteristically include a triad of substernal chest pain, paroxysmal cough and dyspnea.(11) These symptoms are reliably associated with tachypnea and aggravated by deep inspiration or smoking (Behnke's sign).(12) As symptoms progress, breathing becomes progressively more rapid and shallow. The initial dry cough may become productive of frothy, blood-tinged sputum, serving as a harbinger of cardiovascular shock and right heart failure. If treated with prompt recompression therapy, pulmonary DCI symptoms are usually rapidly reversed. However, if left untreated or if bubble accumulation, obstruction and inflammatory damage to the pulmonary vasculature become sufficiently severe, cardiac decompensation, respiratory arrest and death may occur.(13,14)

Case Presentation

This case involves a healthy 23-year-old male, experienced US Navy First Class Diver and Diving Medical Technician, who was performing duty as an inside chamber attendant in support of a research protocol evaluating hyperbaric oxygen's effect on wounded warriors with mild traumatic brain injury.

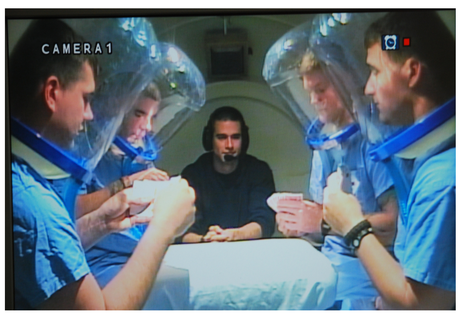
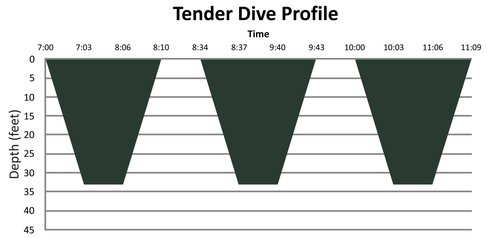


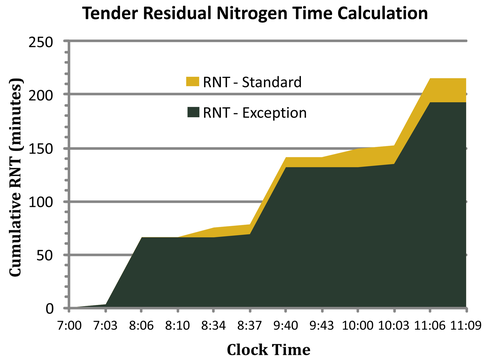
Figure #1 – example of the chamber environment

As a result, at 07:00 hrs, the tender initiated a series of three 66-minute, dry hyperbaric chamber compressions to 33 fswchamber venting and 60 minutes of subject experimental gas breathing, and a 3-minute descent, 63 minutes of sustained chamber pressurization at 33 fsw, and a 3-minute ascent. The corresponding surface intervals between the three chamber dives were 24 and 17 minutes, respectively. Throughout the chamber compressions, the tender breathed ambient chamber air. The third dive in the research protocol series was completed at 11:09. On departing the chamber area approximately one hour later, the tender remained asymptomatic.



