	Title of report: **Emergency decompression from heliox saturation dives**			
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Summary:

This study was done on initiative from PSA. The objective was to investigate emergency decompression as an alternative, in cases where the evacuation from a saturation facility of divers at increased pressure, is not available for whatever reason. It is stressed that it will not be the purpose of such procedures to prevent pain or discomfort, but to prevent serious injury and death.

Historical survey shows that need for hyperbaric evacuation / emergency decompression is not a very rare situation as a "probability-weighted" sum of such incidents was found to be 32.

So it is important that plans including accelerated emergency decompression, are available as a supplement to hyperbaric evacuation of divers. The recommendations in DMAC 31, with a few additions/modifications (possible gas switch to air) is a good start for making practical procedures. The risk of injury will be high, but if the decompression speed is not too high the chance of survival may be good. Probability for injury decrease with shallower saturation depth.

The risk by using emergency decompression by itself, and up against the risk, which hyperbaric evacuation may represent is discussed. A workshop with relevant people from the industry was organized, and documentation related to emergency and accelerated decompression was identified and distributed to participants of the workshop, involving medical and operational expertise. Based on the feedback from workshop participants further documentation was collected and included in this report.

Key Words in English:	Key Words in Norwegian:
Saturation Diving Decompression Emergency procedures	Metningsdykk Dekompresjon Nødprosedyrer

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1 INTRODUCTION

This report was written as a commission for PSA.

Olav Hauso, Bjarne Sandvik and John Arne Ask from PSA have contributed with input and comments to the report.

1.1 Background

In the event that the integrity of a diving vessel with divers in saturation, are threatened, it may be necessary to evacuate the divers. The preferred method is to do a hyperbaric evacuation by keeping the divers at the saturation pressure till they arrive at a place where decompression can be done under controlled conditions.

For various reasons it may not be feasible to apply this preferred method. An alternative method may be to decompress the divers quicker than prescribed by normal procedures, in the hyperbaric chambers on board and evacuate them to a place where they can be safely recompressed.

This project was initiated to analyze this alternative evacuation method.

The project have connection to the earlier project which described "The status of knowledge and future perspectives 2012 - Good journey home - safe decompression of the diver" [1].

1.2 Scope

The project started with identification of a number of scenarios where such procedures could be appropriate. Procedures for emergency decompression in saturation diving were reviewed and risk conditions compared with the evacuation under pressure in the lifeboat described.

Actual emergency decompression procedures are not presented. It is stressed that it will not be the purpose of such procedures to prevent pain or discomfort, but to prevent serious injury or death.

1.3 Definitions & abbreviations

This report will use the term "emergency decompression" for any accelerated decompression procedure initiated due to an emergency. This "emergency" could be fire, loss of stability, hull integrity or similar.

CO_2	carbon dioxide
DMAC	Diving Medical Advisory Committee
DP	dynamic positioning
DSV	diving support vessel
HAZID	hazard identification
HAZOP	hazard and operability study
HRC	hyperbaric rescue or emergency/evacuation chamber
HLB	hyperbaric lifeboat (include SPHL)
HRF	hyperbaric reception facility
HRU	hyperbaric rescue unit (covers HLB, HRC, HRV and SPHL)
HRV	hyperbaric rescue vessel (Hyperbaric Lifeboat)
HSE	Health and Safety Authority (UK)
IMCA	The International Marine Contractors Association
IMO	International Maritime Organisation
IOGP	The International Assosiation of Oil and Gas Producers
kPa	kilopascal
LSP	life support package
msw	meters of sea water
N_2	nitrogen
NOROG	the Norwegian Oil and Gas Assosiation
NEDU	Navy Experimental Diving Unit (US)
O_2	oxygen
pCO_2	partial pressure of CO_2
pO_2	partial pressure of O_2
PSA	Petroleum Safety Authority (Petroleumstilsynet)
SPHL	self-propelled hyperbaric life boat

2 METHODS

Documentation related to emergency evacuation and accelerated decompression was identified and evaluated.

Relevant background material was distributed to participants of a workshop. This included links to:

1. IMCA D052 [2]
2. DMAC 31 [3]
3. A DMAC workshop report [4]
4. IMO guidelines [5]
5. A report on hyperbaric evacuation contingency planning [6]
6. A directory of commercial diving incidents [7]
7. *A report of US-Gulf of Mexico Diving Safety Work Group* on Hyperbaric Evacuation System Planning. [8]
8. Relevant IOGP reports [9, 10]

Preliminary versions of annexes 1-3 were also distributed.

Minutes (in Norwegian language) from workshop is found in annex 4.

Based on the feedback from workshop participants, further documentation was collected and formed a basis for this report. Annex 5 and 6 were supplied by Øyvind Lønnechen.

Then the inputs were processed, further documentation collected and a draft report was sent out for comments.

In this final report the annex 7 has been added after check by Jan Risberg.

3 RESULTS

3.1 Scenarios imposing differentiated approach for evacuation of divers in saturation

The preferred method of evacuation of divers from a pressurized saturation system is transfer to a HRU. Transfer of divers to and launching of a HRU is a complex procedure, which involves risk, and may fail due to human, technological or operational constraints. In such a situation the remaining alternative would be to accelerate decompression of the divers within the chamber complex.

Incidents where evacuation and/or emergency decompression have or could have been initiated are listed in annex 1.The data is mostly retrieved from an internet database [7] and The Gulf of Mexico, US Diving Safety Work Group [8], but also sources that include books, magazines, internet, reports, and personal communication (Leif-Tore Skjerven etc).

Some of the incidents point out scenarios where HRU cannot be used. A number of the incidents occurred at a time where HRUs were not installed. In incident 13, ID85.01, there was a problem with launching the only one SPHL because the DSV was grounded and there was no water under the SPHL. In incident 24, ID97.01 the HRC was ripped off. In incident 26, ID105.01 apparently the surface system was damaged and a wet bell to bell transfer to another DSV was carried out and in 27, ID105.02 the SPHL was inside the fire.

It is fair to say that the need for hyperbaric evacuation / emergency decompression is not a very rare situation as a “probability-weighted” sum of such incidents was found to be 32.

It may also be situations where there is insufficient personnel to man the SPHL.

In addition there may be cases with some divers at a very shallow depth while others are deeper (split level). In this situation *the risk of an accelerated decompression has to be compared to the risk of being compressed and evacuate at a much deeper depth*.

3.2 Regulations and recommendations

The Gulf of Mexico diving safety work group report (2016) [8] contain a table which give a good comparison on differences in requirements related to hyperbaric evacuation that have been set by different organizations (e.g. IMCA, IMO and IOGP). It does not, however, cover emergency decompression.

The regulations on the Norwegian and UK continental shelves require plan for evacuation, without specifically requiring emergency decompressions plans. Both refer to , IMCA D 052 [2] (Norwegian through NORSOK U-100 [11]). IMCA D 052 [2] refer in turn to DMAC 31 [3]. The IOGP recommendations [10] don’t stipulate requirements for emergency decompressions plans.

3.3 Relevant physiological/medical factors

Physiological and medical factors relevant for decisions on emergency decompression are described in annex 2 “Emergency decompression from saturation – from the physiology perspective” by Andreas Møllerløkken.

3.3.1 Rapid decompression from saturation exposure – case stories

Description of known cases internationally where emergency decompression was made, were presented in the 2011 DMAC workshop [4].

1. North Sea. Rapid surfacing of bell with two divers from 70 msw after loss of bell weights (ascent within minutes) led to one immediate death and one survivor with severe permanent injury from DCS [12] . For more detailed description of this case, see annex 7.

2. North Sea. Typhoon event. Rapid decompression from saturation at 170 ft in 31.5 hours achieved without ill effect.

3. India. Decompression from storage depth of 42 msw. Transient descent to 85 msw followed by upward excursion to 54 msw over 3 hours. Ascent to 34 msw over 8 hours, then 2 msw per hour to 11 msw. Final ascent from 11 msw to surface over 25 min (total about 23 hours from 85 msw but effectively 20 hours from 54 msw).

Also the following trial information was considered in the workshop:

Ascent trials. Divers during routine sat ascent from 200 msw, after 30 min on high pO_2 breathing mixture ascended from 125 to 104 msw in 2 minutes – all developed knee pain. Divers at 27 msw saturated in heliox given 37min at high pO_2 1.85 ata then ascent to air filled chamber at 9 msw followed by 6 hour hold then sat decompression to surface without any signs of decompression illness.

Additional examples of rapid decompressions have been identified by searching the literature;

Already in 1942, in the very early work with helium oxygen diving it was observed that by changing from helium-oxygen to air, and then to oxygen at the 60-foot level, it was possible to shorten decompression time. A saturation (6 hours) dive on helium at a wet or dry chamber depth of 150 feet required only 128 minutes decompression [13] .

Bühlmann (1975) describe some remarkable rapid decompressions, mostly by applying a switch of inert gas from helium to nitrogen [14]. From saturation (68-78 hours) at 23 ATA (220 msw) 7 decompressions (5 different subjects) were performed in 62-68 hours without any symptoms. The last 100 ft (4 ATA) were accomplished in only 10 hours after switching to 50 % nitrox. Vann (1982) [15] (p.364) explains the theoretical basis for the advantage by the gas switch and also the possible adverse inner ear effects if the switch is carried out too deep. Further explanations and description of the mechanisms for the benefits and problems of inert gas switching may be found in Hamilton and Thalmann (2003) [16] (p.474), Doolette and Mitchell (2003) [17] , Lever et al. (1971) [18] and Farmer (1993) [19] (p.289).

In other experiments in Zurich, volunteers were decompressed after 2 hours at 90 msw to surface with decompression times of 475 – 560 minutes [20].

It was discussed how it could be so large differences in the fourth Underwater Symposium in 1969 [21]. Selection was mentioned as a possibility.

Lin has reported theoretical arguments and animal experiments that he claims predict that large pressure reductions from saturation is safe also for humans [22, 23].

