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**STUDY: METHODS FOR TREATMENT
OF DECOMPRESSION SICKNESS**

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Introduction

The historical collection of *treatment tables TT* up to the 1980s can be found in the report by *US Naval Medical Research Center* (1). One of the methods available in the reviewed literature is the classification agreed upon in *NATO*. The complete list of the *TT* used by individual countries is presented in *Chapter 5 National therapeutic recompression tables* (2). This document is the Annex to the *NATO Standardization Agreement* (3).

The existing *TT* can be divided into saturation, sub-saturation, and outside the saturation zone *TT* – **Tab.1**. And the last mentioned *TT* can be divided into those using oxygen therapy or other therapy¹.

The *US Navy* procedures (4) constitute the most widespread treatment system². An interesting extension of air treatment procedures is the set of the air-helium³ tables developed by the Russians (5). However, the system which can support the whole range of depths, available for divers at present, is the system developed by *COMEX* (6).

Treatment procedures

There exist different classifications of decompression sickness *DCS*⁴. The classification proposed by *Wienke* has been adopted here – **Tab.2**. The most common cases encompass: *Type I DCS* and *Type II DCS*. They are caused by formation of gas in free phase in tissues. They can be effectively treated by hyperbaric methods causing decrease in the size of bubbles in free gas phase occurred into tissues and caused the possibility of their reabsorption and elimination by the gas exchange through the blood into the respiratory system.

In the traditional operational diving⁵ *Type I DCS* is most often caused by formation of gas in free phase in tissues, especially in blood, adipose and

¹ air, air-helium, helium-oxygen *Hx* tables

² understood as a method for moving between the treatment procedures in the course of treatment decompression or when next sickness symptoms occurred after the completion of treatment

³ oxygen-nitrogen-helium mixture is called *Trimix* and marked *Tx*

⁴ **D**ecompression **S**ickness

⁵ outside the saturation zone

Example of treatment tables *TT* apportionment (1)

Type	Example of national tables	Equivalent <i>US Navy</i> tables
Short oxygen tables	TABLE V Goodman 1 (BE) OHB (FR) HBO (GE) 9SH (NL) 60 & 61 (UK)	TT USN 5
Long, shallow oxygen tables	TABLE VI 62 (UK) Goodman 2 (BE)	TT USN 6
Long, deep oxygen tables	TABLE VII 6A (mod) (GE, CA) TABLES A and B (FR) SND (GE) 63 (UK)	TT USN 6A
Saturation or sub-saturation tables	TABLE VIII US 7 US 8 TABLE C (FR) 64 & 65 (UK)	TT USN 4
Air tables	TABLE I TABLE II TABLE D (FR) 52 (UK)	1A 2A

connective tissue and *Type II DCS* in nervous tissue. Blocked flow of blood by gas free phase can cause local ischemia leading to necrosis. Occurrence of gas in free phase in tissue usually causes neurological symptoms.

There exist a few theories relating to the probable pain inducing mechanism with regard to *Type I DCS* (7). It occurs when bubbles in gas in free phase reach the size big enough to:

- dislocate or irritate nerve endings

Types of decompression sickness *DCS* (8)

<i>Typ I</i>	Limbs. Symptoms : localized pain in joints, skin itching, bends, rashes, mottling, lymphatic swelling, ascending weakness in the legs, hips, elbows, muscles or skin
<i>Typ II</i>	Central nervous system. Symptoms: confusion, anxiety, paralysis, dyspnoea, chest pain, breathing difficulty, loss of conscientiousness, lack of alertness, impaired balance problems in keeping upright posture, especially keeping the spine straight
<i>Typ III</i>	Inner ear. Symptoms: impaired hearing, dizziness, ringing in the ears, and tinnitus or nausea. It is the result of the impact of pressure on keeping balance in the organs in the ear
<i>Typ IV</i>	Avascular necrosis. Symptoms: mechanical bone injury, structural injury, local mineralization. It affects mostly long bones

- block capillaries leading to ischemia and tissue necrosis, which while excreting chemical substances warn the brain about injuries inducing feeling of pain generated by brain
- activate the biochemical reaction of immunoglobulins production, which also stimulates pain signalling the presence of harmful substances in living cells.

The destructive actions in tissues caused by gas existing in free phase are not the only pathological symptoms induced by it. Defensive reactions of an organism induced by gas in free phase do not have to be directly linked to the mechanical impact, but they also have biochemical origins of pain. This theory, called the complement activation⁶, seems to be well documented (9).

Typ II DCS can be a result of blocked blood flow to the spinal cord, which causes slowing down stimulation or inhibiting higher brain functions⁷.

Type III DCS can be caused by gas in free phase, inert gas counterdiffusion and increased pressure of bodily fluids in the inner ear. Theoretically, anomalies occurring on the surface of cell membranes can be responsible for *Type III DCS*, and gas counterdiffusion caused by occurrence of content

⁶complement is a system of plasma proteins contributing to defensive reaction of that occurs on the surface of pathogens and generates active components with various effector functions.

⁷central nervous system does not tolerate becoming deprived of the flow of information, that is why its activity dies out quickly, causing *Type II DCS*

gradients of various gases is responsible for these anomalies. One gas can defund in one direction, whereas the other in the opposite direction. Their counter-current streams cause gas exchange disturbances on the borders of phases, which causes a change in physical conditions of gas solubility in tissue⁸ (10). Their mutual competing for presence in the solution can lead to displacing one of them⁹ (11). *Type III DCS* symptoms can be a result of an osmotic reaction. Bodily fluids tend to balance the concentration of gases contained in it through dislocation, causing dilution of the more concentrated solution. This can entail an increase in the amount of fluids in the inner ear organs which leads to dysfunction of the labyrinth as a consequence (12).

It is difficult to determine to what extent *Type IV DCS* is caused by wrongly performed decompression. Its formation can be influenced by the free gas phase, ischemia, embolism¹⁰ and a combination of these factors. Statistically this sickness is important with regard to professional divers and people working in caissons. Both of these groups are often exposed to high pressure actions. The same symptoms as in *Type IV DCS* are found in racing horses and alcoholics. In this case, this sickness is called aseptic bone necrosis. The mechanisms of formation of *Type IV DCS* are considered theoretically and there is no exhaustive evidence based on medical observation. These symptoms are osteonecrosis that is not induced by infection¹¹. If pathological hardening of bone occurs in the central part it does not affect vitals. If pathological changes occur in the end of a bone, where the head of one of the bones fits into the socket of another one, splinters can form and joints can be damaged. Probably, repeated fast compression and the content of dioxide can have an influence on inducing avascular osteonecrosis. Long exposition to partial oxygen pressure above 60 kPa can cause osteonecrosis in people predisposed to developing this sickness. The probable mechanism is associated with blocking blood flow into marrow and living bone tissue by bubbles in gas in free phase, locating themselves in capillary vessels (8). A connection has been found of pain located in bones region with the yellow marrow, which is far less supplied by blood¹² than the red marrow. Connecting this with the generally known good solubility of inert

⁸natural solubility is understood here as the condition in which gases occur independently

⁹mechanism similar to the salting-out effect in water solutions; the addition of sodium chloride to a another solution to reduce the solubility of the latter

¹⁰gas embolism

¹¹hence the term aseptic

¹²*perfunded*; perfunde is the verb from perfusion, which means the passage of fluid through the circulatory system or lymphatic system to an organ or a tissue

gases in fats it can be anticipated that the difficulty in transporting the gas from the inside of a bone can be the cause of the sickness symptoms.

Type III DCS and *type IV DCS* should not be treated with hyperbaric methods using the recompression treatment. In many cases, the recompression treatment can lead to a worsened condition of the patient (13). It is important as *Type II DCS* and *Type III DCS* are difficult to distinguish from each other.

During saturation dives *Type I DCS* can occur after an excursion to the depth less than the plateau of saturation or during decompression on completion of saturation. The symptoms are pain in skeletal muscle areas and joint or joints, especially knee joints. It can be preceded by itching, a rash or skin spots. Most often, at the beginning increased stiffness occurs in a knee joint area hindering movement. Then, within a few hours, the pain steadily grows in joint areas. However, divers should be able to distinguish the pain, which occurs as a result of small injuries or after some physical effort, caused by joint overload or mechanical injuries which occur during work from which the cause may be related to decompression. To this end, it is necessary to thoroughly consider the history of pain formation and its increase. It is little likely that the pain which occurred before the beginning of the decompression was caused by *DCS*. *Type I DCS* formed during an excursion from the plateau of saturation and up to 60 *min* after the excursion over the depth of the plateau of saturation should be regarded as *type II DCS*, because *type I DCS* symptoms are most often a signal of more serious complications.

Treatment of symptoms of diving sicknesses during saturation differs from that administered after completed decompression following saturation diving and operational diving outside the saturation zone (14). Usually the treatment methods in saturation can also be applied as the final *DCS* treatment methods following diving beyond the saturation zone, as in the system developed by *Comex* (6). The effectiveness of saturation treatment methods should be the highest than other ones, but they are long-term methods and that is why treatment using treatment tables can be performed more effectively. Hence the *US Navy* system is most common (4).

An almost complete historical review of the most important trends in the progress recorded with regard to the decompression sickness treatment can be found in many places, but it is easy to find it in the Internet, on

the website address¹³:

http://www.wikiwand.com/en/Hyperbaric_treatment_schedules

It presents a set of tables implemented in the *US Navy* and used by many *NATO* countries together with other methods.

US Navy treatment tables

The set of *USN* treatment tables, implemented the *US Navy*, encompasses procedures using only air¹⁴ or air and *Nx*¹⁵, and procedures using both air/*Nx* and oxygen alternately¹⁶ as treatment breathing agents (4). Procedures utilizing oxygen are more effective and should be administered first. Procedures utilizing air only should be administered when oxygen is not available¹⁷, in emergency conditions, where the patient does not tolerate oxygen under high pressure¹⁸ and he shows symptoms of oxygen toxicity during the treatment or the occurrence of oxygen toxicity is highly probable¹⁹.