At approximately 14:00 hrs, while seated watching a movie, the tender experienced acute onset of non-radiating substernal chest pain and dyspnea. By the time of presentation to the Emergency Room an hour later, he reported worsening 8/10 substernal chest discomfort, increasing air hunger and, as a new symptom, paroxysmal dry cough exacerbated by deep inspiration. Although the patient did complain of some mild lightheadedness, he denied concurrent difficulties with balance, coordination, motor strength or sensation. On physical exam, the patient was noted to be somewhat anxious and tachypnic, while leaning forward on his outstretched arms in a seated, tripod position. His vital signs revealed a respiratory rate 19-26 breaths per minute, pulse 126-148 beats per minute, blood pressures ranging 142-175/83-106 mmHg, but sustained 100% saturation while breathing 100% oxygen via a non-rebreathing facemask. Examination of the head, eyes, ears, nose and throat was unremarkable, except to note the patient's paroxysmal cough had now become productive of small amounts of white frothy sputum. Regardless, the patient's lungs were clear to auscultation bilaterally and, other than the noted tachycardia, the cardiac sounds were unremarkable. The abdomen was also benign and examination of the lower extremities revealed no palpable cords, tenderness or edema. The patient's neurological examination was remarkable for normal mental status, intact and symmetric cranial nerves, motor strength, reflexes, sensation and, as could be conducted with the patient remaining on the gurney, cerebellar function. An electrocardiogram demonstrated sinus tachycardia with borderline right axis deviation. A portable chest x-ray revealed no evidence of pulmonary consolidation, pleural effusions or pneumothorax. Laboratory assessment of the patient's coagulation times, cardiac enzymes, complete blood count and metabolic profile returned normal values, except for a mild postprandial elevation in the patient's blood glucose. In considering the differential, despite this patient's relatively innocuous, "No-Decompression" chamber exposure, a preliminary diagnosis of pulmonary DCI was entertained and the patient urgently referred for recompression treatment at the same hyperbaric chamber facility in which he performed tender duty earlier that day.

On arrival at the recompression chamber, after approximately one hour of continuous surface level breathing, the patient's dyspnea, paroxysmal coughing and substernal chest pain were somewhat improved, now being rated at a 5/10 in intensity. However, he remained tachypnic (20-22/min), tacycardiac (110-123 bpm) and hypertensive (146/106 mmHg). The patient's physical examination was otherwise unchanged. Consequently, recompression therapy was initiated using a US Navy Treatment Table VI at 16:09. Within ten minutes of initiating hyperbaric oxygen breathing at 60 fsw equivalent, the patient reported significant improvement in his dyspnea. By the end of the first 20-minute oxygen-breathing period, the patient stated that his substernal chest pain was now only 1-2/10 in intensity. Within five minutes of starting the second oxygen-breathing period at 60 fsw, the patient noted complete resolution of his dyspnea, cough and chest pain symptoms. In chamber vitals demonstrated a respiratory rate of 12 breaths per minute, heart rate of 104 bpm, blood pressure of 124/84 mmHg and 100% saturation on pulse oximetry monitoring. This symptomatic relief and normalization of vital signs persisted through the end of recompression treatment. On returning to ambient atmospheric pressure, the patient's physical exam was noted to be completely normal, including the resolution of the previous borderline right axis deviation recorded on electrocardiogram testing. Additionally, a post-recompression chest CT was interpreted as having no evidence of effusions, infiltrates or prior pulmonary embolism. An MRI of the brain two days later was equally unremarkable.



Discussion

From the outset, this case presented a diagnostic challenge: not because of the clinical symptoms that were reported, but because of the significant history that was not. As previously noted, pulmonary DCI generally occurs either in aviators or divers who incur severe decompression stress secondary to rapid ascent from a deep dive or saturation exposure. With the tender being free of a history of altitude or diving exposure during the preceding 72 hours, neither of these noxious, predisposing exposure situations applied. Indeed, after completing the day's research dive series, the tender was calculated to be an "O" diver in terms of his Repetitive Group Designator, having accumulated an "Equivalent Single Dive Time" of 198 minutes or, if the "RNT Exception Rule" is not applied, a maximum of 215 minutes . In either case, this tender's dive series remains within the U.S. Navy Diving Manual's "No-Decompression Limits."(15) Even when the tender's two identified risk factors for DCI are factored into the differential (i.e. mild dehydration from alcohol consumption the night prior and borderline obesity (BMI 29.8)), the estimated risk for developing DCI as a result of this chamber research dive series remains low. Table #1 provides "conditional" and "cumulative" DCI risk estimates for three common U.S. Navy air decompression algorithms.(16)

Prior to the onset of this tender's pulmonary symptoms, the three-dive, chamber exposure profile used in the traumatic brain injury research protocol was conducted uneventfully a total of 55 times: 16 of them involving this same inside tender. In retrospect, given an approximate 2% probability that DCI could still occur as a consequence of this ostensibly "No-D" dive profile, a case of DCI is not completely unexpected. What nevertheless remains unforeseeable is that any DCI encountered would be so severe. Accordingly, a range of alternative diagnoses must necessarily be entertained. Other potential conditions included in the differential, such as immersion pulmonary edema, pulmonary embolism, barotrauma and primary cardiac disease, seem unsupported by the patient's history, serial physical examinations, laboratory findings and clinical course. In particular, the tender's rapid and complete response to recompression therapy suggests that inert gas bubble accumulation in the pulmonary vascular tree was, in fact, causative in this case. Consequently, pulmonary DCI ("chokes") still remains the primary working diagnosis in this patient.