3.3.2 Drugs against oxygen toxicity

There is good scientific support to the assumption that decompression rate may be increased proportionally to the inspired pO_2. The hyperoxic side effects – mainly on CNS and lungs – limit the tolerance, and means to extend the hyperoxic tolerance could be expected to have a positive influence on the decompression rate. Symptoms of pulmonary oxygen toxicity include coughing, chest pain and dyspnea. From a clinical point of view, such are similar to lower airway inflammation and *could* potentially respond positive to corticosteroids – either locally or systemically applied. However, there is not sufficient evidence to conclude regarding the effect of corticosteroids on hyperoxic induced pulmonary injuries. The clinical use of this drug to treat hyperoxic lung injuries is controversial. We have identified two experimental studies on rats and mice [24, 25]. Methylprednisolone increased the toxicity of oxygen in adult mice [24]. Gross and Smith [25] suggested that the corticosteroid methylprednisolone may worsen the pulmonary effects of hyperoxia. At present, there is no other drug immediately available to alleviate symptoms of pulmonary oxygen toxicity except for analgetics (pain reliever) and cough syrup.

There have been suggestions for drugs against CNS toxicity also [26, 27, 25, 24, 28-38].

In the event that hyperoxic treatment gas is provided to a diver for treatment of DCS, a sedative like Diazepam or Midazolam should be available for reducing the risk of oxygen induced seizures. The drug may even be considered in the remote event that extreme hyperoxia in the breathing gas (>1.5 bar) is warranted.

3.3.3 Other ways to increase decompression speed

Animal experiments indicate that use of perfluorocarbons may have potential to increase the speed of inert gas offgassing [39-45].

As mentioned other places in this report switching inert gas from He to N_2 may be utilized to increase decompression speed, [13] , [16] , [17] , [46] , [47] and [48, 49] . But it has also been suggested that sequencing of other inert gases like Ar may be useful Bühlmann (1975) [14] . Lever et al. (1971) [18] discuss the potentials of N_2O, CF_4 and SF_6.

Research has also pointed to the possibility that if H_2 is used as inert gas, it can be metabolized [50] .

There is also documentation that indicate that an increased CO_2 level at 2 kPa [51] is beneficial before an ascent, but another study [52] points to the risk of potentiating oxygen toxicity in the presence of hypercarbia with pCO_2 of 5 kPa. A modest increase of pCO_2 to around 1 kPa, seems at least warranted [53].

3.3.4 Possible injuries, pathology

There is no doubt that decompression sickness may be fatal and it has been estimated that as many as 10 000 sponge divers died in the Aegean archipelagos and off the North African coast, in the period 1866-1915 [54].

In this century the situation fortunately has changed and Caruso (2003) [55] states “DCS rarely results in death but may cause permanent morbidity in divers.”

In Annex 7 it is relatively detailed described which medical problems that may be expected if accelerated decompression from heliox saturation is carried out.

It is concluded that:

If a controlled emergency decompression from heliox saturation is carried out, the most probable medical problems will be pain only (limb) bends.

Next to this may be vestibular problems with dizziness (vertigo) and hearing loss (deafness) as probable symptoms.

Life-threatening conditions is not to be expected.

3.4 Existing procedures for emergency decompression

There have been much effort to develop accelerated decompression procedures for submarine rescue operations based on air/ nitrox as breathing gas, see e.g. Dromsky et al. (2000) [56] and Reid et al. (2017) [57]. Here we focus on saturation diving with heliox as breathing gas.

The diving companies have available procedures for emergency decompression, but are reluctant to publish these as they are untested and closely connected to each company's normal procedures, equipment, training and organization. Some allow increased pO_2 of up to 90 kPa, even pure O_2 at 12 msw, with accompanying decompression speeds up to 45 msw/d.

But the Italian regulations (UNI 11366 Safety and Health in Hyperbaric and Commercial Diving Activities) contains emergency decompression procedures and these are included in annex 6. They are found in the original Italian language, but an attempt on English translation ("machine translation" with best choice adjustment by the author of this report - kindly and quickly checked and corrected by Dr. Constanino Balestra) is also shown.

It is noted that the Italian emergency procedures cover saturation depth till 180 msw. They start with an upward excursion, use pO_2 at 65 kPa and reduce the decompression rate from 3 msw/h to 0.5 m/h. Chamber gas is switched from heliox to air at arrival 18 msw - to take advantage of the "Bühlmann-effect" as explained in 3.6.

These procedures will bring divers from 180 msw to surface in slightly less than 4 days, which is somewhat quicker than what is recommended by one diving company. On the other hand these procedures do not take advantage of the fact that it should be safe to increase the pO_2, if the initial depth is shallower, since the exposure time and oxygen toxicity dose then get smaller, which is used by one company.

The use of initial upward excursion is probably wise when starting from saturation. However, if the saturation-decompression has been started a report indicate that this should be avoided [47].

A study utilizing a decompression model to predict bubble formation both in whole body and brain, indicate that reducing excursion ascent rate to 5 msw/min reduces formation of bubbles in the brain substantially [58], [59] . So if an upward excursion is to be performed it might be wise to use a reduced speed.

3.5 Risk factors related to the evacuation by lifeboat versus emergency decompression and other evacuation

3.5.1 General

The generic risk factors related to the evacuation by means of either a HRU/lifeboat or emergency decompression in the chamber followed by normobaric evacuation are described in the sections below.

Four documents were found to be useful in mapping these risk factors. They correspond to 1, 2, 5, and 7 in the list of items distributed for the workshop and listed under 2 METHODS. All these four sources list a number of risk factors and some that are considered most relevant in the case where hyperbaric evacuation is compared to emergency decompression, are found below.

IMCA D052 (2018) [2] is a thorough instruction on hyperbaric evacuation and includes a risk assessment guidance in chapter 9 (p.39-57). Naturally it does not elaborate on emergency decompression, but briefly mention it with a reference to DMAC-31 [3] as cited below.

Attempts/starting point for quantitative risk estimation is given at Willekes (2009) [6]. There is no mention of emergency decompression, on the contrary it seems as they do not consider this as a possible option to hyperbaric evacuation.

The Gulf of Mexico, US Diving Safety Work Group [8], also identify hazards connected to a hyperbaric evacuation, but not emergency decompression.

The DMAC recommendations [3] are reproduced in annex 3. As to be expected risk factors relevant for compared hyperbaric evacuation against emergency decompression are discussed.

3.5.2 Lifeboat evacuation

In IMCA D052 (2018) [2] it is stated (p.39):

"Special arrangements and procedures, which need to be risk assessed, should be in place, to evacuate the divers safely while keeping them under pressure in a purpose built HRU, capable of being removed from the worksite to a safe location while maintaining the divers at the correct pressure and with life support for a minimum of 72 hours [5]."

The guideline includes a risk assessment guidance of hyperbaric evacuation in chapter 9 (p.39-57). After an explanation and overview of the risk assessment process a table over 13 pages list the different activities of an evacuation (drills and exercises, launch, transit, speed/tow trials, manning, recovery, road transport/transfer, deployment and set up, hook up and operating trials, LSP operation, portable HRF deployment and setup, HRF-mating and operation, injury treatment, decompression and the event of inability to launch HRU – in the last two emergency decompression is mentioned) and identifies "subgroups", participants, considerations and requirements for each. After this an example of an HAZID form is given and a risk analysis matrix.

Attempts/starting point for quantitative risk estimation is given in Willekes (2009) [6]. They treated "*Initial risk assessment*" (transport and decompression) "*Completion of decompression*" ("*3rd party complex*" = HRF and "*by means of LSP*"), "*Methods of transport*" "*sailing to LSP*", towing, lifted on another vessel and "*connecting to SPHL*" and considered the following risks: lifeboat damage/sickness/injury due to sea state, failure of life support, lack of breathing gas, poor hygiene and possibility for additional injury to previously injured divers. The report is written up for a specific DSV's SPHL, but contain mostly aspects that have general character. It was

written before the first version of IMCA D052 was published in 2013. Part of the conclusion state: "*There is not one single preferred method of transporting the SPHL and decompressing the divers. In an actual emergency evacuation of the NHC, transport and decompression methods will need to be reevaluated.*"

The Gulf of Mexico, US Diving Safety Work Group [8], also identify hazards connected to a hyperbaric evacuation.

The main objective for the risk analyses cited above, are to minimize the risks when actually performing a hyperbaric evacuation. Nevertheless, the listed risk factors are also highly relevant when comparing the risks between evacuation and emergency decompression. The following factors are considered most relevant in the case where hyperbaric evacuation is compared to emergency decompression; competent and available crew in all necessary positions, injury to personnel, weather (sea state and wind), mechanical damage of crucial components essential for mating, launch, pressure integrity and life-support.

3.5.3 Emergency decompression and evacuation in another way

Although as stated above, the risk of a fatal outcome from an accelerated decompression from heliox saturation is assumed to be small, this must be regarded as very uncertain. Also the risk for serious or permanent health injuries is impossible to estimate.

The DMAC-31 recommendations [3] which is reproduced in annex 3, contain an introduction which in a good and concise way describe relevance of risk evaluation. Similar to what is said in the introduction to this report, the objective of the DMAC guidance "*is to reduce mortality and it is recognized that there may be a high risk of injury*". It is stated that risk is less when the storage depth is shallower and that recent excursions may increase risk. The guideline also points out that "*it will always be safer to reduce the rate of decompression (or stop and recompress), in the event that the emergency resolves, than to speed up the rate of decompression if the emergency scenario progresses more rapidly than anticipated*".

The only note on emergency decompression in IMCA D052 (2018) [2] is on p.4:
"Emergency decompression tables are available; however, that would still mean a number of days under those cramped conditions. Note: the Diving Medical Advisory Committee (DMAC) has published guidance DMAC 31 – Accelerated emergency decompression (AED) from saturation [3] , however the guidance states that it is not considered appropriate for use in an HRU. It is obvious that one of the main points of the LSP is to provide enough additional gas and consumables to ensure efficient management of decompression."

Our interpretation of this text is that *if* divers are committed to transfer in a HRU, then decompression should be planned according to the standard decompression procedure.

3.5.4 Relevant risks when weighing between the two methods

Evacuation by hyperbaric lifeboat will always be the first choice. If this possibility is practically unavailable the emergency decompression alternative certainly need to be regarded. It may be that HRU and/or mating/launch-systems are completely damaged or the status of the DSV is not compatible with hyperbaric evacuation. In such cases it is more a question of "the only possibility". But situations may be foreseen where the decision between the alternatives have to be taken on the basis of risk evaluation.

