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MEDICAL CENTER**

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**EVALUATION OF THE RAPID RECOMPRESSION-HIGH PRESSURE
OXYGENATION APPROACH TO THE TREATMENT OF
TRAUMATIC CEREBRAL EMBOLISM**

by

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Bureau of Medicine and Surgery, Navy Department
Research Work Unit MR005.04-0057.01

Released by:

Gerald J. Duffner, CAPT MC USN
COMMANDING OFFICER

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SUBMARINE MEDICAL RESEARCH LABORATORY
U. S. NAVAL SUBMARINE MEDICAL CENTER REPORT NO. 519
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SUMMARY PAGE

THE PROBLEM

To compare the conventional Standard Navy Recompression Treatment Tables with the newer rapid recompression-high pressure oxygenation (RR-HPO) method for the treatment of traumatic cerebral air embolism.

FINDINGS

Seven cases occurring during the course of submarine escape training at the Escape Training Tank, Naval Submarine Base, Groton, Connecticut, and successfully treated with the newer method are presented. Discussion is presented of the treatment of traumatic cerebral air embolism by both Standard Tables and the newer approach. Factors regarding the rationale of the use of the new approach are given.

APPLICATIONS

It is expected that the Navy Department will approve the treatment schedules recommended in this report for the treatment of traumatic air embolism.

ADMINISTRATIVE INFORMATION

The authors conducted this investigation while both were attached to the Submarine Medical Center, during 1967, as a part of Bureau of Medicine and Surgery Research Work Unit MR005.04-0057. It is Report No. 1 on that Work Unit; however, a related report was published as SMRL Report No. 500, August 1967, under MR005.04-0055.10. The present report was approved for publication on 25 March 1968, and designated as SMRL Report No. 519.

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ABSTRACT

Comparison of the Navy conventional recompression treatment tables and the newer rapid recompression-high pressure oxygenation (RR-HPO) method for the treatment of traumatic cerebral air embolism is made. The embolism cases treated were incurred during submarine escape training at the Naval Submarine Base, New London, Connecticut. In view of its theoretical and proven practical superiority, the RR-HPO approach described here and in Navy Recompression Tables V-a and VI-a is recommended for the treatment of traumatic air embolism.

EVALUATION OF THE RAPID RECOMPRESSION-HIGH PRESSURE OXYGENATION APPROACH TO THE TREATMENT OF TRAUMATIC CEREBRAL EMBOLISM

INTRODUCTION

Cerebral air embolism may result from any condition in which a gas or mixture of gases enters the systemic circulation, ultimately becoming lodged in small cerebral vessels and occluding the blood supply distally. The treatment for this true medical emergency is prompt recompression. This paper will discuss cases occurring as a result of training in escape from submarines, which were treated by both conventional U. S. Navy recompression treatment tables and the recently developed rapid recompression-high pressure oxygenation treatment tables (RR-HPO), utilizing a considerably shorter treatment time and intermittent oxygen breathing beginning at 2.8 atmospheres absolute. In this series the embolism resulted from overexpansion and rupture of alveoli with the release of air into the pulmonary venous circulation, left heart, aorta and ultimately the cerebral circulation.

DESCRIPTION OF ESCAPE TRAINING

At the Naval Submarine Medical Center, Naval Submarine Base, New London, Connecticut, the air embolism cases seen are coincident to training in submarine escape techniques taking place in the Escape Training Tank. At the present there are two methods of submarine escape being taught, the buoyant ascent method and the free-breathing, buoyant ascent method.

In buoyant ascent training the candidates are placed in a pressure lock located fifty feet below the surface of the water and opening into the side of the tank by means of a door. The lock is then partially filled with water to the level of the top of the door leading into the main tank and then pressurized to the ambient pressure, exerted by the water column at that level. Following pressurization the door may then be opened, allowing free access between the tank and the lock. The trainees take a deep breath of the pressurized air, step through the door into the tank, commence a steady controlled exhalation, and assisted by an inflated life jacket, make a rapid

ascent. They travel the fifty feet at 375 feet per minute in seven to eight seconds, continuously and positively exhaling during the ascent to the surface. Since its initiation in 1956, there have been over 128,000 successful buoyant ascent runs.