To treat *Type I DCS* the *TT 5 USN* is used – **Fig.1**. If after $\tau = 10 \text{ min}$ of stop at the depth $H = 18 \text{ mH}_2\text{O}$ the sickness symptoms do not disappear, the treatment has to be continued using the *TT 6 USN* – **Fig.2**. If after $\tau = 10 \text{ min}$ of stop at the depth $H = 18 \text{ mH}_2\text{O}$ the sickness symptoms disappear, the treatment can be discontinued in accordance with the *TT 5 USN* or it can be prolonged by maximum two cycles $\tau_{O_2} = 20 \text{ min}$ cycle of breathing oxygen and $\tau_{air} = 5 \text{ min}$ cycle of breathing air at the depth $H = 9 \text{ mH}_2\text{O}$. Treatment using the *TT 6 USN* can be prolonged by two $\tau_{O_2} = 20 \text{ min}$ cycles of breathing oxygen and $\tau_{air} = 5 \text{ min}$ breathing air at the depth $H = 18 \text{ mH}_2\text{O}$ and two $\tau_{O_2} = 20 \text{ min}$ cycles of breathing oxygen and $\tau_{air} = 5 \text{ min}$ breathing with at the depth of $H = 9 \text{ mH}_2\text{O}$. More serious cases, such as *Type II DCS*, are treated following the algorithm presented in **Fig.3**. To treat gas embolism or *Type II DCS* treatment tables: *TT 4 USN*,

¹³available June 2017

¹⁴*TT 1A USN, TT 2A USN, and 3 TT USN*

¹⁵*TT 7 USN*; nitrogen-oxygen mixture is called *Nitrox* and is marked *Nx*

¹⁶*TT 4 USN, TT 5 USN, TT 6 USN, TT 6A USN, TT 9 USN*

¹⁷e.g. as a result of built-in-breathing system malfunction, exhaustion of stocks, etc.

¹⁸develops oxygen toxicity (4)

¹⁹considered when the diver was, just before, exposed to high partial pressure of oxygen and in this case occurrence of symptoms indicating central nervous system oxygen toxicity or pulmonary oxygen toxicity is highly probable, e.g. when the dose of oxygen toxicity was clearly exceeded during the dive (7; 15; 24)