Again some good points may be found in the introduction to the DMAC recommendations in annex 3 [3]. A possible dilemma is described; the earlier a potential emergency decompression may be started, the safer it will be, but at an early state of the emergency events the uncertainty in predicting the alternative risks may be greatest.

Those most relevant for the weighing between use of lifeboat and emergency decompression are described in this table:

Factors in favor of emergency decompression	Factors in favor of lifeboat evacuation
Unfavorable weather: sea state, wind, temperature	Deep saturation depth
(Potential) equipment damage (lifeboat, flanges, davit etc.)	Recent large excursion
Lack of competent personnel for launch	Short time available
Lack of competent lifeboat crew	Support crew/personnel unavailable
Vessel listing/heading	Possible failure of function of decompression chamber(s) and/or support
Possibility to have a very short (ideally<10min) stay under normal pressure before recompression	Individual factors among divers (age, overweight, unfit, sickness or injury)
Injured divers	Potential DSV damage (fire, mechanical impact)

3.6 Implementation of emergency decompression with contingency measures

The bulleted list in the DMAC-recommendation [3] that is presented as a numbered list (for easier commenting) in annex 3, provides a good starting point for a practical plan from the initial phase to complete decompression.

Comments/deviation to the different points;

To point 4. Although it is mentioned in point 12 it may be worth pointing it out also under this point that administration of pure O_2 also after surfacing is recommended. Surface oxygen breathing has been shown to reduce the risk of DCS after caisson work as well as repetitive nitrogen-oxygen diving [16] .

From a theoretical point of view, sequencing of inert breathing gases is expected to decrease gas supersaturation during decompression as the slower N_2 is taken up slower than the fast He is offgassed. This has been studied in animal models eg. by Lillo et al. who studied this in a series of studies (1985-2000) [60] [61] [62] . They found by switching breathing gases between He, Ar and N_2 in experiments with more than 5000 rats differences between the gases in decompression risk (He<N_2<Ar) and exchange rate (He> Ar ≈N_2).This practice with switching from heliox to air to oxygen, during decompression was said by Dr. Behnke [63] to be routine

already “in the 30’s” (As stated above he had reported earlier that by changing from helium-oxygen to air, and then to oxygen at the 60-foot level, it was possible to shorten decompression time [13]). He also stated that “At no time were there any vestibular hits, nor did we observe the phenomenon of counterdiffusion”. In this session of this Sixth symposium on Underwater Physiology that was under discussion at least three of the presentations had described the gas-sequencing technique and none of the participants seems to be in any doubt of the beneficial effect of speeding up the off-gassing. But some state that by doing the switch to deep, vestibular problems may be provoked.

Already in 1942, in the very early work with helium-oxygen diving it was observed that by changing from helium-oxygen to air, and then to oxygen at the 60-foot level, it was possible to shorten decompression time. A saturation (6 hours) dive on helium at a wet or dry chamber depth of 150 feet required only 128 minutes decompression [13] .

The gas switching technique was applied during Predicitve studies IV in Pennsylvania [46] , in the Deep-Ex dives at NUI [47] and on the “3DP” (Statpipe) dives in 1983 [48, 49] .

Gas switch during decompression is commonly used in recreational “technical” diving. The trimix (or heliox) bottom gas is switched in sequence to air, nitrox and/or oxygen. The procedure is well established, is considered to increase decompression rate without increasing DCS incidence. However, there are scarce data available regarding formal scientific testing and we have not been able to identify operational safety records. Gas switch during decompression has been applied in “dry” chamber dives, but the results seem somewhat conflicting. While gas switch to air and nitrox during shallow depth seems to allow increased decompression rate, a too deep gas switch from heliox to air may cause vestibular DCS [64]. However a shallower shift seems safe and is likely to be beneficial for the outcome of the decompression. Greene et al (1978) [46] state that in Predictive studies IV in Pennsylvania “The transfer from a helium to an air environment shortened the decompression time significantly”. It should be considered to switch chamber gas to air at 18 msw or shallower.

To point 8. The potential benefits of creating a hyperhydration, should be weighed against the disadvantage of the practical/contamination problems that increased urine production may impose. Predive dehydration of swine has been shown to increase the risk for DCS [65] , while predive hydration has been shown to reduce venous gas embolism in man [66] .

To point 11. There is good reason to believe that additional medication could have a role, e.g. by increasing tolerance to oxygen toxicity and this should be further looked into.

4 CONCLUSIONS

Historical survey shows that need for hyperbaric evacuation / emergency decompression is not a very rare situation as a “probability-weighted” sum of such incidents was found to be 32. Lessons learned from these cases is that, not only should HRUs be mandatory, but that emergency decompression procedures, made as optimal as reasonable possible, should be available for all saturation facilities as a supplement to hyperbaric evacuation of divers in saturation. The recommendations in DMAC 31, with a few additions/modifications (eg. possible air flush) is a good start for making practical procedures.

If accelerated heliox saturation decompression speed is not too high, the most probable medical problems will be pain only (limb) bends. Next to this may be vestibular problems with dizziness (vertigo) and hearing loss (deafness) as probable symptoms. The chance of survival may thus be good, and even better if the saturation depth is relatively shallow.

5 ANNEXES

Annex 1 Incidents where evacuation and/or emergency decompression have or could have been initiated

Annex 2 “Emergency decompression from saturation – from the physiology perspective” by Andreas Møllerløkken

Annex 3 DMAC 31 Transcription with numbered list

Annex 4 Minutes from Workshop 2018-09-27

Annex 5 Hyperbaric Evacuation Process diagram (from TechnipFMC, origin from Sintef in 1990-ies)

Annex 6 Extract from Italian Regulation UNI 11366 “Safety and Health in Hyperbaric and Commercial Diving Activities”: “7.3.12 Decompressione di emergenza” and unofficial translation “Emergency decompression”

Annex 7 Possible injuries, pathology

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Annex 1. Incidents where evacuation and/or emergency decompression have or could have been initiated

A summary of known cases where divers have been evacuated or transferred to HRU (also where HRU was not launched), or events where the emergency decompression could have been applicable (e.g. fire on board the vessel) is shown in table over the two nexr pages. The data is mostly retrieved from [7] , [8], and a spreadsheet that has been compiled by Tim Chesshire and made available by an email from ADAS November 2019 (TC/ADAS2019), but also sources that include books, magazines, internet, reports, and personal communication (Leif-Tore Skjerven etc) as indicated. Incidents marked L(2018) indicates that information about the incident was retrieved from www.longstreath.com in 2018, but that it has not been possible to find it again.

It should be noted that not all information is equally accurate. Different sources in some cases give different information and evaluations have been necessary. *Italic types* are used to indicate especially great doubt about the correctness of the actual information. That information is omitted ("open box"), does not necessarily mean that the information is unavailable. This is somewhat random as the main purpose was all the time to identify the highest number of incidents with a reasonable degree of confidence.

Color coding is used for easier oversight. Green indicate that a transfer to HRU took place. Yellow means no transfer took place, light brown means that a bell-to-bell transfer was carried out or attempted, light blue means that vessel/chamber sunk at sea, and white means that information on transfer, if any, is lacking. Special color code is used in the column "*incident type*".

The content of the different columns is as follows.

ID
This is a unique identification of the incident given by the (estimated) year (2-3 digits) and one serial number (2 digits after the comma).

"Validity factor"
The numbers in this column represent a subjective attempt to weigh the probability for / the correctness of that the incident really happened. The incidents listed as ID 73.01 and ID 74.01 are e.g. both given the validity factor 0.5 to indicate that these quite certainly describes the same incident, but with the information available, it is not possible to decide which is most correct.

Start of incident: Date and year when the incident started is shown if it is known.

Transfer type
This should be self-explanatory and is determining the color as explained above

Indication of country or ocean is based on geographic position and not where the involved personnel or vessel, came from. Mostly two-letter (ISO 3166-1 alpha-2) country code is used. Exceptions are GOM which is "Gulf of Mexico" and XX, indicating position is unknown.

N.o. Divers

The assumed number of involved divers is shown, if it is known.

See 1.2 for explanation of abbreviations, eg. HRC, HRU and SPHL.