In the free breathing, buoyant ascent method the trainee also makes an escape from the fifty foot level, entering the tank from the lock and ascending to the surface. The difference is the utilization of a more sophisticated escape appliance in this procedure. The appliance, called the "Steinke Hood" after its inventor, CDR H. E. Steinke, USN, is constructed with a head hood attached to the collar of an inflatable life jacket. During ascent the expanding air from the jacket is vented through two relief valves into the hood expanded and allowing the escapee to inhale if necessary. To ensure the steady and timely decrease of the expanding volume of air in the lungs, the trainees are instructed to shout "ho-ho-ho" in quick succession between each shallow inhalation while ascending. With the additional buoyance of the air filled hood, the trainee travels somewhat more rapidly, approximately 425 feet per minute. Approximately 45,000 successful hood runs have been made since the institution of this mode of training in 1963.

Embolization occurs in those instances in which the intra-pulmonary pressure is not reduced with the concomitant reduction of the intra-pleural and intra-arterial pressures. The latter pressure decreases with the reduction of hydrostatic pressure on ascent. Schaefer found that embolization into the pulmonary veins occurs in dogs when the transpulmonic pressure, (the difference between the intra-pulmonary and the intra-pleural pressure) exceeds 50-70mm. Hg.¹ Such a pressure differential can be developed merely by breath-holding after inhaling air pressurized to three feet of water pressure and ascending to the surface.

DESCRIPTION OF TREATMENT

In the period covered by this report from

1956 through mid-1964, treatment has been according to Tables III and IV of the standard Navy Recompression Tables,² promulgated in 1945. These tables involve recompression to six atmospheres pressure absolute,

165 feet depth equivalent, and slow stage decompression in ten foot increments over a 19 to 38 hour period, respectively. Treatment Tables III and IV are shown in Figure 1.

Figure 1. Standard Navy Recompression Tables for the Treatment of Air Embolism
(from Table i-21, U. S. Navy Diving Manual).

If symptoms relieved within 30 min. after recompression to 165'		If symptoms NOT relieved within 30 min. after recompression to 165'	
use Table III		use Table IV	
Rate of Ascent between stops—one minute.			
Feet	Table III	Table IV	
165	30 min (air)	30 to 120 min (air)	
140	12 min (air)	30 min (air)	
120	12 min (air)	30 min (air)	
100	12 min (air)	30 min (air)	
80	12 min (air)	30 min (air)	
60	30 min (O ₂ or air)	6 hr (air)	
50	30 min (O ₂ or air)	6 hr (air)	
40	30 min (O ₂ or air)	6 hr (air)	
30	12 hr (air)	11 hr (air), then 1 hr (O ₂ or air)	
20	2 hr (air)	1 hr (air), then 1 hr (O ₂ or air)	
10	2 hr (air)	1 hr (air), then 1 hr (O ₂ or air)	

In an effort to reduce the treatment time in the pressure chamber, the risk of decompression sickness occurring as a result of recompression treatment, and the rate of symptom recurrence and residual after treatment, a departure from the standard treatment of air embolism was undertaken in the cases occurring since 1964. The basis for this departure was derived from the work of Goodman and Workman at the Navy Experimental Diving Unit, Washington, D. C.³

This regimen consists of a rapid recompression and a high-pressure oxygenation phase. In the first phase, initial repressurization to 165 feet depth equivalent is made rapidly in order to relieve the major symptoms occurring from the acute embolization. This aspect of the treatment is unchanged from previous methods. Upon improvement in the patient's condition, the second phase is begun in which an ascent to 60 foot depth equivalent is made over a four minute period.

At this point oxygen breathing, alternating with shorter periods of air breathing, is begun. After completion of the O₂ — air sequence at the 60 feet stop, continuous decompression to the 30 feet stop is accomplished at a one foot per minute rate while the patient is breathing oxygen. At 30 feet alternate oxygen and air breathing is again carried out, followed by continuous decompression to the surface at the same rate as before again breathing oxygen.