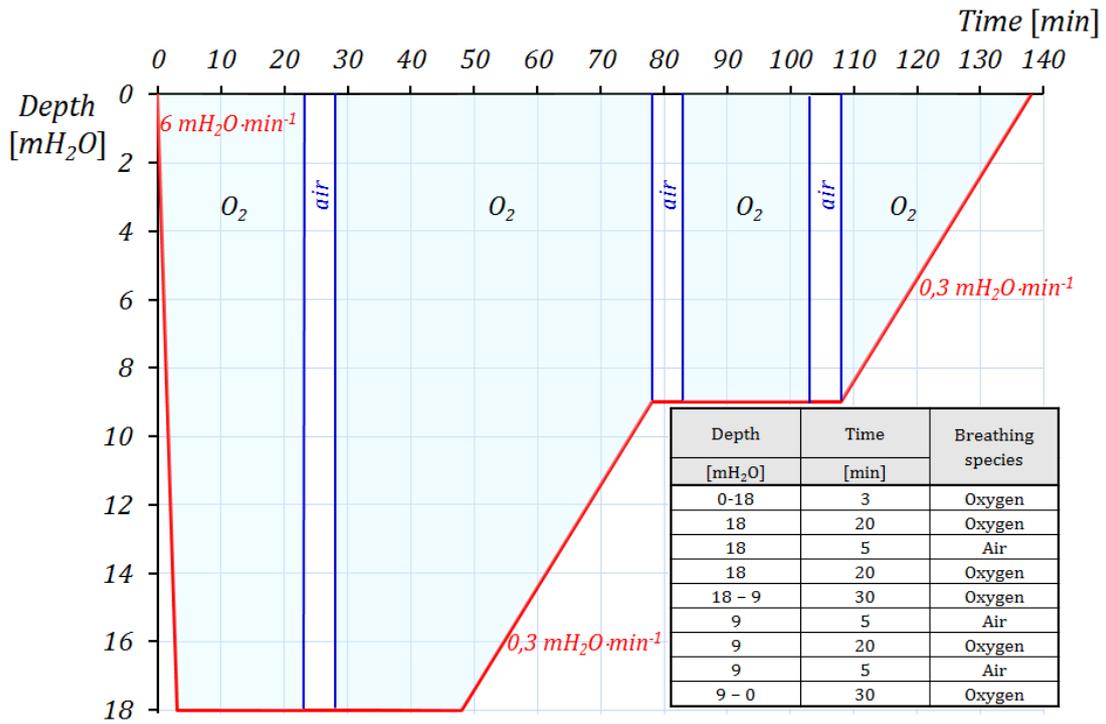


Fig.1. Treatment table TT 5 USN

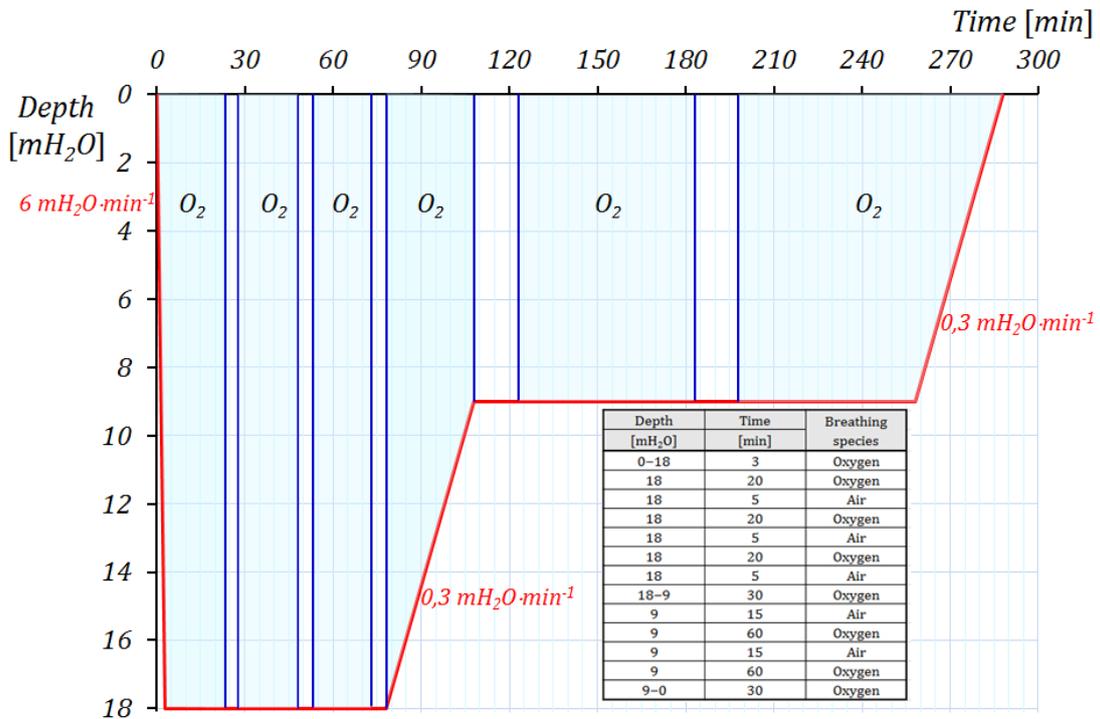


Fig.2. Treatment table TT 6 USN

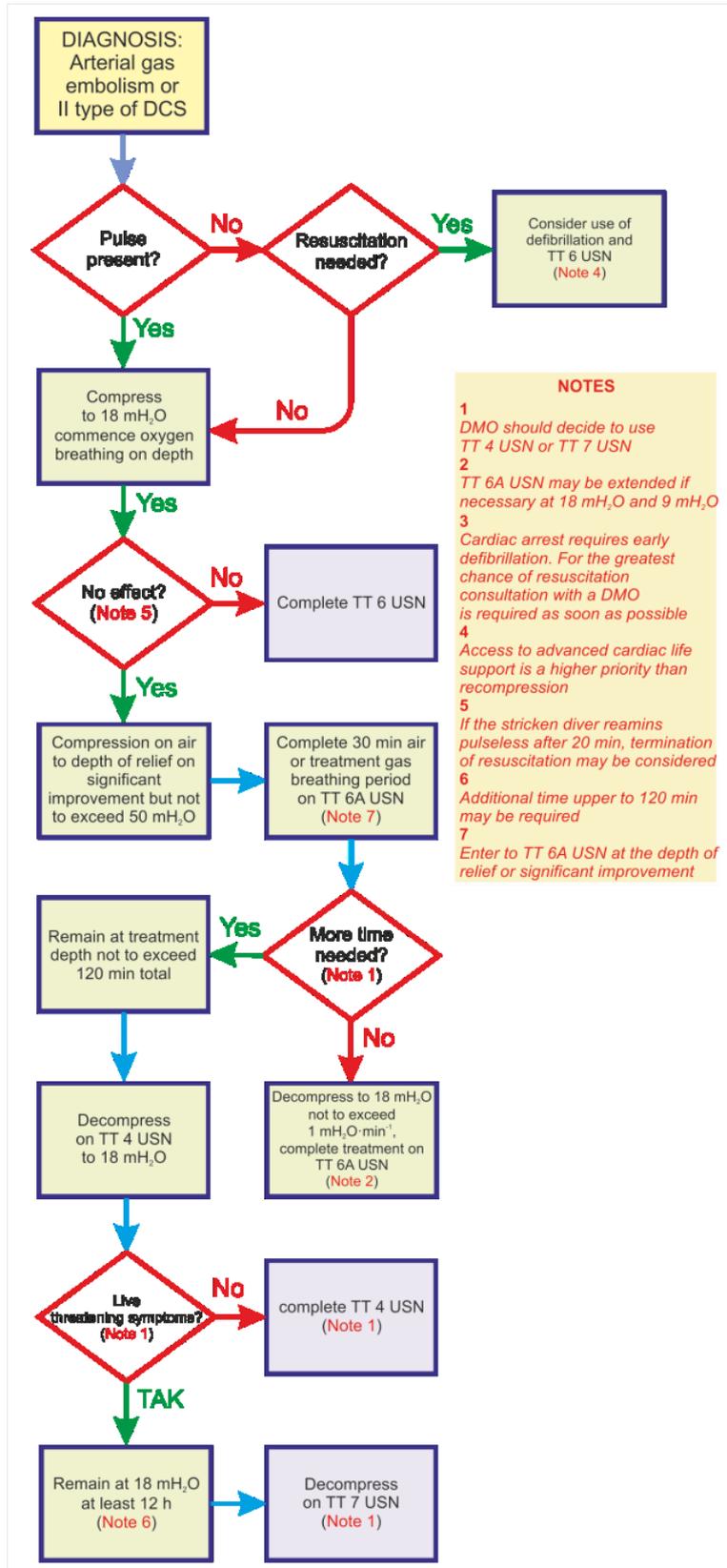


Fig.3. Algorithm for treatment of gas embolism *Type II DCS*

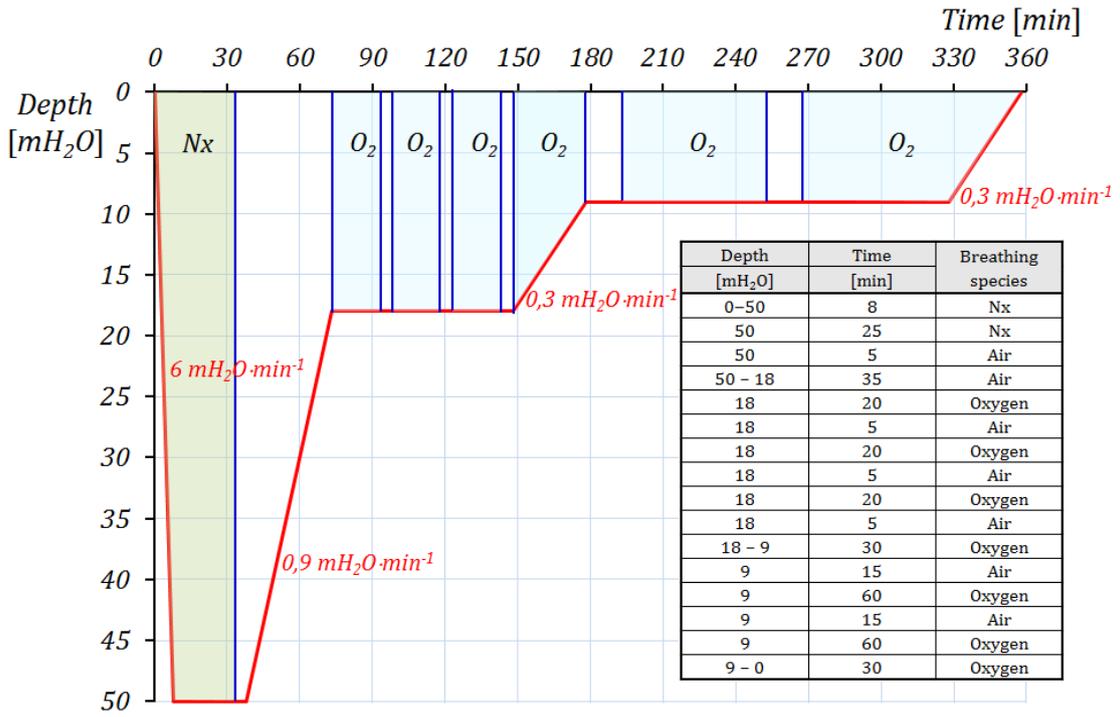


Fig.4. Treatment table TT 6A USN

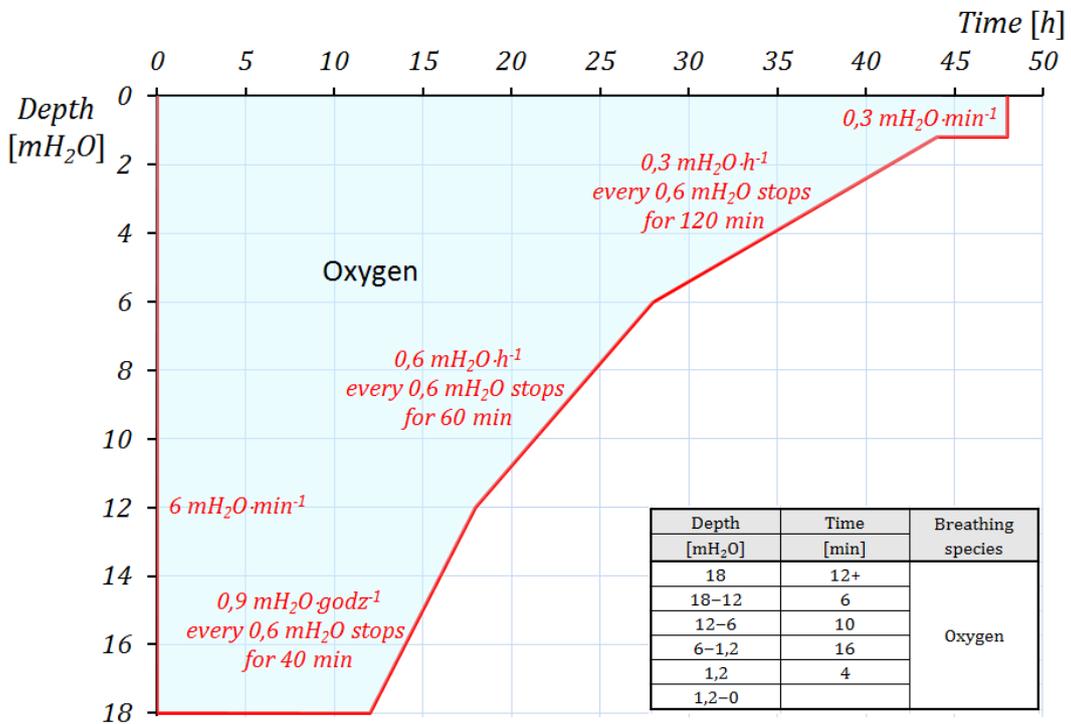


Fig.5. Treatment table TT 7 USN

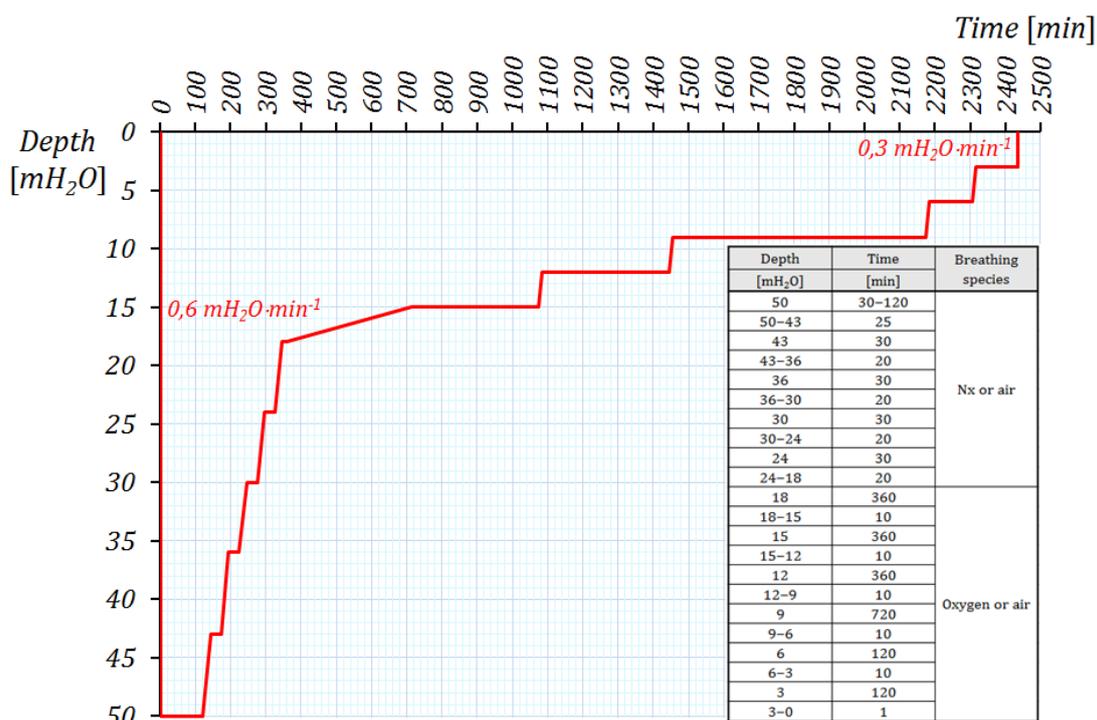


Fig.6. Treatment table *TT 4 USN*

TT 6A USN, *TT 7 USN* are used. They are shown **Fig.4–Fig.6**, and the procedure in **Fig.7**.

Air treatment table *TT 1A USN* is used to treat light symptoms of *DCS*, when the depth of relief does not exceed $20\text{ mH}_2\text{O}$ – **Fig.8**. Tables *TT 2A USN*, and *TT 3 USN* are used interchangeably to treat more serious cases: **Fig.9–Fig.10**. When the depth of relief exceeds $50\text{ mH}_2\text{O}$, the *TT 4 USN* should be used– **Fig.6**.