	ID	Inci-dent type	"Validity factor"	Start of Incident Date	year	Transfer type	Reason	Dive company or Client	Vessel or facility	Location	Country/ocean code	N.o div-ers	Comments	www.cargolaw.com/vc archive.php	www.longstreath.com	DSWG worksho	TC/ADAS 2019	Personal communication or other
1	0,01		0,5	?	?	UKOOA medical ch.	Medical / commercial		Apache	Peterhead harbor	UK		Road transfer to NHC		L			L(2018)
2	0,03		0,9	?	?	Unknown	Grounding	Rockwater	Semi II	Sandbank offshore	NL		Dr. Lambertsen was consulted about emergency decompression proceedures					Olav Hauso
3	73,01	Near-miss	0,5	?	1973	In bell	Hit gas pocket	Rank Diving	Japanese drillship	Offshore Warri	NG	2	Drillstring blown out. Bell picked up by crewchangevessel- later mated on DSV - heavy injuries, but decompr & survived				TC	Spreadsheet maintained by Tim Chesshire. Emailed from ADAS
4	74,01	Near-miss	0,5	*1974 or*	*1975*	In bell	Bubbling gas	*Comex?*	Discovery II Jap. drillship	Offshore Pointe-Noire	CG	2	Bell pulled onboard anch. handl. tug - emerg. decomp. - surface without phys. damage					Leif-Tore Skjerven, Olav Hauso
5	75,01	Near-miss	1,0	30/3	1975	No transfer	Oxygenfire in chamber room	Scan-Dive	Arctic Surveyor	Ekofisk/Emden pipeline	NO	4	No HLB, only HRC			D		Leif-Tore Skjerven, Eyolf Assersen, Gjerde & Ryggvik (2014)
6	81,01	Near-miss	1,0	24/11	1981	ToHRC	Weather Storm	Ocea-neering	Sedco PhillipsSS	Ekofisk	NO	10	6 divers in sat, 4 in decomp. pressurised to sat. depth. HRC disconnected from chambers. Left on deck till normalisation			D	TC	David Hutchinson, Bjørn Venn, Leif-Tore Skjerven
7	81,06	Near-miss	1,0	24/11	1981	prob. no transfer	Weather (storm)	Hamilton Brothers	Transworld 58	Argyll field	UK	2	adrift				TC	P. James in Subtech'84 David Hutchinson
8	81,02	Near-miss	0,2	1/11	1981	ToSPHL	Collision				NO		Vessel collision with Gas Platform in the North Sea, badly damaged			D		
9	81,03	Near-miss	1,0	9/1	1981	No transfer	Power blackout	Seaway	Seaway Falcon	Ekofisk	NO	12	DSV adrift. Risking collision with fixed installation. SPHL equalized with chamber pressure and prepared for divers transfer.			D?		Leif-Tore Skjerven, Arvid Bertelsen
10	81,05	Nearmi	0,6	?	1981	Unknown	leak	Comex	Seabex 1	Valhall	NO		During tie-in. "HRU-box" on deck					Olav Hauso
11	82,01	Fata-lity	1,0	15/2	1982	Sunk	Weather (storm)	Hydro-space	Ocean Ranger	Hibernia field, Newfoundland	CA	5+	no HRU, semirig sank, 84 died		L	D	TC	
12	82,02	Near-miss	0,9	*"mid-year"*	1983	No transfer	Fire	Wharton Williams	Stena Workhorse	Fulmar field	UK	2+	HLB damaged by fire, fire contained - no injury		L			Reports by Bernard Martin and Dave Hutchinson
13	83,01		0,2	?	*1983*	BellToBell		Stena		North Sea	*UK*		"HSE not very impressed"					L(2018)
14	83,02	Nearmi	0,2	?	*1983*	No transfer	Fire				GOM		No HRU, fire contained - no injury			D		
15	84,01	Nearmi	0,2	?	*1984*	No transfer	Fire				GOM		No HRU, fire contained - no injury			D		
16	84,02	Near-miss	0.9	*1984 or*	*1985*	BellToBell	Leaking vessel		Stena *Xxxxxxxx*	Offshore	BR		Vesselhull rift - leaking. Divers rescued by Seacom					Olav Hauso
17	85,01		1,0	22/6	1985	ToSPHL	Grounding		Wildrake	Marsteinboen	NO	6	SPHL disconnected from chamber. Kept over side till vessel alongside at CCB.			D		Bjørn Venn, Leif-Tore Skjerven, Sjur Lothe
18	85,02	Fata-lity	1,0	15/12	1985	Sunk	Weather	Protexa	Huichol II (ex Kattenturm)	off Carmen del Cuidad	GOM	4	Not possible to use HRU		L	D	TC	L(2018)
19	85,03	Near-miss	1,0	10/2	1985	BellToBell	Blow out (no fire)	Comex	PGP-1 platform	Garoupa field, Campos basin	BR	4	Gas blow out. Divers rescued by Stena Workhorse		L	D	TC	Leif-Tore Skjerven
20	86,01	Fatalit	0,2	Feb.	1986	Sunk	Weather (storm)		Semi drilling rig		XX	4	no HRU			D		

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	ID	Inci-dent type	"Validity factor"	Start of Incident Date	year	Transfer type	Reason	Dive company or Client	Vessel or facility	Location	Country/ocean code	N.o div-ers	Comments	www.cargolaw.com/vc archive.php	www.longstreath.com	DSWG worksho	TC/ADAS 2019	Personal communication or other
21	86,02		1,0	4/3	1986	ToSPHL	Grounding	Royal Navy	Seaforth Clansman	Kyle Lochalsh, Scotland	UK	8	SPHL launced, retransferred 2 times, finally decompressed in Clansman				TC	L(2018)
22	87,02	Near-miss	1,0	26/12	1987	BellToBell & ToSPHL	Fire, port engine	Ocea-neering	Regalia	Gullfaks	NO	10	5/6 thrusters out, drifted off. 2 divers in habitat at seabed - bellToBell transfer to Stadive. 8 divers transferred to SPHL briefly - back & decompr. in chambers.			D		Bjørn Venn, Leif-Tore Skjerven
23	89,01		1,0	2/6	1989	ToHLB	Grounding	Seaway	Seaway Pelican	Tungenes (Trans. from Stavanger)	NO	6	Proceed toTommeliten after hull inspection					Bjørn Venn, Leif-Tore Skjerven
24	89,02	Fata-lity	1,0	4/11	1989	Sunk	Weather (ty-phoon Gay)		Seacrest (Scan Queen?)	Gulf of Siam, South China sea	TH	5	no HRU, drillship capsized, 91 (incl. diveteam?) perish		L	D	TC	
25	89,03	Near-miss	0,1	?	1989	BellToBell	Blowout, Fire?	Petrobras	PGP-1 platform	Garoupa field, Campos basin	BR	4	Gas blow out. Probably confused with ID85.03		L	D	TC	
26	90,01	Near-miss	1,0	Feb.	1990	No transfer	Weater (storm)	Seaway	Norskald	Statfjord	NO	16	28 m seas. Sat control flooded. SPHL equalized - prepared for transfer					Leif-Tore Skjerven, Arvid Bertelsen
27	91,01		1,0	15/5	1991	ToSPHL	Fire	*Rockwater*	Semi II	Ekofisk	NO						TC	Bjørn Venn
28	91,02	Fata-lity	1,0	15/8	1991	Sunk	Weather (ty-phoon Fred)	McDermott	DB29	South China sea	TH	4	no HRU. Emergency decomp. with high PO_2 to 60'. 22 (incl. 4 drowned divers) died		L	D	TC	
29	94,01		1,0	July	1994	No transfer	Fire incine-rator room	Seaway	Amethyst	Transit Statfjord - 37/11 Ekofisk	NO	16	SPHL equalized - prepared for transfer, after fire extinguished				TC	Leif-Tore Skjerven, Arvid Bertelsen
30	97,01	Near-miss	1,0	1/7	1997	No transfer	Crane collaps	Total	DLB Kurishio1	Bongkot field	TH	9	HRC ripped off		L		TC	
31	105,01	Near-miss	1,0	1/4	2005	BellToBell	Crane collaps		DLB Regina		IN	2	Surface system damaged, bell down, wet transf. to DSV Samudra Sevak		L		TC	
32	105,02	Near-miss	1,0	25/7	2005	No transfer	Fire		M/VSamudra Suraksha	Mumbai High North	IN	6	SPHL in fire. Divers decompressed, initially by themselves	c	L		TC	
33	105,03		0,1	1/7	2005	BellToBell					IN		Two transfers		*L*			L(2018)
34	107,01	Near-miss	0,9	14/8	2007	No transfer	Collision	Saipem	DSV Bar Protector	Dunbar Platform	UK		DSV crane collision with platform		L	D	TC	
35	107,02	Nearmi	1,0	18/8	2007	No transfer	DP-incident	Technip	DSV Orelia	Tartan A	UK		DP software bug. Stopped 10m from platform.		L		TC	Sjur Lothe
36	108,01	Near-miss	1,0	12/11	2008	BellToBell	Grounding		Sea Lion	off Fourchon	GOM	4	No HRU. Transfer bell by crane to Global Pioneer		L	D	TC	
37	111,01	Near-miss	1,0	30/3	2011	No transfer	Medical accident	Technip	DSV Oceania Venturera	offshore Broome	AU	1	HP water jet failure, severe left forearm wounds. Emergency decomp, medivac, off work 6 months.		L		TC	Ref. *Western Australian* , Sjur Lothe
38	111,04	Nearm	1,0	8/4	2011	No transfer	Grounding	Bibby Offsh.	Bibby Topaz	Lerwick harbour	UK	12	DSV refloat by own power. No damage/injury	c				*The Scotsman*
39	111,02	Near-miss	1,0	6/9	2011	No transfer	Collision	SS7	Acergy Discovery	Deep Panuke gas platform	CA		DP-fault. Collided with the Deep Panuke gas platform, hitting a leg and the riser caisson, no injuries	c	L	D	TC	*Professional Mariner journal*
40	111,05	Near-miss	0,7	3/9	2011	No transfer	Collision	SS7	Seven Atlantic	Dutch sector	NL	?	DSV collided with the Gaz de France platform, nobody hurt	c				https://www.vesseltracker.com/en/Ships/Seven-Atlantic-9419125.html
41	111,03	Fata-lity	1,0	20/10	2011	Sunk	Weather	Adsun ODC	DSV Koosha	South Pars, off Lavan island	IR	6	Total 13 lost, 4-man HRC	c	L		TC	
			32															

Annex 2. “Emergency decompression from saturation – from the physiological perspective” by Andreas Møllerløkken

INTRODUCTION

The following chapter is a result of an identified need within the overall perspective given in the request from the Norwegian Petroleum Safety Authorities regarding emergency decompression from saturation, to include a section related to the various physiological challenges in such special situations, and with special emphasis on accelerated decompression from saturation. The chapter is further limited to situations where no other alternative means of evacuation from the hyperbaric system at risk is possible.

The need to perform an accelerated decompression from saturation in an emergency situation, is still the most feared situation for both a saturation diver and the operating team supporting the diving operation. We strive to keep all potential dangers to a minimum.

The Diving Medical Advisory Committee (DMAC) has published two guides with relation to accelerated decompression from commercial saturation diving. The oldest one is from a workshop arranged in 2011 [1], but the guide were published in 2013, entitled “*Accelerated emergency decompression from saturation in commercial diving operations*”. The second guide is DMAC 31 – “*Accelerated emergency decompression (AED) from saturation*, also published in 2013 [2]. In the latest guide, it is highlighted that:

“It is important to recognize that accelerated emergency decompression is a last resort when alternative means of evacuation from the hyperbaric system at risk is not possible. The knowledge underlying the guidance is limited and the objective of this guidance is to reduce mortality and it is recognized that there may be a high risk of injury.”

In addition, Brubakk, Ross and Thom published in 2014 a review article on saturation diving, where they commented on emergency decompression from saturation, stating that new procedures for such decompressions are needed [3]. The aim of such procedures should be to prevent serious damage or death of the diver, but still get the diver to the surface, were then final decompression can be initiated.