The relative amount of time allotted to oxygen breathing and air breathing respectively in each of the sequences has varied during the evaluation of this approach to treatment. The change in schedules for this second phase have essentially followed the evolution of the oxygen breathing tables developed by Goodman and Workman for the treatment of decompression sickness and are shown in Figure 2.

Figure 2. Comparison of Oxygen Sequences Evaluated for the Second Phase of the RR-HPO Treatment of Air Embolism.

Symptoms relieved within fifteen minutes after recompression to 165 feet		
Depth (Feet)	Earlier Table V (1964-1965)	Later Table V (1966-1967)
60	40 min. O ₂	one or two periods — 20 min. O ₂ , 5 min. air
60-30	30 min. O ₂	unchanged
30	30 min. O ₂	one or two periods — 20 min. O ₂ , 5 min. air
30-0	30 min. O ₂	unchanged

Symptoms NOT relieved within fifteen minutes after recompression to 165 feet		
Depth (Feet)	Earlier Table VI (1964-1965)	Later Table VI (1966-1967)
60	30 min. O ₂ , 15 min. air, 30 min O ₂	three or four periods — 20 min. O ₂ , 5 min. air
60-30	30 min. O ₂	unchanged
30	two periods — 15 min. air, 60 min. O ₂	unchanged
30-0	30 min. O ₂	unchanged

Initially, thirty to forty minutes of uninterrupted oxygen breathing was performed at the 60 foot stop. Later, in order to reduce the risk of oxygen toxicity, the time of uninterrupted oxygen breathing at 60 feet was limited to twenty minutes. In cases in which more than one twenty minute exposure was utilized, the oxygen breathing periods were separated by five minutes of air breathing as opposed to the earlier schedule in which a fifteen minute air breathing stop separated a sequence of thirty minute oxygen exposures.

Two treatment sequence aspects have remained unchanged. One is the use of sixty minute oxygen exposures separated by fifteen minute air exposures at 30 feet in those cases in which major symptom response at 165 feet took longer than ten minutes. The second is that the ascents from 60 feet to 30 feet and again from 30 feet to the surface has occurred over a thirty minute period

during which time oxygen breathing is utilized.

The selection of the treatment table is determined by the amount of time required for major symptom relief at 165 feet. The shorter table, corresponding to that used for severe decompression sickness responding promptly to high pressure oxygenation treatment, is known as Table V. If more than fifteen minutes was required for major relief, then a longer oxygenation schedule is required. This sequence is now known as Table VI, and corresponds to the table used for the treatment of serious decompression sickness not responding after ten minutes of oxygenation at 60 feet. Tables Va and VIa now refer to the total treatment schedules for air embolism consisting of both the rapid recompression and the respective oxygen breathing phases. They are shown in Figure III along with instructions for their use.

Figure 3. RR-HPO Treatment of Air Embolism.

TABLE V-a — PATIENT SYMPTOM FREE WITHIN 15 MINUTES

Depth (Feet)	Time (Min.)	Breathing Media	Total Time (Min.)
165	15 or less	Air	15 (*)
165-60	4	Air	19
60	20	O ₂	39
60	5	Air	44
60	20	O ₂	64
60-30	30	O ₂	94
30	5	Air	99
30	20	O ₂	119
30	5	Air	124
30-0	30	O ₂	154

TABLE VI-a — PATIENT SYMPTOM FREE WITHIN 30 MINUTES
AND/OR RAPIDLY IMPROVING

Depth (Feet)	Time (Min.)	Breathing Media	Total Time (Min.)
165	30 or less	Air	30 (*)
165-60	4	Air	34
60	20	O ₂	54
60	5	Air	59
60	20	O ₂	79
60	5	Air	84
60	20	O ₂	104
60	5	Air	109
60-30	30	O ₂	139
30	15	Air	154
30	60	O ₂	214
30	15	Air	229
30	60	O ₂	289
30-0	30	O ₂	319

(*) Total time will vary as function of this stop.