The supplement to the *DCS* treatment system are two *US Navy* tables: *TT 8 USN* and *TT 9 USN*. The *TT 8 USN* is an adaptation of the *TT 65 RN* used in the *DCS* treatment system by the *Royal Navy* and can be used when decompression stations have been omitted or the diver is thrown to the surface in an uncontrollable way. The *TT 8 USN* starts with compressing the diver to the depth of relief, but not higher than $68\text{ mH}_2\text{O}$ – **Fig.11**. The *TT 9 USN* is used to treat mild *DCS* symptoms following the previous treatment – **Fig.12**. It is also used to treat cases of carbon monoxide poisoning or poisoning by exhaust fumes.

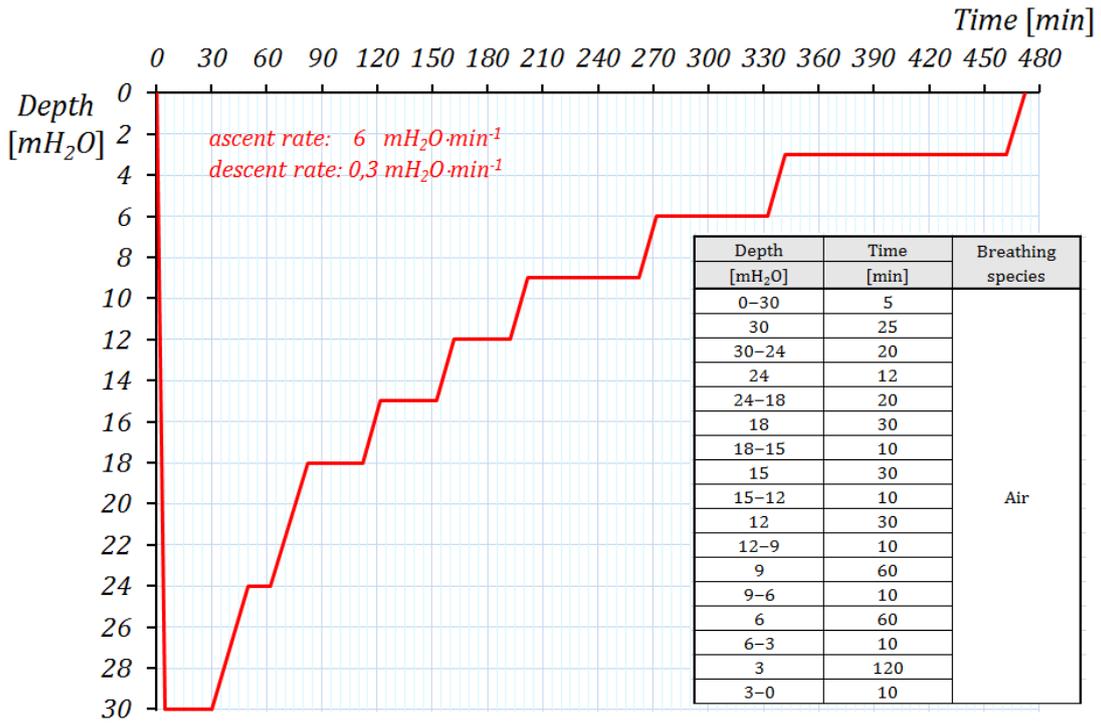


Fig.8. Treatment table TT 1A USN

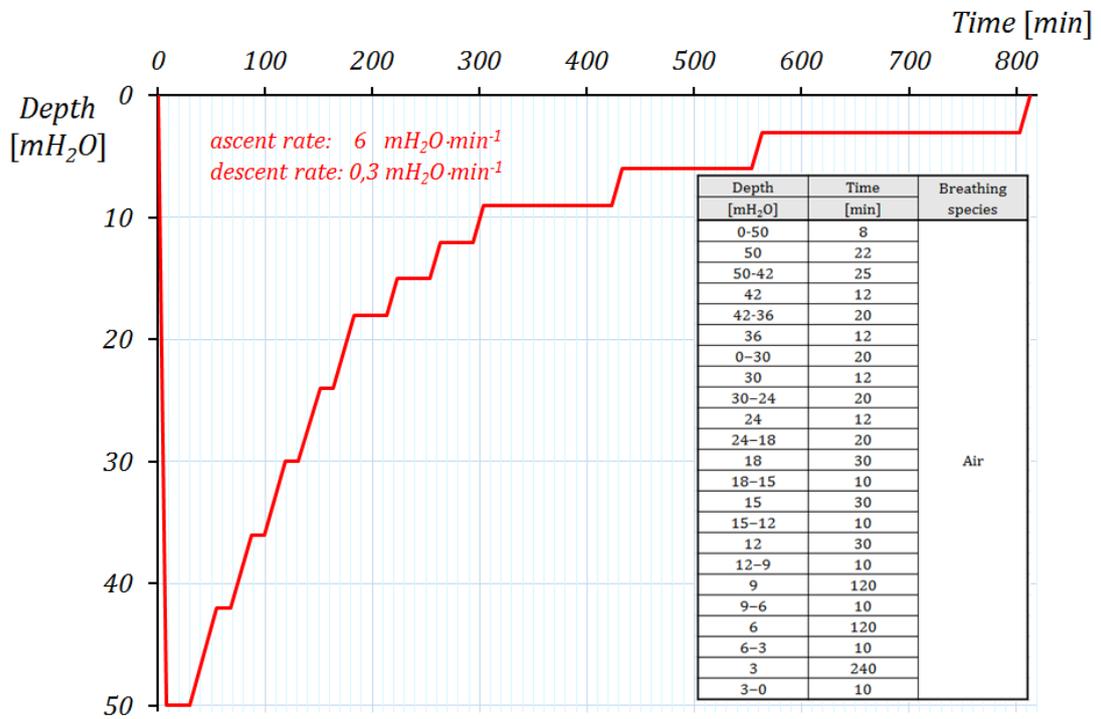


Fig.9. Treatment table TT 2A USN

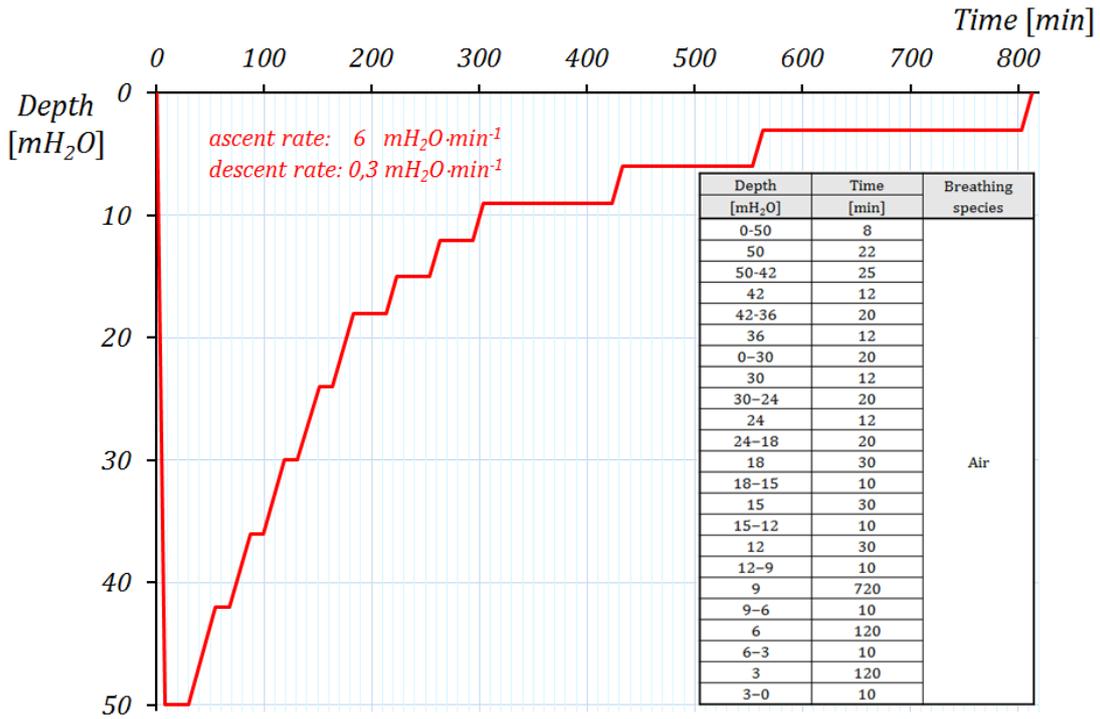


Fig.10. Treatment table TT 3 USN

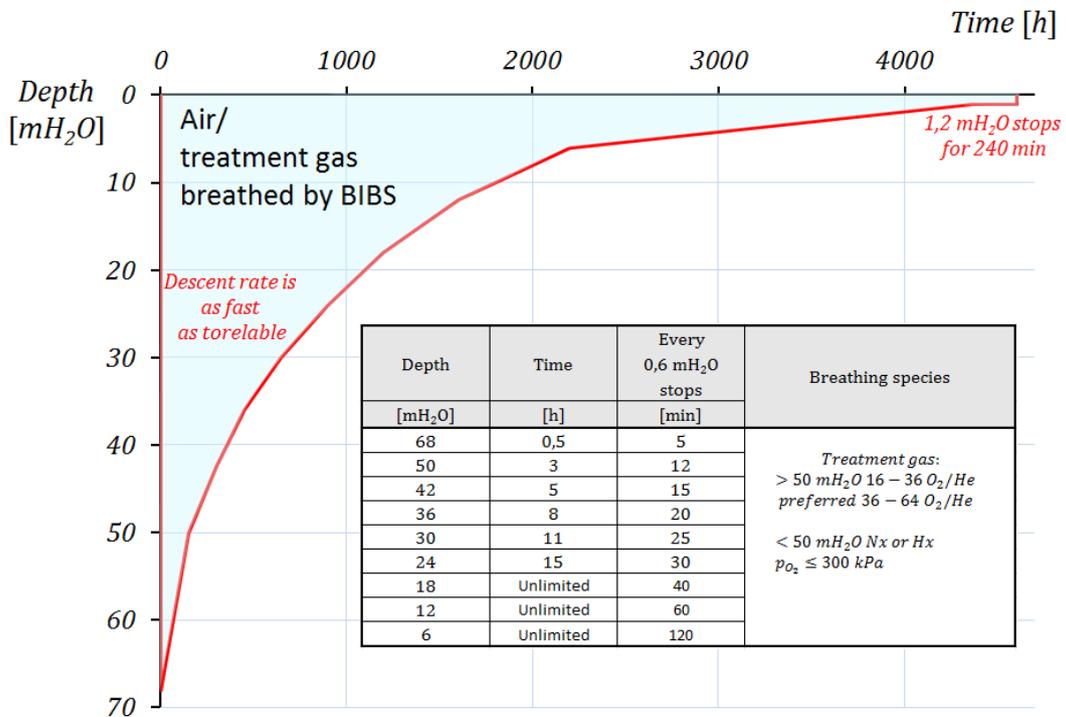
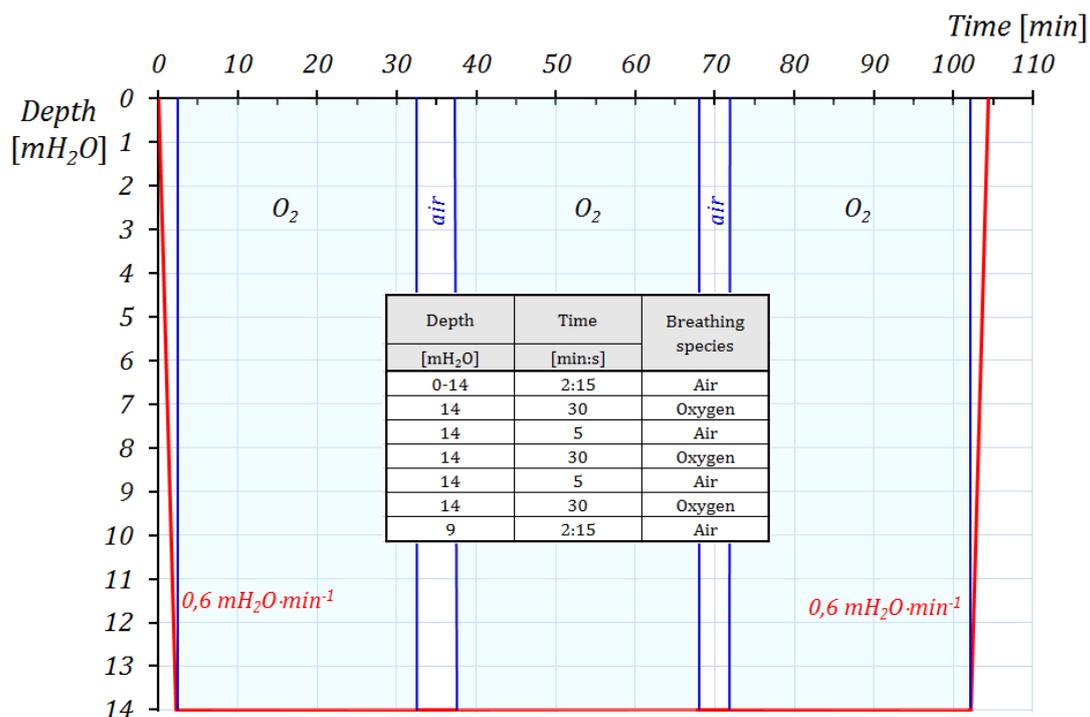


Fig.11. Treatment table TT 8 USN / TT 65 RN

Fig.12. Treatment table *TT 9 USN*

Russian air treatment tables

An interesting supplement system to the methods for air *DCS* treatment used by the *US Navy* are treatment tables air/*Tx*²⁰ proposed by *Военно Морской Флот России* which provide a capability of reaching the pressure of relief up to the depth of 100 *mH₂O* (5) – **Tab.3**.

Russian treatment tables *TT I BMΦ* – *TT III BMΦ* are tables which use only air/*O₂* as the treatment agent. The *TT I BMΦ* is dedicated to treating light symptoms of decompression sickness *Type I DCS* which disappear below the depth of 30 *mH₂O* during compression. The *TT II BMΦ* is dedicated to treating light symptoms of decompression sickness *Type I DCS*, where the depth of relief occurs below the depth of 50 *mH₂O* during compression. The *TT III BMΦ* is dedicated to treating mild symptoms of decompression sickness *Type I DCS*.

²⁰nitrogen-helium-oxygen mixture called *Trimix*

Tab.2A.

Russian air/oxygen treatment tables (5)

Recompression regime	Decompression regime	Maximum depth	Max time at max depth	Time to 1 st stop	1 st stop depth	Time at 1 st stop	Time to 2 nd stop	Next decompression stops [mH_2O]												
								30	28	26	24	22	20	18	16	14	12			
								Time at next decompression stops												
								[min]												
I	A	50	60	20	30	30	20													
	Б		120				14										107	202	245	
	В		180				10							101	166	182	202	245		
	Г		360				73	2	106	112	121	130	152	166	182	202	245			
II	A	70	60	20	50	30	32						65	182	203	245				
	Б		90				28					39	164	179	196	218	262			
	В		120				24		75	131	140	164	179	196	218	262				
III	A	100	15	30	70	60	46				113	152	164	178	196	218	262			
	Б					90	42		63	132	140	152	164	178	196	218	262			
	В					120	40	113	123	132	141	152	164	178	196	218	262			

Tab.2A. Continuation

Russian air/oxygen treatment tables (5)

Recompression regime	Decompression regime	Next decompression stops [mH_2O]					Total time				
		10	8	6	4	2	air	O_2	Totality		
		Time at next decompression stops on air (B) or O_2								[min]	[h: min]