"*there are no method available today that can reduce the decompression to hours instead of days without significant injury*" [3]. This chapter on emergency decompression from saturation highlights some of the challenges that needs to be discussed and solved in order to bring new knowledge to the table, if one wants to solve this challenge in the future.

BACKGROUND

The principles of saturation diving is well recognized in the professional diving community. When the duration of an exposure approximates the time that is required to saturate the poorly perfused tissues with inert gas, the decompression back to normal pressure will be the same regardless of the duration of the exposure [4].

Regarding decompression from saturation, the principles are the same regardless of which inert gas is used. It is the tissues with the poorest gas exchange that at start of a decompression will have the highest partial pressure of the specific inert gas. And it is the slowest tissue(s) and processes affecting bubble formation in these tissues that determine the ascent rate to surface. This is the reason why computations of decompression from saturation is based on estimates of gas exchange in a single, hypothetical tissue compartment [4] [5] .

When selecting decompression patterns, there are various factors that needs to be considered.

- The inert gas itself. Nitrogen is thought to have a half-time for the slowest exchanging tissues to be at least 480 minutes. Helium at least 240 minutes. And it is reported of situations where the gas exchange can be even slower [4] [6] [5] .
- The depth (pressure). At high ambient pressures the tendency for supersaturation with a particular inert gas to nurture bubble formation is less compared with shallower depths. Hence, degree of supersaturation allowed in slowest tissues is greater at the beginning of decompression at great depths compared with the final steps in shallower waters [4] [6].
- Increased oxygen pressure. Lowering inspired inert gas pressure by increasing oxygen pressure improves the gradient for inert gas from tissues to blood and lungs. Limits are the oxygen partial pressure and duration of exposure to avoid oxygen toxicity [6], [3].
- Substitute inert gas. Switching inert gas can lower total inert gas pressure in the slowest exchanging tissues. Nitrogen for helium or argon for nitrogen [4] [6].

- Isobaric bubble formation. When a less diffusible gas is breathed while a more diffusible gas leaves the body, gas gradients are built up through the skin that lead to cutaneous bubble formation.

There are traditionally two ways of bringing divers back from saturation in an emergency – either linearly or through an initial upward excursion followed by continued decompression. The linear decompression will theoretically keep the gradient for bubble formation at a minimum through Boyle's law, whereas an initial jump towards shallower water potentially can get the divers closer to a better or safer way of escaping from the situation. Either through mobilization of other rescue means (other diving vessels) or that the situation changes to the better for the diver, meaning that the situation brings forward possible solutions within a better time-scale. For instance, that the situation changes and/or stabilizes so that one gets more time to perform a controlled decompression.

Review of the majority of different aspects related to decompression following conventional dives, both theoretically and practically, are reviewed elsewhere. But, most tables has to a large degree been validated through subjective interpretations by the participating divers [7]. One exception is the application of Doppler [8].

In saturation diving, subjective symptoms have also modified theory and thus influenced early decompression calculations [9]. Decompression from the storage or living depth is not fast (with or without overnight stops) and the rate varies between authorities but averages some 30m per day.

With special emphasis on accelerated decompression from saturation, I would like to present four experimental setups to highlight possibilities from basic research that can be exploited for further basic understanding of the mechanisms involved in the decompression process.

EXAMPLE 1 – FROM [10]

Following a saturation dive using air, three different decompression profiles were tested using pigs as experimental animals. The first decompression followed a USN staged decompression profile. The second decompression profile was a linear decompression with the same total decompression time as the tested USN profile. And the third profile were tested where the total

decompression time were reduced with 50%. The study demonstrated that the fastest linear profile produced the least amount of bubbles of these three. It demonstrated that the controlling tissues changes when the fastest linear profile were tested. These profiles were combined with an additional surface decompression using oxygen, but it also highlighted that it is possible that the reduced time spent on decompressing from depth, changed the amount of gas loaded in the tissues.

EXAMPLE 2 – FROM [11]

Using pigs as experimental animals, it has been demonstrated that recompression during decompression significantly reduced the amount of detectable vascular bubbles following an experimental dive to 500 kPa for 90 minutes.

The results in these experiments was similar to what Gernhardt has suggested regarding intermittent recompression during decompression from saturation and the effect on decompression stress [12]. As was pointed out at that time, the results would benefit from being seen as a scientific approach to pinpoint new knowledge within decompression from both saturation- and nonsaturation dives. But the result from this experiment is not possible to explain using a Haldanean supersaturation model – it highlights the possibility that a bubble model of decompression is more correct.

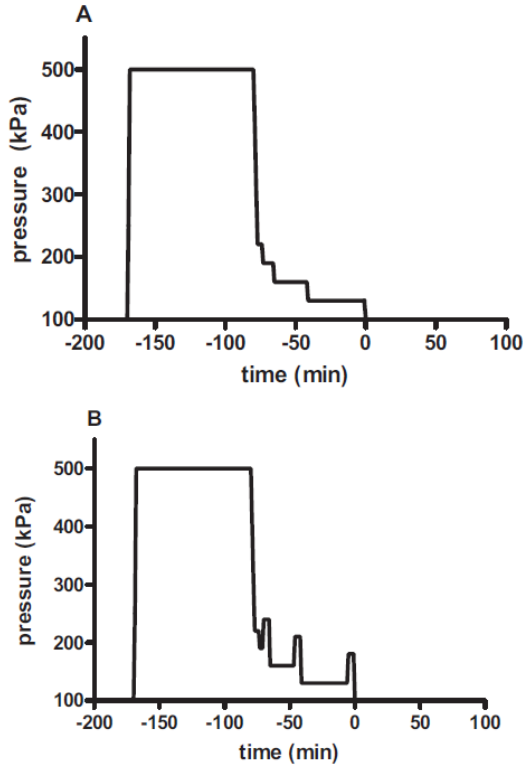

Fig. 1. Pressure profiles for both A) the control and B) the experimental group. Time = 0 is at surface after decompression.

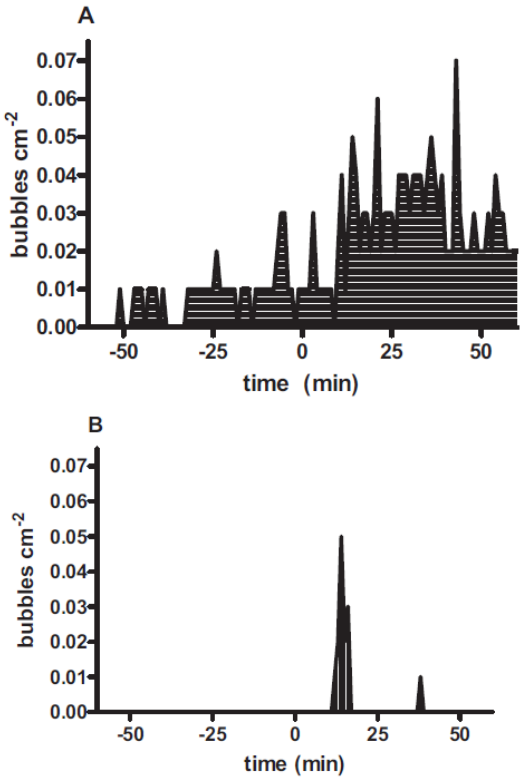

Fig. 2. The mean amount of bubbles detected in the pulmonary artery from start of decompression in both A) the control and B) the experimental group. Time = 0 is at surface after decompression. In both groups, n = 6.

Figure 1 & 2 from [11]

EXAMPLE 3 – FROM [13]

Once again, experimental work using pig as the experimental animal. The idea was to investigate the effect of a nitric oxide (NO) donor on decompression from saturation, as a result of the promising findings related to the important effects of NO with regards to limiting the amount of vascular gas bubbles detected following non-saturation dives in both experimental animals and in men. The experiment investigated whether one could affect the amount of vascular bubbles following a saturation exposure.

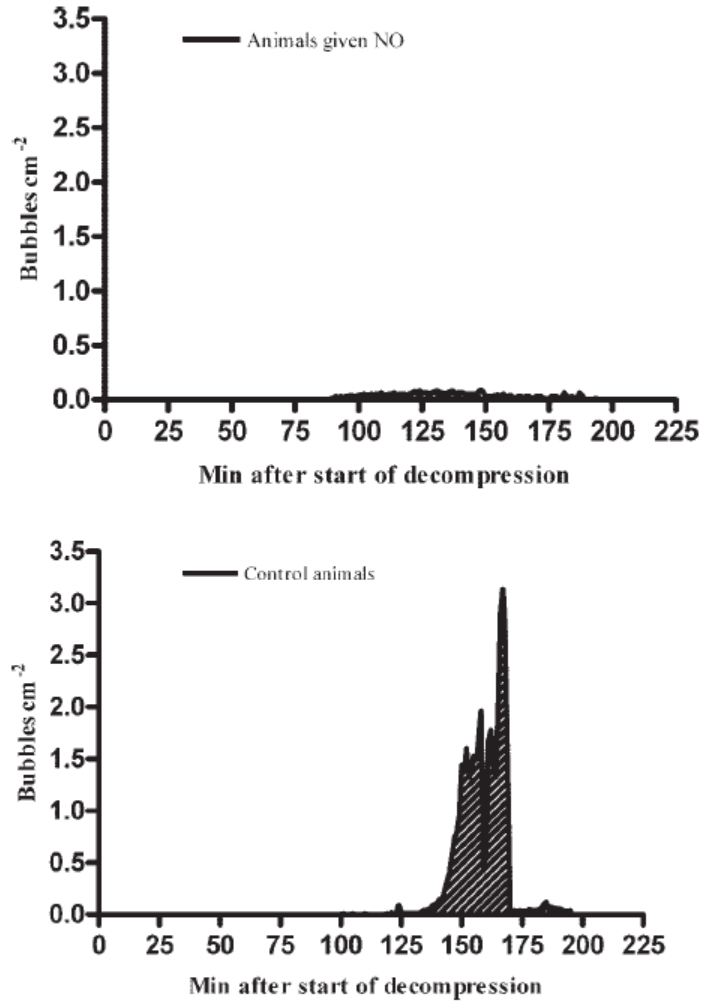

Fig. 2. Amount of vascular gas bubbles during and after decompression in animals receiving nitrate and the control group. By calculating the area under the curve, we get the amount of gas that dissolves during decompression and after surfacing in the blood. Area = 4.21 bubbles·cm^{-2}·min^{-1} in the experimental group. Area = 39.59 bubbles·cm^{-2}·min^{-1} in the control group.