INSTRUCTIONS FOR USING TABLES

1. DESCENT TIME — Recompression to 165 feet should be accomplished as rapidly as possible (usually less than one minute).
2. TIME AT DEPTH — Total time at 165 feet will vary with the clinical status of the patient. The medical attendant should take the time to make a thorough physical appraisal of the patient, since the ensuing treatment is based on the patient's physical status.
3. USE OF TABLE V-a — If all major symptoms and signs are gone before 15 minutes total bottom time, proceed to 60 feet at 25 feet per minute and begin oxygen breathing according to Table Va.
4. USE OF TABLE VI-a — If serious or major symptoms or signs persist beyond 15 minutes, but show signs of moderating within 30 minutes total bottom time,

proceed to 60 feet at 25 feet per minute and begin oxygen breathing according to Table VI-a.

5. SERIOUS SYMPTOMS — Such include unconsciousness, convulsions, major paralysis or weakness, cranial nerves signs, and cerebellar signs.
6. PERSISTENT SYMPTOMS — Should serious symptoms and signs persist beyond 30 minutes **without moderation**, begin Table IV treatment.
7. ATTENDANTS — Inside tenders routinely breathe air; however, if treatment constitutes a repetitive dive for a tender, he must breathe O₂ from 30 feet to the surface.
8. OXYGEN TOXICITY — Should symptoms or signs of oxygen intolerance develop at the 60 foot stop, the oxygen should be discontinued temporarily and begun again on leaving the 60 foot stop.

9. FOLLOW-UP — On completion of the treatment table the patient should be routinely held for observation and given a thorough medical examination, including appropriate radiographic and laboratory studies. Patient should be released only if completely asymptomatic.

RESULTS OF TREATMENT

There have been a total of twenty-four over-pressurization accidents occurring at the New London tank from 1956 through June 1967, as a result of buoyant ascent and Steinke hood training. In addition, one case of air embolism developing as a result of free ascent was treated and is included. (In this latter technique the individual on ascent releases only enough air from his lungs, initially fully inflated with air pressurized to depth, to prevent embolization and yet maintain positive buoyance. The training in this

particular method is considerably more dangerous and is not generally taught at the present time.)

Four of the cases exhibited the production of extra-alveolar air, such as pneumothorax or subcutaneous emphysema, but not cerebral air embolism. Recompression was not a part of their treatment, and these cases are not included in the treatment figures.

One of the twenty-four cases resulted in death. While this is certainly a treatment failure, it cannot be considered a failure of any particular regimen because of the short time, approximately four to five minutes, that the patient was under recompression prior to death.

Nineteen of the cases developed air embolism and were treated by recompression in one form or another. A summary of the methods of treatment is given in Figure 4.

Figure 4. Treatment of Surviving Cases of Air Embolism Occurring at the Escape Training Tank, New London, Connecticut, 1956-1967.

	Table III		Table IV		RR-HPO	Method	Other	Methods
	Used	Failed	Used	Failed	Used	Failed	Used	Failed
Bouyant Ascent	7	1	2	0	6	0	0	0
Steinke Hood	2	0	0	0	1	0	1 170 ft	0
							20 min.	
Free Ascent	1	0	0	0	0	0	0	0
Total	10	1	2	0	7	0	1	0

Eleven of the nineteen surviving cases in this report were treated a total of twelve times according to the standard recompression tables. There was one treatment failure on Table III, occurring while under pressure at 30 feet depth pressure equivalent. The case was ultimately treated with Table IV, with a resulting failure rate of 8.3%, in this series for the Standard Tables.

The Navy-wide experience with Tables III and IV for the treatment of air embolism is even poorer.⁴ From 1946 through 1962, forty-six surviving cases of air embolism of all causes were treated by several Navy facilities, primarily the escape training tanks at the Naval Submarine Bases, Groton, Conn., and Pearl Harbor, Hawaii. This group includes the escape training accident cases discussed in this paper which occurred at the New London Tank during that time period. In the seventeen year interval, the rate of

symptom recurrence and/or residual incidence following treatment was 19.5%, seven failures having come in the use of Table III in thirty-four cases and two failures occurring in the twelve cases treated according to Table IV.