		[min]									
I	A	30+140 _B	30 _B +60	90 _B +60	180 _B +60	60 _B +60	570	270	14:00		
		247 _B	305 _B	368 _B	235 _B	235 _B	1460	-	24:20		
	Б	280 _B	60+35 _B	105 _B +60	210 _B +60	70 _B +60	1318	240	25:58		
			328 _B	396 _B	253 _B	253 _B	2128	-	35:28		
	B	280 _B	60+35 _B	105 _B +60	210 _B +60	70 _B +60	1656	240	31:36		
		328 _B	396 _B	253 _B	253 _B	2466	-	40:16			
	Г	280 _B	328 _B	396 _B	253 _B	253 _B	3021	-	50:21		
II	A	30+150 _B	35 _B +60	105 _B +60	210 _B +60	70 _B +60	1347	270	26:57		
		280 _B	328 _B	296 _B	253 _B	253 _B	2287	-	38:07		
	Б	300 _B	60+40 _B	110 _B +60	225 _B +60	75 _B +60	1886	240	35:26		
			351 _B	425 _B	271 _B	271 _B	2754	-	45:54		
B	300 _B	351 _B	60+110 _B	225 _B +60	75 _B +60	2500	180	44:40			
			425 _B	271 _B	271 _B	3057	-	50:57			
III	A	300 _B	351 _B	60+115 _B	230 _B +60	75 _B +60	2490	180	44:30		
				425 _B	271 _B	271 _B	3037	-	50:37		
	Б	300 _B	351 _B	425 _B	60+230 _B	75 _B +60	3048	120	52:48		
271 _B					271 _B	3285	-	54:45			
	B	300 _B	351 _B	425 _B	271 _B	271 _B	3487	-	58:07		

Tab.2B.

Russian air/ Tx treatment table recompression regime IV (5)

Recompression regime	Decompression regime	Maximum depth [mH_2O]	Max time at max depth [min]	Time to 1 st stop [min]	Next decompression stops [mH_2O]								
					88	86	84	82	80	78	76	74	72
					Time at next decompression stops when breathing with the Tx 7% O_2 [min]								
IV	A	100	60	6				2	7	11	11	11	12
	Б		180	8			5	20	20	25	35	35	45
	B		360	24	90	90	100	90	100	100	110	100	110

Decompression regime	Next decompression stops [mH_2O]																										
	70	68	66	64	62	60	58	56	54	52	50	48	46	44	42	40	38	36	34	32	30	28	26	24	22	20	
	Time at next decompression stops when breathing with the Tx 10% O_2 [min]																		Time at next decompression stops when breathing with the air [min]								
A	8	13	18	18	19	20	29	33	35	46	54	66	79	108	136	136	144	152	159	91	94	98	102	108	117	130	
Б	40	40	50	55	65	75	90	95	100	110	115	125	130	140	145	150	155	160	170	95	95	100	105	110	120	135	
B	80	80	80	80	90	90	90	100	100	110	120	125	130	140	145	150	160	160	170	180	190	200	220	230	250	120	

Tab.2B. Continuation

Russian air/ T_x treatment table recompression regime IV (5)

Decompression regime	Next decompression stops [mH_2O]									Total time			Totality	
	18	16	14	12	10	8	6	4	2	T_x 7% O_2	T_x 10% O_2	air	[h]	[min]
	Time at next decompression stops when breathing with the air													
	[min]													
A	146	162	181	202	227	255	290	334	394	60	1273	2931	71	64
B	150	170	190	210	240	270	310	360	430	193	2010	3090	88	13
B	140	160	170	210	250	300	370	470	610	914	3470	2800	119	44

Treatment tables $TT IV BM\Phi - TT V BM\Phi$ are tables using Tx as the treatment agent, because from the depth of $70 mH_2O$ pure helium or Hx^{21} , having the content of helium within Hx : $[18; 20]\%_{v} O_2/He$, are used for compression. The $TT IV BM\Phi$ is used to treat severe forms of decompression sickness *Type I DCS* and the $TT V BM\Phi$ to treat neurological *Type II DCS* symptoms.