Figure from [13]

EXAMPLE 4 (RECENT RESULTS AND NOT YET PUBLISHED)

The effect of perfluorocarbons (PFCs) and the possibility of using PFCs in increasing the decompression rates / lowering the decompression times has long been discussed in the baromedical world, but the nature of the previous PFCs have not been ideal for further development based on toxic effects of accumulation of the PFCs in the organism. But these days, a new albumin-based fluorocarbon have been developed, which once again have made this an attractive candidate for further testing in regards to increasing decompression safety. And in a small animal model, we performed initial testing of this component in order to investigate whether it could reduce any adverse effects of decompression from a dive. The results is not published, but the experiment demonstrated that it would be possible to investigate this hypothesis further.

These four examples highlights the possibilities of studying saturation decompression scenarios in animal models, using both large- and small animals as the choice of experimental animals. In addition, controlled studies, which includes such experiments, is essential if one want to study accelerated decompression from saturation.

All of the above-mentioned examples are with use of experimental animals. For the very special situation we are highlighting here, there is still a need to develop our understanding through translational research. It is premature to suggest testing on humans, and we need more understanding of the various physiological reactions taking place before we can start model the different reactions.

The understanding of saturation diving from a physiological point of view is not new knowledge, and neither is different suggestions related to how to accelerate decompression from saturation. However, there is a mismatch in the developed understanding and the following testing of hypothesis with laboratory experiments. The existing guidelines do state some general advice, but at the same time, the guidelines do point on the necessity to keep researching and testing the guidelines.

THE RED PILL OR THE BLUE PILL?

In 2012 professor Alf Brubakk gave a status on where the knowledge within diving was at that time, with emphasis on whether new acquired knowledge had influenced or had the potential to influence procedures to increase the overall safety of the divers [14]. Following a review on different decompression models, one part of the report is on emergency decompression from

saturation. Professor Brubakk highlights that at that time there was no procedures available for bringing divers back to normal pressures within hours compared with the standard procedures, which will bring the divers back over several days of decompression. Since 2012, no major steps has been taken in order to bring potential procedures from ideas to testing.

An emergency abort procedure has been developed by the U.S Navy, but it is stressed that this procedure has received very little testing. The procedure will allow the divers to surface earlier than normally allowed by the decompression table, but when looking at the actual time "saved" in the emergency procedure, the obvious question to ask is if it is worth the risk. From a saturation dive to 400 fsw (approximately 122 msw), the emergency procedure will demand a total of 93.4 hours of decompression. The standard saturation decompression procedure would demand a total decompression of 99 hours [15].

I will highlight, as professor Brubakk did, the need for new thinking. New ideas should be tested and evaluated further in order to prepare one selves more in case of an emergency. But before we discuss potential new ideas, we need to state if this is needed or not. If there is a need for a better understanding of- and better procedures to bring divers back to normal pressure from saturation in case of an emergency, then the focus should be towards *how* to move forward.

One area where further research is needed, is on the role of arterial bubbles in decompression. In 1980, Doppler examination of divers following excursions documented gas bubbles in *A. Carotis* for as long as 23 hours following the excursion. None of the divers had any acute symptoms of DCS [16]. Arterial bubbles can arise from pulmonary barotrauma which, if severe, can lead to cerebral gas embolism with immediate, characteristic and potentially serious neurological manifestations. It follows that lesser degrees of arterial gas embolism from this cause may remain 'silent'. Other arterial bubbles may arise from relatively benign venous bubbles of decompression that, instead of their gas being excreted on reaching the lungs, manage to slip through the pulmonary filter and left side of the heart into the arterial circulation. These too may be clinically 'silent' [9]. In 69 no-stop and symptom-free dives well within the safety limits of the UK Health and Safety Executive (having a $p\sqrt{t}$ exposure below 25) and the Norwegian air tables, arterialization of bubbles occurred after 11 dives (of the 12 divers one was found to have a PFO) [17]. If this study is confirmed in a wider population of divers and, should such bubbles indeed persist briefly in de-gassed arterial blood, this observation would reflect not specifically on saturation diving but upon all diving. The earlier report of arterial bubbles detected during an upward excursion from 300m to 250m [16], they document arterial bubbles that are present for

23 hours following the excursion. Both this report and the article from the air dives warrant a follow up. If the findings are independently confirmed, studies should be initiated to investigate the integrity of the pulmonary filter. Such a study would hence put the spotlight on the safety of all diving, not only related to saturation.

The majority of the efforts that has been made throughout the 1980´s and 1990´s in Norway within diving-related research has never been published in the open literature. It would be a major contribution if the reports could be collected, de-classified to open accessible literature and then be put into some sort of publication. The scientific approach to findings and conclusions should always be the selected procedure.

Brubakk, Ross and Thom highlighted in 2014 that there are no procedures today that will allow reduction of required decompressions from saturation from days to hours, should the divers be forced to evacuate rapidly from saturation [3]. The authors point to the fact that the DMAC guidelines give notes on how to shorten the decompression time without significant risk of serious injury. And they comment that "*there is a need for a new type of procedures were the focus must be on preventing serious damage, especially damage to the CNS, or death of the diver. Prevention of pain and discomfort during or after decompression is not the aim of such a procedure. This will not be a final treatment but procedures that can bring the diver to the surface where the final decompression may be initiated after a shorter or longer time".*

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Annex 3. DMAC 31 Transcription with numbered list

Accelerated Emergency Decompression (AED) from Saturation

Contingency planning for managing a situation in which the safety of a diving support vessel (DSV) is at risk normally involves hyperbaric evacuation using a hyperbaric rescue system (HRS). However, the circumstances of the emergency may put the hyperbaric evacuation system (HES) out of action, the sea state may prohibit launch of the HES or reception facilities for an evacuation system may not be available. In any of these situations an emergency decompression from saturation may offer the best opportunity for the divers' survival. Although many diving manuals contain emergency rapid decompression procedures, in many situations these are too slow to be of value, and decompression over the estimated time available in the emergency may be the only option.

This guidance note is based on the conclusions of a workshop set up to consider the issues involved in rapid decompression from saturation. It is important to recognise that accelerated emergency decompression is a last resort when alternative means of evacuation from the hyperbaric system at risk is not possible. The knowledge underlying the guidance is limited and the objective of this guidance is to reduce mortality and it is recognised that there may be a high risk of injury.

The following conclusions may be helpful in the management of such an emergency.

A risk evaluation exercise should be conducted in any circumstance in which the safety of divers in a decompression chamber system is put at risk as a result of fire or mechanical damage to the vessel or chamber system, which may result in loss of the vessel (sinking) or inability to provide continued support to the divers under pressure. Such circumstances have the potential to result in multiple fatalities amongst the divers.

The chances of an emergency situation resulting in fatalities may range from a possibility to an absolute certainty. Both level of risk and the timescale of progression of an emergency situation are difficult to assess but prediction of the outcome is likely to be more accurate as time progresses.

Actions to remove the divers to safety need to be considered at the earliest stage possible.

Two possible actions may be available. These are evacuation using a hyperbaric rescue vessel (HRV) or emergency decompression. Both carry risks of illness, injury and even a fatal outcome for the divers depending on conditions.

It has proved possible to evacuate divers using a hyperbaric lifeboat in calm seas when a vessel was at risk and to return the divers to the same vessel when the emergency had been resolved. Conversely, evacuating divers into an HRV in rough sea when there is no facility for recovery within 48 hours is likely to carry a risk of fatality.

Emergency decompression will carry a relatively lower risk when storage depth is shallow, divers have made no recent excursions (i.e. within 24 hours) and when there is a longer time window of opportunity in which to conduct the decompression.

The safest evacuation procedures are likely to be available early in the development of the emergency when the final outcome of the emergency may be most difficult to predict.

In using an accelerated decompression it will always be safer to reduce the rate of decompression (or stop and recompress), in the event that the emergency resolves, than to speed up the rate of decompression if the emergency scenario progresses more rapidly than anticipated.

This guidance is not considered appropriate for use in a hyperbaric rescue unit (HRU).

Chamber decompression issues:

1. Where rapid decompression in a chamber facility is considered, a risk evaluation exercise is required to assess the threat to the divers of remaining in the chamber compared to the risks associated with a rapid decompression, taking into account the storage depth (see below).
2. The decompression should be planned to take place at the slowest rate consistent with a safe evaluation of the emergency timescale.
3. In planning a rapid decompression the selection of either a linear decompression or commencing with an upward excursion (1 msw per minute) should take into account the divers' recent excursion dive (pressure profile) exposure.
4. During the decompression a high pO_2 in the divers' breathing gas is advantageous. The level of pO_2 selected will depend on anticipated duration of exposure. At deeper depths, the chamber pO_2 could be raised to 1.0- 1.5 ata. Use of a built-in breathing system (BIBS) would be required for higher pO_2 mixtures and at shallow depths.
5. Decompression rates as fast as 10-20 msw per hour using a high pO_2 may be possible with divers who have not done any excursion in the previous 24 hours.
6. Breathing a high pO_2 gas mixture before starting decompression may be helpful if the opportunity exists without reducing total time available for decompression.
7. All attempts should be made to obtain assistance from another dive vessel with chamber facilities for the recompression of divers completing decompression at the earliest available opportunity.
8. Maintaining adequate hydration is considered important. This will require an adequate oral fluid intake. Some advocate the administration of higher volumes of fluid by mouth or by intravenous route if practical. The volumes taken or administered will be dependent on the duration of the decompression, but oral intakes as high as 1 litre per hour might be reasonable during a short decompression. For oral hydration water or oral rehydration mixture should be locked into the chamber shortly before use.
9. Thermal control of the chamber should be maintained. If environmental control is compromised, this may increase the risk of the procedure.
10. Where practical, divers should be encouraged to move around but not undertake vigorous exertion during the decompression.
11. There is no human evidence that any drug would offer benefits but analgesia may be valuable. Glyceryl trinitrate, non-steroidal anti-inflammatory agents and clopidogrel may all offer some advantage in protection against decompression illness and are unlikely to increase the risk.
12. A plan for the management of complications arising during and after the decompression should include access to analgesia and antiemetics, the availability of continued surface

oxygen therapy after completion of decompression and access to recompression elsewhere for treatment of decompression illness.