Table #1 – DCI Risk Estimates for the Tender Dive Series

Model	Last Dive Conditional P(DCS), %	Series Cumulative P(DCS), %
USN93	1.82	2.30
NMRI98	1.94	2.45
BVM(3)	1.49	1.65

(N.B.: "Conditional" DCI risk is the risk that the last dive will result in DCI given that the condition (DCI) has not yet occurred. "Cumulative" risk is the overall risk that DCI will occur as a result of completing the dive series.)

Conclusions

Pending the identification of a suitable, alternative diagnosis, this tender's case seemingly represents the first known report of pulmonary DCI ("chokes") occurring as a consequence of hyperbaric chamber exposure to a "No Decompression" dive profile.

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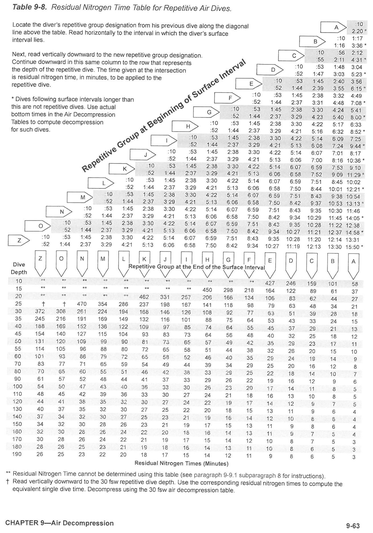
Table 9-7. No-Decompression Limits and Repetitive Group Designators for No-Decompression Air Dives.

Depth (fsw)	No-Stop Limit	Repetitive Group Designation															
		A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	Z
10	Unlimited	57	101	158	245	428	*										
15	Unlimited	36	60	88	121	163	217	297	449	*							
20	Unlimited	26	43	61	82	106	133	165	205	256	330	461	*				
25	595	20	33	47	62	78	97	117	140	166	198	236	285	354	469	595	
30	371	17	27	38	50	62	76	91	107	125	145	167	193	223	260	307	371
35	232	14	23	32	42	52	63	74	87	100	115	131	148	168	190	215	232
40	163	12	20	27	36	44	53	63	73	84	95	108	121	135	151	163	
45	125	11	17	24	31	39	46	55	63	72	82	92	102	114	125		
50	92	9	15	21	28	34	41	48	56	63	71	80	89	92			
55	74	8	14	19	25	31	37	43	50	56	63	71	74				
60	60	7	12	17	22	28	33	39	45	51	57	60					
70	48	6	10	14	19	23	28	32	37	42	47	48					
80	39	5	9	12	16	20	24	28	32	36	39						
90	30	4	7	11	14	17	21	24	28	30							
100	25	4	6	9	12	15	18	21	25								
110	20	3	6	8	11	14	16	19	20								
120	15	3	5	7	10	12	15										
130	10	2	4	6	9	10											
140	10	2	4	6	8	10											
150	5	2	3	5													
160	5		3	5													
170	5			4	5												
180	5			4	5												
190	5			3	5												

* Highest repetitive group that can be achieved at this depth regardless of bottom time.

Table 9-8. Residual Nitrogen Time Table for Repetitive Air Dives.



** Residual Nitrogen Time cannot be determined using this table (see paragraph 9-9.1 subparagraph 8 for instructions).
† Read vertically downward to the 30 fsw repetitive dive depth. Use the corresponding residual nitrogen times to compute the equivalent single dive time. Decompress using the 30 fsw air decompression table.