COMEX treatment tables

Treatment tables of the French firm *COMEX* constitute the base for treating the most severe *DCS* forms which can occur during both saturated diving and outside the saturation zone, to the maximum operational depth of $450 mH_2O$ (6). Light cases are treated out of saturation zone but more severe ones require facilities for providing saturation.

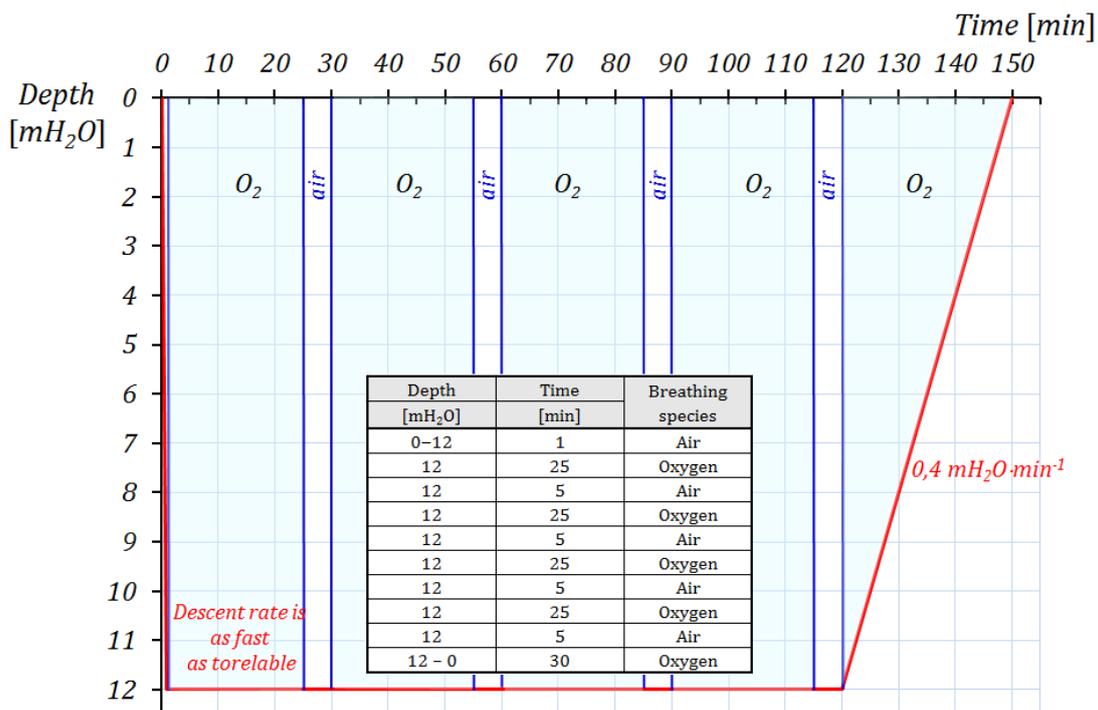


Fig.13. Treatment table $TT 12_{86} Cx (6)$

²¹helium-oxygen mixture called *Heliox*

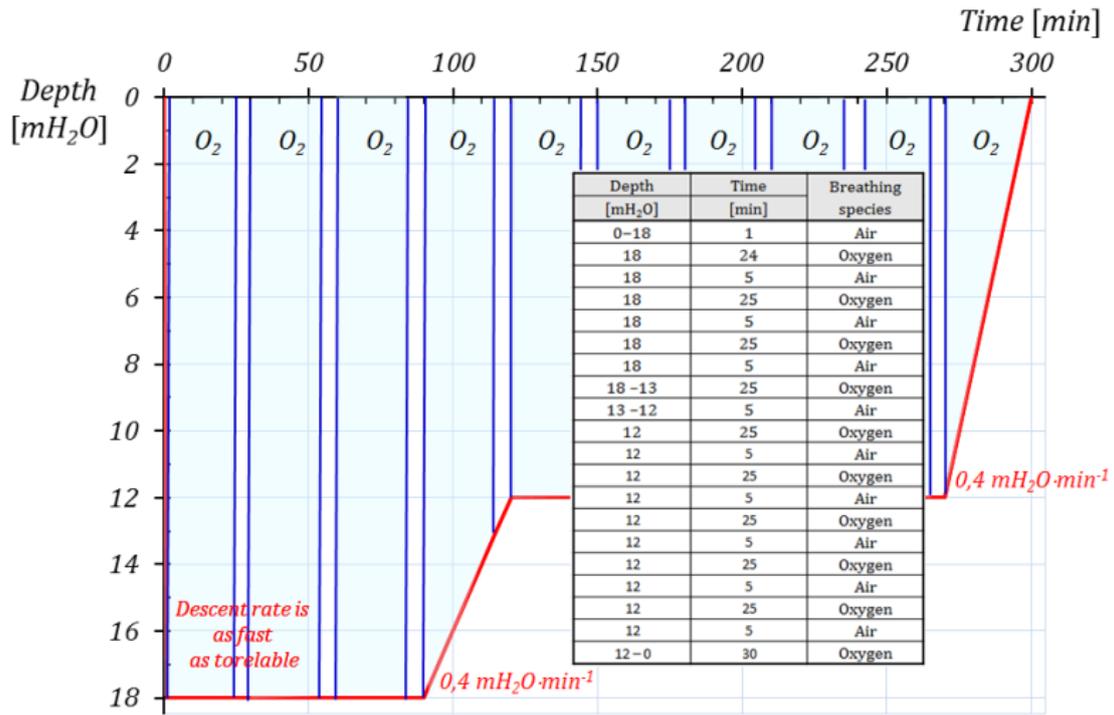


Fig.14. Treatment table TT 18₈₆ Cx (6)

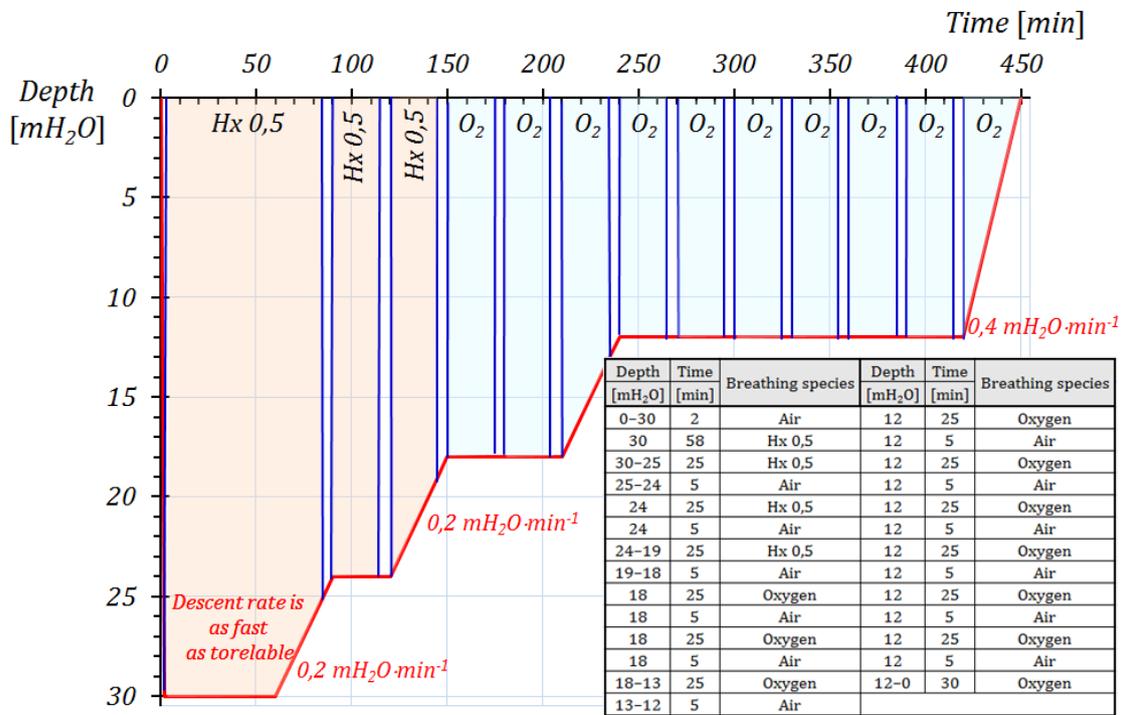


Fig.15. Treatment table TT 30₈₆ Cx (6)

Treatment tables: $TT\ 12_{86}\ Cx$, $TT\ 18_{86}\ Cx$ and $TT\ 30_{86}\ Cx$ are similar to the *US Navy* oxygen treatment tables (4). The $TT\ 12_{86}\ Cx$ is used to treat light *DCS* symptoms and the $TT\ 18_{86}\ Cx$ to treat more severe *Type I DCS*: **Fig.13–Fig.14**. The $TT\ 30_{86}\ Cx$ is used to treat neurological symptoms *Type II DCS* – **Fig.15**. These tables assume pharmacological support through administering acetylsalicylic acid²² and in the case of the $TT\ 30_{86}\ Cx$ also an anti-inflammatory medication, hormone medicine having inflammatory properties, and isotonic infusion liquids²³.

Treatment tables: $TT\ 12_{86}\ Cx$, and $TT\ 18_{86}\ Cx$ constitute a treatment recompression system for symptoms *Type I DCS* occurring after air or *Hx* diving. If after $\tau = 15\ min$ from compression to $12\ mH_2O$ *DCS* symptoms disappear the treatment is based on the $TT\ 12_{86}\ Cx$. If this is not the case, treatment continuation is based on the $TT\ 18_{86}\ Cx$. Symptoms *Type II DCS* are treated following the $TT\ 30_{86}\ Cx$.