This larger group of air embolism cases resulted not only from submarine escape training at the two training tanks but also from accidents occurring while diving in open water. In the latter situation the time between the occurrence of the accident and the application of treatment recompression is often greater than that following escape training accidents because of the distance of the patient from the recompression chamber at the time of the accident. This may be one of the factors accounting for the higher residual and recurrence rate.

During the 17 year period under review, five persons died as a result of air embolism.

Though they may be classed as treatment failures, none is included in the group, since all died before or shortly after recompression had been instituted. It may be noted that four of the deaths occurred following free ascents. The fifth fatality is the one mentioned earlier in the paper.

A summary of the cases occurring since 1964, and treated by the newer, shorter approach is given in Figure 5. All seven of the cases in which RR-HPO method was used have been successfully treated without symptom recurrence or permanent residual. None of the patients developed decompression sickness as a treatment sequelae. The average treatment time was approximately three hours. In all but one of the cases, relief of major symptoms was obtained within fifteen minutes after the initiation of rapid

recompression, and so their treatment schedule more nearly approximated Table V-a. In the remaining case, Table VI-a was followed because of incomplete resolution of paresthesia and paresis.

There was one case which was treated with rapid recompression and then decompression according to a standard Navy 170 feet for 20 minute table with no Hyererbark Oxygen Breathing. The patient had already responded completely before reaching 100 feet on recompression. After spending two minutes at 165 feet he was successfully decompressed in twenty-five minutes, utilizing a recompression ascent rate of 25 feet per minute. Six minutes were required for ascent to the first stop at 20 feet where four minutes were spent, followed by fifteen minutes at 10 feet.

Figure 5. Summary of Eight Cases of Air Embolism Associated with Escape Training at Groton, Connecticut, Oct 1964—Mar 1967.

CASE	DATE	DIVE	TREATMENT	OUTCOME & ADDTL DIAGNOSIS
1. M.H.M.	10-15-64	B.A.	165' + early Table 5	Return to duty in 48 hrs.
2. C.C.N.	3-12-65	S.H.	165' + early Table 5 — modified — *	Return to duty in 24 hrs.
3. B.S.T.	1-14-66	S.H.	165' + Table 170/20 — no O ₂ — *	Return to duty in 24 hrs.
4. J.H.D.	5-26-66	B.A.	165' + later Table 5	Return to duty in 24 hrs.
5. J.H.	6-15-66	B.A.	165' + later Table 5*	Return to duty in 24 hrs.
6. F.J.B.	9-23-66	B.A.	165' + later Table 5	Mediastinal Emphysema* Return to duty in 5½ days
7. D.D.M.	10-24-66	B.A.	165' + later Table 5	Residual Chest Pain* Return to duty in 2½ days
8. B.R.M.	3-8-67	B.A.	165' + later Table 6*	Return to duty in 3 days

***SPECIAL NOTES:**

Case 2—60 feet stop was bypassed. O₂ breathing at 30 feet stop and 30-0 feet. Attendant developed mild decompression sickness.

Case 3—Standard Navy Decompression Table used.

Case 5—Developed signs of early O₂ intolerance after 15 min. at 60 feet—changed to air breathing—returned to O₂ on leaving the 60 feet stop.

Case 6—Subsequent chest X-ray showed pneumopericardium.

Case 7—Subsequent EKG showed inverted T waves which reverted to normal in 2 days.

Case 8—Because of a question of persistence of paresis and paresthesias at 165 feet, Table 6A was used.