Annex 4. Minutes from Workshop 2018-09-27

Møtested: Ptil (Besøksadresse: Professor Olav Hanssens vei 10, Stavanger), møterom Kollsnes

Dato: 2018-09-27 Tid: 10-15

Deltakere:

Morten Hellang, Fylkesmannen i Rogaland
Einar Thorsen, UiB/HUS
Marit Skogstad, STAMI
Olav Eftedal, Equinor
Bård Humborstad, Repsol
Stein Modahl, Technip FMC
Jan Risberg, NUI
Øyvind Lønnechen, Technip FMC
John Arne Ask, PTIL
Olav Hauso, PTIL
Bjarne Sandvik, PTIL
Kåre Segadal, NUI
Andreas Møllerløkken, NUI

Også invitert:

Roman Benz, Fylkesmannen i Rogaland
John Hjelle, Subsea7
Joar Gangenes, Subsea 7
Leif Morten Rasch, IndustriEnergi
Halvor Erikstein, SAFE
Henrik Phillipsen, Technip FMC
Dag Atle Ask, Subsea 7

Saker/Agenda

1- Velkommen, innledning ved Olav Hauso. 2- Gjennomgang av utsendt Bakgrunnsstoff. 3- Presentasjon ved Jan Risberg. 4-Oppsummering, aksjoner

Bakgrunnstoff var sendt ut med epost på forhånd (2018-09-25)

Referat

1. Velkommen, innledning ved Olav Hauso

Olav ønsket velkommen og forklarte om bakgrunn og motivasjon for prosjektet. Det essensielle er at det aldri kan utelukkes situasjoner hvor akselerert/nød-dekompresjon kan være et alternativ eller supplement til evakuering i hyperbar livbåt og at det er viktig å samle kunnskap i beredskapssammenheng.

Det var innledningsvis en diskusjon om det var fare for at aksel erte dekompresjonsprosedyrer, eksklusivt beregnet for nødsituasjoner ved misforståelse kunne misbrukes og om dette var et argument for å ikke lage rapport på engelsk. Etter grundig vurdering ble det enighet om at det var viktigere at informasjonen ble tilgjengelig for flere enn norsktalende og at rapporten lages på engelsk.

2. Gjennomgang av utsendt Bakgrunnsstoff

Kåre/Andreas – powerpointpresentasjoner vedlagt

Det ble lagt vekt på å få innspill til om de identifiserte referanser var dekkende og dette var det enighet om. Men det ble etterlyst informasjon om veldig hurtige dekompresjoner fra «gamle dager» og Kåre skal prøve å fremskaffe dette.

Ellers ble det utsendte bakgrunnsstoff gjennomgått punkt for punkt med diskusjon og innspill fortløpende. Det var enighet om at anbefalinger gitt i DMAC 013 var bra utgangspunkt, men at justeringer kan gjøres.

Det ble forespurt om dykke-entreprenørene kan stille til rådighet sine nødprosedyrer. Øyvind skulle følge opp, men gav uttrykk for at dette ikke var uproblematisk

3. Presentasjon ved Jan Risberg

Jan - powerpointpresentasjon (vedlagt) gjennomgått
Jan hadde etterspurt informasjon fra Dr. Gerth i US Navy og venter på svar. Det ble diskusjon om i hvilke medikamenter som kunne være aktuelle og Jan ville undersøke om han kunne skaffe frem informasjon om dette

4. Oppsummering, aksjoner

Aksjon: Kåre/Andreas skriver draftrapport med kommentarer og innspill inkludert, for distribusjon og til kommentarer

Aksjon: Øyvind a) undersøker om TechnipFMCs nøddekompresjonsprosedyrer kan gjøres tilgjengelig b) leter opp gammelt flytdiagram fra SINTEF c) finner kapittel om nøddekompresjon i Italiesk regelverk

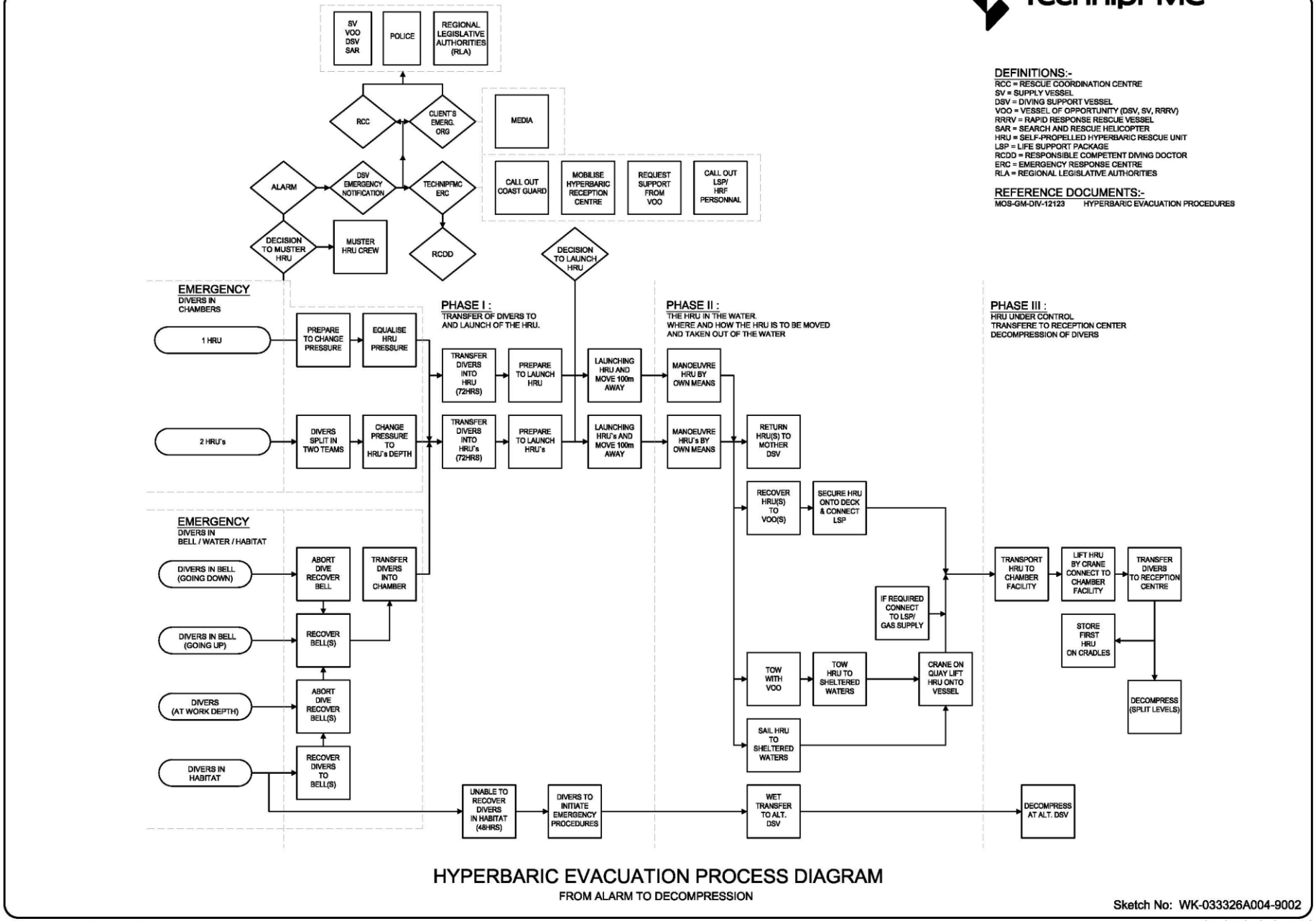
Aksjon: Jan gir innspill om aktuelle medikamenter, følger opp svar fra Dr. Gerth

Bergen 2018-11-30

Kåre Segadal

referent

Annex 5. Hyperbaric Evacuation Process diagram (from TechnipFMC, origin from Sintef in 1990-ies)



HYPERBARIC EVACUATION PROCESS DIAGRAM

FROM ALARM TO DECOMPRESSION

Sketch No: WK-033326A004-9002

Produced By TechnipFMC ~ UK Drawing Office

(13/06/18) (MIR)

Annex 6. Extract from Italian Regulation UNI 11366

“Safety and Health in Hyperbaric and Commercial Diving Activities”

“7.3.12 Decompressione di emergenza”

and unofficial translation

“Emergency decompression”

7.3.12 **Decompressione di emergenza**

In caso di necessità/urgenza di decomprimere uno o più sommozzatori in stato di saturazione (per motivi medici o altro) il supervisore alla saturazione deve valutare attentamente i rischi associati a una decompressione accelerata. Se non esiste un pericolo di vita effettivo per il personale, si deve effettuare una normale decompressione omettendo solamente le soste notturne. In caso di emergenza estrema (previa autorizzazione del capocantiere subacqueo) il supervisore alla saturazione deve applicare la seguente procedura:

a) se l'emergenza coinvolge solamente una parte della squadra in saturazione e l'impianto è composto da almeno 2 camere iperbariche indipendenti, applicare la decompressione accelerata al minimo degli operatori possibile;

b) se l'emergenza coinvolge un solo sommozzatore, comprimere un operatore esterno nella camera iperbarica che sarà utilizzata per la decompressione accelerata, in modo da non avere mai un solo operatore in camera;

c) decomprimere la camera per l'escursione prevista ad una velocità di 0,75 m/min;

d) durante l'escursione aumentare, e mantenere fino alla quota di 18 m, la pressione parziale di ossigeno in camera a 650 mbar;

e) iniziare la decompressione applicando le seguenti velocità in base alla quota di fine escursione:

- da 180 m a 90 m velocità 3 m/h,
- da 90 m a 30 m velocità 2,4 m/h,
- da 30 m a 18 m velocità 1,2 m/h,
- da 18 m a 0 m velocità 0,6 m/h;

f) a 18 m effettuare il lavaggio ad aria;

g) non superare mai il 23,5% di ossigeno in camera.