When decompression stations have been omitted or the diver has been blow up to the surface during an air dive outside the saturation zone, for *Type I DCS* the $TT\ 18_{86}\ Cx$ is used and for *Type II DCS* symptoms treatment is based on the $TT\ 30_{86}\ Cx$. If there exists a need for extending the treatment time in accordance with the $TT\ 30_{86}\ Cx$, the time of stop at the depth $H = 30\ mH_2O$ can be prolonged to maximum $\tau = 3\ h$.

When *DCS* occurs during air saturation or *Hx* saturation dives, or operational *Hx* diving outside the saturation zone, for *Type I DCS* symptoms the $TT\ B_{86}\ Cx$ is used, if the symptoms disappear within $15\ min$ with standard decompression, if not, the $TT\ SB_{86}\ Cx$ is used with standard decompression also – **tab.3**. When the condition of the diver worsens, special saturation table $TT\ N_{86}\ Cx$ should be used – **Tab.4**.

Saturation table $TT\ N_{86}\ Cx$ is also used to treat *Type II DCS* symptoms following air or *Hx* saturation, or operational dives *Hx* outside the saturation zone. When using saturation table $TT\ N_{86}\ Cx$ for treating cases of *DCS* developed during or after *Hx* dives, both saturated and outside the saturation zone, decompression is conducted on the continuous basis, with the time passage through the individual depth $t = 60\ min \cdot mH_2O^{-1}$ within the depth range $H \in [15; 200]mH_2O$, keeping the oxygen partial pressure at the level $p_{O_2} \in [60; 63]kPa$ in *Hx* making the atmosphere of complex. Decompression from the depth $H = 15\ mH_2O$ to the surface is conducted with

²²*Aspirin* (Germany), *Aspegic* (France), *Polopiryna* (Poland) itd.

²³Ringer's solution or balanced salt solution (physiological pH and isotonic salt concentration)

Tab.3.

Treatment tables *TT B₈₆ Cx* i *TT SB₈₆ Cx* (6)

<i>TT B₈₆ Cx</i>			<i>TT SB₈₆ Cx</i>				
Depth of treatment	<i>Hx</i> bounce dive and saturation	Air saturation	Depth of accident	Depth of treatment	BIBS breathing duration	<i>Hx</i> bounce dive and saturation	Air saturation
[<i>mH₂O</i>]			[<i>mH₂O</i>]	[<i>mH₂O</i>]	[<i>min</i>]		
0–12	Oxygen	Oxygen	0–12	+6	120	Oxygen	Oxygen
19–40	<i>Hx</i> 0,50	<i>Nx</i> 0,50	13–31	+9	120	<i>Hx</i> 0,50	<i>Nx</i> 0,50
41–110	<i>Hx</i> 0,20		32–98	+12	120	<i>Hx</i> 0,20	
111–210	<i>Hx</i> 0,10		99–195	+15	120	<i>Hx</i> 0,10	
211–360	<i>Hx</i> 0,05		196–345	+15	120	<i>Hx</i> 0,05	
361–450	<i>Hx</i> 0,03		346–450	+15	120	<i>Hx</i> 0,03	

Tab.4.

Treatment table *TT N₈₆ Cx* (6)

<i>TT N₈₆ Cx</i>			
Depth of accident	BIBS breathing duration	<i>Hx</i> bounce dive and saturation	Air saturation
[<i>mH₂O</i>]	[<i>min</i>]		
20–40	120	<i>Hx</i> 0,50	<i>Nx</i> 0,50
41–110	180	<i>Hx</i> 0,20	
111–210	240	<i>Hx</i> 0,10	
211–360	300	<i>Hx</i> 0,05	
361–450	360	<i>Hx</i> 0,03	

the time passage through the individual depth $t = 80 \text{ min} \cdot \text{mH}_2\text{O}^{-1}$, keeping the oxygen volume content at the level $C_{O_2} \in [23; 24]\%_v$ in Hx making the atmosphere of complex. When treating *DCS* cases developed during or after saturation dives based on nitrox Nx , for the depth range $H \in [15; 30]\text{mH}_2\text{O}$ decompression is conducted on the continuous basis at the speed of passage $v = 120 \text{ min} \cdot \text{mH}_2\text{O}^{-1}$, keeping the oxygen partial pressure in Nx making the atmosphere of complex at the level $p_{O_2} \in [60; 63]\text{kPa}$. From the depth $H = 15 \text{ mH}_2\text{O}$ to the surface decompression is conducted at the speed of passage $v = 180 \text{ min} \cdot \text{mH}_2\text{O}^{-1}$, keeping the oxygen volume content in Nx making the atmosphere of complex at the level $C_{O_2} \in [23; 24]\%_v$.

As there are no special procedures for cases when *DCS* symptoms appear during decompression in water, as is the case in other systems²⁴, when they appear, the decompression is discontinued. The diver is brought to the surface as quickly as possible and further treatment is administered as in the case when a diver is blow up to the surface.

Discussion

The commonly used worldwide dive decompression tables usually allow for only a low workload of the diver underwater²⁵. This is caused by the existence, in hyperbaric conditions, of additional workload of the respiratory system and action of the water environment itself. For example, divers use suits which require additional effort during work, or because they cannot breathe under water divers have to carry a substantial amount of breathing agent, which requires additional effort to carry it, etc. Only some of the decompression tables take into account the additional effort necessary to perform effective work under water, determining its maximum border magnitude as the mean load. This limitation is associated not only with performing work in the water environment itself but also with the necessity to later undergo decompression. The decompression process will be disturbed by metabolism products, which occur after the a high amount effort is taken. For example, acidification of muscles with lactic acid caused by anaerobic changes leads to acidification of blood and because of this hemoglobin loses its capacities of carrying oxygen (15). This hampers gas exchange and as a result it disturbs the process of decompression. A detailed analysis of the effect of workload on the planned decompression is very difficult. It is only

²⁴for example (23)

²⁵almost without doing any extra work except diving

saturation dives that give divers the possibility to make relatively high physical efforts as their decompression process is separated from the work period by a suitably long rest period dedicated to stabilization.

Sometimes, there exists a high probability for a diver to get stuck underwater, e.g. inside a wreck during penetration. Divers, who work in support of a rescue operation, outside the saturation zone, under emergency situations, may, as a result of face improper decompression, face a hazard of e.g. blow up to the surface without decompression or emergency omitting some elements of decompression.

It follows from the carried out analysis that it is relatively easy to select a treatment recompression system for *DCS* cases, which occurred in the course of short-term operational dives²⁶ outside the sub-saturation or full saturation zone. Out of many existing systems the most common is the system developed and published by the *US Navy* (4). It can be used to support the *DCS* treatment process in divers, providing support to saturation divers in the course of wreck penetration. Such divers can be employed to provide assistance in straightening and winding umbilicals of the saturation diver and underwater vehicles, deliver tools, render assistance in transporting casualties and in other support actions carried out outside the sub-saturation and full saturation zone. These methods can also be used to treat light *DCS* occurred after saturation diving with *Nx*. The *US Navy* system does not allow exceeding $70 \text{ mH}_2\text{O}$ to produce the relief effect in a patient. Extending the *US Navy* system by air tables system $\text{BM}\Phi$ offers additional treatment capabilities. However, the treatment is conducted in difficult conditions, hard to accept without any doubt. Complications will appear when respiratory failure occurs in in the casualty, e.g. as a result of pulmonary barotrauma²⁷, which occurs when the diver is blown up to the surface. In such a case effective breathing *Tx*, *Nx* or oxygen under high pressure can cause airway resistance in the patient so high that it not only will hamper the treatment process but it also may prevent him from breathing and induce asphyxia²⁸. When this is the case, using *Hx* may turn out necessary. It should be borne in mind, however, that transfer from the heavy breathing agent to a lighter one often causes occurrence of *DCS* symptoms through counterdiffusion, even if this is not accompanied by change in pressure (16; 17; 18; 19). A comparative analysis of the proposed tables, based on one selected approach to estimation of *DCS* hazard, is worth conducting.

²⁶except for severe cases when the diver was thrown to the surface

²⁷pulmonary barotrauma

²⁸insufficient amount of oxygen in the body leading to loss of consciousness

Treatment of *Type I DCS* and *Type II DCS* cases is based, first of all, on hyperbaric method. The first treatment parameter is the pressure causing compression of gas in free phase precipitated in tissues allowing it to dissolve in fluids existing in tissues. This causes a decrease in mechanical impact of gas on tissues, unblocking blood flow through capillaries, cessation of response reaction of the immunity system identifying gas bubbles as aggressors.