B.A.—Buoyant Ascent

S.H.—Steinke Hood

DISCUSSION

The rationale for the use of the high pressure oxygen breathing phase after initial symptom relief is two-fold. One is the desire to reduce the length of time at such depths as is required in treatment according to Table III or IV. With each increase in time and depth under pressure there is a greater uptake of inert gas by the tissues. This not only increases the possibility of the subsequent development of treatment related decompression sickness, either following or during treatment, but also diminishes the rate of diffusion from the tissues of gas that has been absorbed in the same pressure exposure that led to the embolization accident. With the use of oxygen, the uptake of inert gas by surrounding tissues is completely eliminated, thereby creating a maximal gas exchange gradient from the tissue to the surrounding fluids. The second reason is the increased tissue oxygenation aiding in "functional restoration of tissues rendered hypoxic by the ischemic action of bubble emboli."³

The minimal depth, oxygen breathing tables were tested both clinically and hypothetically and found to provide adequate decompression for pressure exposures which initially had received grossly inadequate decompression. If the tables are not properly followed, however, decompression sickness may result. Such did occur with a chamber attendant caring for patient 2 (C.C.N.). In bypassing the 60 foot stop, inadequate decompression resulted and the attendant who did not breathe oxygen during the treatment sequence, developed mild decompression sickness, manifested by skin rash and joint pains of the upper extremities.

The importance of rapid recompression is obvious in all cases treated, as generally symptoms are relieved within minutes after recompression. Its contribution to the total treatment regimen can be seen especially in two cases. In case 3 (B.S.T.), treatment consisted only of rapid repressurization, there being no oxygenation phase at all. The oxygenation phase in case 2 (C.C.N.) was markedly reduced. The patient was decompressed from 165 to 30 feet before beginning an oxygen stop. This second treatment, however, did create the setting for decompression inadequacy mentioned above.

The rapidity with which cerebral vascular function is restored by repressurization following the occurrence of experimentally produced cerebral air embolism has been graphically demonstrated by Waite, et al.⁵ Dogs were prepared by making a window in the calvarium and exposing cerebral vessels. Air was injected into an isolated carotid artery and the subsequent appearance of bubbles and vascular occlusion was observed in the exposed cerebral circulation. The group of animals which were treated were then pressurized to 165 feet pressure equivalent. Maximal restoration of circulation was seen at between 33 feet, two atmospheres pressure absolute, with evidence of a change in bubble size and partial restoration just beyond 33 feet and removal of all bubbles by 100 feet. These depths were achieved in a matter of seconds to a few minutes after the initiation of pressure. Decompression was accomplished with Navy tables using a 170 feet for ten minute schedule, using a 25 feet per minute rate of ascent and a two minute decompression stop at ten feet.

Five of the six embolized animals treated in this manner responded completely, indicating that it is not the prolonged decompression in a schedule such as Table III or IV that is the beneficial factor in treating air embolism. Rather it is the promptness of the application of pressure that determines the speed of restoration of cerebral circulation. The one case not responding properly to treatment was the one in which rupture of a blood vessel with subsequent cerebral hemorrhage and edema occurred after embolization. This sequence of events may well give insight into the mechanism of the situation in which embolization is not promptly relieved by repressurization.

The fact that the RR-HPO treatment regimen is not only effective but also shorter in length than the standard Navy treatment schedule makes it even more beneficial. The patient and tenders are not subjected to the confining and uncomfortable environment of a recompression chamber for prolonged periods of time. Also the reduction of the length of the chamber stay permits the utilization of the full supportive facilities of the hospital sooner.

SUMMARY

Discussion of the treatment of traumatic cerebral air embolism by both standard Navy Recompression Tables and the new rapid recompression, high pressure oxygen breathing approach is presented. Seven cases occurring as a result of submarine escape training at the Escape Training Tank, Naval Submarine Base New London, Groton, Connecticut, and successfully treated with the latter method are presented. Factors regarding the rationale of the use of this new approach are given. POSTSCRIPT: Since the writing of this paper, the Navy Department has approved the treatment schedules recommended herein for the treatment of traumatic air embolism.

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This study compared the conventional Navy recompression treatment tables and the newer rapid recompression-high pressure oxygenation (RR-HPO) method for the treatment of traumatic cerebral air embolism. Seven cases are presented. These cases occurred as a result of submarine escape training at the Naval Submarine Base, Groton, Connecticut, and were successfully treated with the latter method. Factors regarding the rationale of the use of this new approach are discussed. Since the writing of this paper, the Navy Department has approved the treatment schedules recommended herein for the treatment of traumatic air embolism.			

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