The second parameter is the time needed for homeostasis, resorption of gas in tissues and bodily fluids, and transport of dissolved gas excess to the blood. Then eliminating the gas excess from the body during the decompression by the blood prefunding the bodily tissues and lung tissue – **Fig.16.**

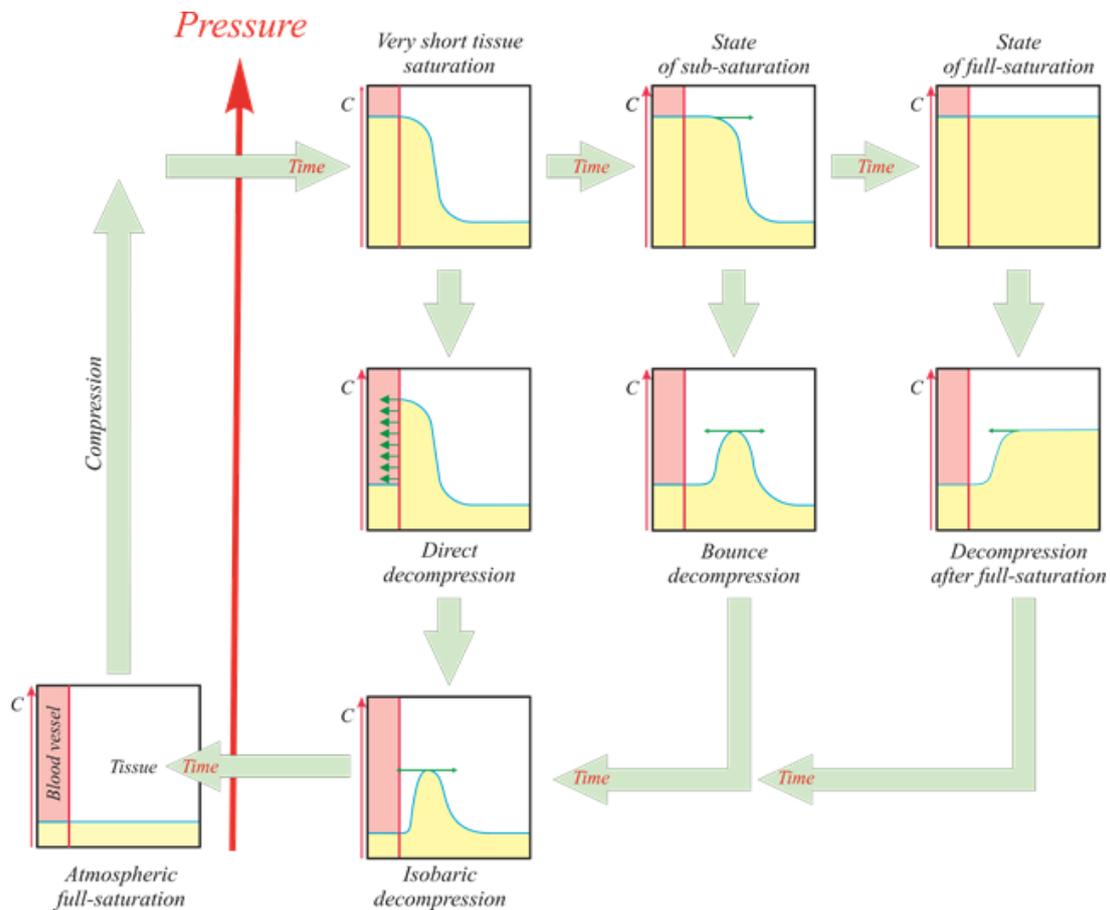


Fig.16. Main overview for overload of inert gas in body tissues in the hyper baric environment and its elimination during the decompression process

The factor intensifying the treatment process is the use of oxygen or mixtures enriched with oxygen. Oxygen is rapidly metabolized in the body tissues leaving a free space to transport inert gas by the blood. Sometimes, other drugs are needed in emergency cases and for the treatment intensification. The therapeutic recompression process can be supported pharmacologically by blood thinning, maximizing hydration, administering pain killers, anti-coagulants²⁹ and anti-inflammatory medication.

The systems of treating *DCS* with therapeutic recompression, described here, constitute a vivid example of uses of these briefly presented parameters. But free use of pressure, time and composition of the breathing agent is often made difficult because of the phenomenon of oxygen toxicity, counterdiffusion, respiratory failure, etc.

Modelling counterdiffusion has, so far, not resulted in developing a comprehensive theory taking counterdiffusion into account during the decompression process, despite the fact that counterdiffusion cases often occur in diving (20; 21). The occurrence of *DCS* cases accompanied by counterdiffusion can be divided into two forms: *SICD*³⁰ and *DTICD*³¹. The *SICD* symptoms occur when the diver is breathing a gas different than gas surrounding the body (10). For example, in the *Polish Navy* the deep diving technology is used with *Tx* as a breathing agent during work performed under water, and the drysuit is inflated with air. During decompression the diver substitutes the breathing agent for air during the transfer under pressure and then into oxygen decompression. In all divers light skin *DCS* symptoms occur, e.g. general skin itching. The *DTICD* symptoms accompany sequential changes in the breathing agent, usually during decompression. The sequential changes in the breathing agent from lighter to heavier mixture and from heavier to lighter mixture can cause *DCS*, even if they are not accompanied by changes in depth – *ICD*³². A combination of counterdiffusion phenomena through cell membranes and dissolution of gases in tissues can lead to exceeding the dissolution border and the precipitation of gas in free phase in tissues, causing *DCS*, due to the differences in diffusion and solubility coefficients especially of helium and nitrogen (22).

A possible counterdiffusion occurrence has to be taken into account when the *US Navy* method is used for treatment after an accident during deep diving or saturation diving. When the treatment methods recommended by the French firm *COMEX* are used the probability of counterdiffusion occur-

²⁹anticoagulants are medicines that reduce the ability of the blood to clot

³⁰*Superficial Isobaric Counterdiffusion*

³¹*Deep Tissue Isobaric Counterdiffusion*

³²*Isobaric Counterdiffusion*

rence can be minimized by choosing for treatment the breathing agent on which *DCS* symptoms occurred.

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References

1. **Berghage T.E., Vorosmarti Jr. J., Barnard E.E.P.** *Recompression Treatment Tables Used Throughout the World by Government and Industry*. Bethesda Maryland : US Naval Medical Research Center, 1978. NMRI 78-16.
2. **Standards Related Document.** *Allied guide to diving medical disorders – national information*. Edition A Version 1. 2016. ADivP-02.1.
3. **NATO Standardization Agreement STANAG 1432 UD.** *Allied guide to diving medical disorders*. Edition 4. Brussels : NATO Standardization Office, 2014. NSO(NAVAL)1053(2014)UD/1432.
4. **US Navy diving manual.** *Praca zbiorowa (revision 7)*. The Direction of Commander : Naval Sea Systems Command, 2011. SS521-AG-PRO-010 0910-LP-115-1921.
5. **Praca zbiorowa.** *Справочник специалиста аварийно-спасательной службы ВМФ*. [red.] Н.П. Чикер. Москва : Военное издательство МО СССР, 1968. Том Часть 3.
6. **Comex Marseille.** *Medical Book*. Marseille : Comex, 1986.
7. **Kłos R.** *Aparaty Nurkowe z regeneracją czynnika oddechowego*. Poznań : COOPgraf, 2000. ISBN 83-909187-2-2.
8. **Wienke B.R.** *Basic decompression theory and application*. Flagstaff : Best Publishing Co., 2003. ISBN 1-930536-14-3.
9. **Kłos R., Konarski M., Olszański R.** The implementation of factor analysis for the evaluation of selected blood parameter changes induced by hyperbaric exposure. *International Maritime Health*. **55**(2004)87-101.
10. **Lambertsen C. J., Idicula J.** A new gas lesion syndrome in man, induced by "isobaric gas counterdiffusion ". *J. Appl. Physiol.* **39**(1975)434-443.
11. **Lambertsen C.J.** *Studies in isobaric counterdiffusion* . Filadelfia : Institute for Environmental Medicine , 1986.
12. **Doolette D.J., Mitchell S.J.** Biophysical basis for inner ear decompression sickness. *J. Appl. Physiol.* **94**(2003)2145–2150.

13. **Strauss R.H.** *Diving medicine*. New York : Grune & Stratton Inc., 1976. ISBN 0-8089-0699-2.
14. **Kłós R.** *Helioksove nurkowania saturowane - podstawy teoretyczne do prowadzenia nurkowań i szkolenia*. Wydanie II (poprawione). Gdynia : Polskie Towarzystwo Medycyny i Techniki Hipernbarycznej, 2014. ISBN 978-83-938322-1-7.
15. —. *Możliwości doboru ekspozycji tlenowo-nitroksowych dla aparatu nurkowego typu AMPHORA - założenia do nurkowań standardowych i eksperymentalnych*. Gdynia : Polskie Towarzystwo Medycyny i Techniki Hiperbarycznej, 2012. ISBN 978-83-924989-8-8.
16. **Hamilton R.W., Adams G.M., Harvey C.A., Knight D.R.** *SHAD-NISAT: A composite study of simulated shallow saturation diving*. Groton : Naval Submarine Medical Research Laboratory Submarine Base, 1982. Report Number 985.
17. **Harvey C.A.** *Shallow saturation hyperbaric exposures to nitrogen-oxygen environments and isobaric switches to helium-oxygen*. Toronto : Hyperbaric Medical Society, Inc., 1977.
18. **Hyldegaard O, Jensen T.** Effect of heliox, oxygen and air breathing on helium bubbles after heliox diving. *UHMS Journal*. **34**(2007)107-122.
19. **UHMS 22 Workshop.** *Isobaric inert gas counterdiffusion*. [red.] R.C. Lambertsen C. J. Bornmann. Philadelphia : Undersea Medical Society, 1979. UMS Publication Number 54WS(IC)1-11-82.
20. **Wienke B.R.** *Technical diving in depth*. Flagstaff : Best Publishing Co., 2001. ISBN 0-941332-97-7.
21. **Imbert J-P.** *Proposition of a perfusion limited model for isobaric counterdiffusion*. Philadelphia : University of Pennsylvania Medical Center, 1975. Report Number 07-01-1975.
22. **Karreman G., Lambertsen C.J.** Kinetics of isobaric counterdiffusion. *Bulletin of Mathematical Biology*. 1977, Tom 39, ISSN 0092-8240, strony 587-595.
23. **US Navy diving manual.** *Praca zbiorowa (revision 6)*. The Direction of Commander : Naval Sea Systems Command, 2008. 0910-LP-106-0957.
24. **Kłós R.** Planning special combat operations with the use of the AMPHORA rebreather. *Polish Hyperbaric Research*. **38**(2012)29-130, ISSN 1734-7009.