Nota Valutare l'effettivo vantaggio in termini di tempo della decompressione accelerata rispetto a quella normale prima di iniziare la procedura.

7.3.12 Emergency decompression

In case of need/urge to decompress one or more divers in saturation (for medical reasons or otherwise) the dive supervisor should carefully evaluate the risks associated with an accelerated decompression. If there is no danger to the staff, you should perform a normal decompression omitting only stops at night. In case of extreme emergency (with the permission of superintendent) the life support supervisor needs to implement the following steps:

a) should emergency involve only a part of the team in saturation and the system consists of at least 2 independent hyperbaric chambers, a minimum number of divers should do accelerated decompression;

b) if the emergency involves a single diver, compress an external operator in a hyperbaric chamber that will be used for accelerated decompression, so you never have a single diver in the chamber;

c) decompress the chamber for the planned excursion at a speed of 0.75 m/min;

d) during the excursion increase, and maintain up to 18 m, the partial pressure of oxygen in the chamber to 650 mbar;

e) start decompression by adopting the following speed depending on the depth of the end of the excursion:

from 180m to 90 m	speed 3 m/h,
from 90 m to 30 m	speed 2.4 m/h,
from 30 m to 18 m	speed 1,2 m/h,
from 18 m to 0 m	speed 0,6 m/h;

f) from 18 m	air flushing;

g) never exceed 23.5% oxygen in the chamber.

NOTE evaluate the actual benefit in terms of accelerated decompression time compared to normal before beginning the procedure.

Annex 7. Possible injuries, pathology after accelerated heliox saturation decompression

INTRODUCTION

In this annex we try to predict which medical problems, symptoms and pathological conditions that may be expected after accelerated decompression from heliox saturation in connection with possible emergency situation.

The documents that have been reviewed are referred to, and some are also cited in length.

First comes a part where known cases of fatal decompression accidents are described. Then follows a part, describing in general, DCS in connection with heliox saturation and finally, since audiovestibular problems seems overrepresented in this type of diving, this is described in detail.

The observations are discussed and pertinent conclusions are drawn.

DEFINITIONS & ABBREVIATIONS

DCI	decompression illness
DCS	decompression sickness
DMAC	Diving Medical Advisory Committee
heliox	helium-oxygen gas mixture
msw	meters of sea water
NEDU	Navy Experimental Diving Unit (US)

POSSIBLE INJURIES, PATHOLOGY

FATAL AND NEAR FATAL DECOMPRESSION ACCIDENTS IN HELIOX SATURATION DIVING

There is no doubt that decompression sickness (DCS) may be fatal and it has been estimated that as many as 10 000 sponge divers died in the Aegean archipelagos and off the North African coast, in the period 1866-1915 [1].

In this century the situation fortunately has changed and Caruso (2003) [2] states “DCS rarely results in death, but may cause permanent morbidity in divers.”

Bradley [3, 4] analysed diving fatalities in Gulf of Mexico (1968-1975) and British sector of the North Sea (1971-1978). Five deaths in Gulf of Mexico and five in British sector are reported to be caused by “Decompression sickness/air embolism». Regarding the fatalities from the three deepest dives, the authors report: “Each of the three victims in these dives was brought directly to the surface without any water decompression, but with a predictably unfortunate outcome. Divers are exuberant spirits, who must be gently decanted.” It is not clearly explicitly stated, but the others seem to be air divers.

Warner (1977) [5] : “The first fatal accident recorded in the Department of Energy records happened to a diver in February 1971. He was diving in the Norwegian sector to a depth of

200 feet without a diving bell. The cause of death was drowning. A month later from the same installation Diver No 2 diving to 200 feet with a diving bell died to decompression sickness. The actual details of these two accidents are rather scarce."

Warner (1980) [6] : "Inadequate decompression schedules: Nil - for fatal accidents"

In a master thesis (history) Kahrs discuss all the 58 diving fatalities in the North Sea, he could find information about [7] . Several of these could be characterized as "blow ups", but none as "to quick/wrong decompression" from heliox saturation.

Although it seems that there has never been a fatality caused by DCS from anything that can be called a planned decompression from heliox saturation, there are cases that clearly show that if the decompression from saturation is so fast that it can be characterized as explosive or "blow up", it may be fatal.

In the North Sea the most grave example of this is the Byford Dolphin accident in 1983, where four divers from heliox saturation at 90 msw, within seconds came to surface [8]. Gjertsen et al. (1988) [9] report from the autopsies: "The most conspicuous finding was large amounts of fat in the large arteries and veins and in the cardiac chambers, as well as intravascular fat in the organs, especially the liver. This fat can hardly have been embolic, but must have "dropped out" of the blood in situ. It is suggested that the boiling of the blood denatured the lipoprotein complexes, rendering the lipids insoluble." This is quite similar to what has been reported from autopsies after fatal decompressions from air dives [10-12].

Although, as indicated above, there seem to be some discrepancy between Bradley [3, 4] and Warner (1980) [6] ; five fatalities in the British sector of the North Sea (1971-1978) are probably all explosive or "blow up" accidents, some of which are from heliox saturation. One of these accidents are well documented from the medical viewpoint by Norman et al. (1979) [13] . (It is also briefly mentioned in the DMAC workshop report [14].) "The dive was intended to be a bounce dive, and the two divers involved had been at a depth of 78 m for one hour when the diving bell made an unscheduled ascent to the surface in 42 s, due to the accidental detachment of the counterbalancing weight. The bell could not be sealed during the ascent and one of the divers died from gross pulmonary barotrauma. The other diver survived and was repressurized to a depth equivalent to 81 m within a few minutes of surfacing. Two doctors arrived on the diving ship 3 h 48 min later, and when one entered the deck compression chamber, he found the diver alive but deeply unconscious and cyanosed. A pulse was detectable only in the carotid arteries and its rate was 152/min. Only occasional respirations were noted and the pupils did not react to light. After recompression therapy, this diver was tetraplegic with evidence of patchy microcirculatory damage of brain, cord, liver, kidneys, and gut. All systems eventually returned to normal, except the spinal cord, mainly because of the post-recompression phase of management, in which pharmacological doses of steroids, hyperbaric oxygen, and dextran were used. Although function returned in the upper limbs, the diver remained paraplegic." So it is possible to survive 42 s decompression from near saturation at 78 msw.

General description of DCS

In a description of the physiologic and pathologic consequences of the presence of gas phase caused by decompression, Francis and Mitchell (2003) [15] explained which of the different organs are primarily affected by intravascular bubbles and which by tissue bubbles. Mostly it is not differentiated between different inert gases or whether it has been decompressed from saturation or not. An exception is for musculoskeletal manifestations, where they [15] (p. 541) state "Articular cartilage is also unlikely to be involved in anything other than decompression

from saturation because it is avascular and hence will take up inert gas only extremely slowly (perhaps this is why joint pain is overwhelmingly the most common manifestation of DCS in saturation divers and aviators, furthermore, it is an aqueous tissue and, consequently, would be expected to absorb only a modest gas burden compared with more fatty tissues around the joint".

Moon and Gorman (2003) [16] reviewed the early literature that describes the history of the DCI before recompression became widely used. They indicate that, unless they were rapidly fatal, most cases often improved spontaneously. The exceptions were cases with heavy spinal cord deficits that left the victims bed ridden. But of course, as we know now, the perspectives got better when recompression treatment came to use. They say (p. 601): "Statistical evidence for the benefit of recompression was shown by Keays (1909), who reported a failure rate of 13.7 % in caisson workers with pain treated without recompression vs 0.5 % failure with recompression treatment."

Terminology and classification of decompression disorders are given also by Francis and Mitchell [17]. The affection on the different target organs are described. They mostly do not differ between which diving methods have caused the decompression disorders, except again for musculoskeletal manifestations, where they state pain in the lower limb is more common than in upper limb, for saturation divers.

In another older review, Hallenbeck and Andersen (1982) [18] are much more detailed in differentiating between diving methods (p. 447.): "Although there is a spectrum of clinical illness labelled 'decompression sickness', syndromes occurring following 'bounce' diving segregate themselves from those occurring during saturation decompressions with regard to symptom types, overall incidence, association with detectable bubbles and late sequelae. One further distinction must be that there have been, to date, no successful animal models for saturation decompression sickness. Hence, comprehensive histopathological and physiological data, as derived from animal experimentation for bounce dive-produced decompression sickness, are unavailable to assist in an understanding of saturation syndromes. Most investigators appear convinced that liberated gas serves as the source of symptoms in both types of illness, but for the reasons alluded to in previous sections and reviewed below, it may be prudent to view the issue as unsettled." Further (p. 447-8): "Saturation diving, particularly deep mixed-gas saturation diving, has been associated with an unusually high incidence of decompression sickness. US Navy data indicate that 20 % of decompression incidents occur during saturation dives, although these dives account for less than 0.3 % of total Navy dives (Berghage 1980) [19] . Other studies support this observation (Gardette 1979 [20] ; Greene & Lambertsen 1980 [21]) and suggest that the ultrasonic detection of circulating bubbles, while of value in predicting potentially serious or troublesome decompression syndromes, is of little help in excluding the possibility of a decompression incident. That is, the absence of bubbles at rest and following movement was associated with an incidence of decompression sickness of 14 % and 10 %, respectively, during saturation decompressions, in contrast to an incidence of 1 % for bounce and excursion dives (Spencer et al. 1975 [22] ; Nashimoto & Gotoh 1978 [23] ; Gardette 1979 [20]). They also say (p. 448): "In contrast to the usually asymptomatic experience of the bounce diver, it is rare for a diver undergoing decompression from a saturation exposure to remain entirely free of symptoms. Many divers report a sensation of unease, tightness or mild discomfort, most often associated with knees or elbows, throughout decompression, and a sensitive individual can detect, by an accentuation of low-grade discomfort, the resumption of decompression after a temporary cessation. Decompression sickness, when it is acknowledged, occurs most commonly during the latter stages of decompression as a progression to severe aching pain in a previously uncomfortable joint. Pain-only bends accounts for approximately 86 % of saturation decompression incidents (Berghage 1980) [19] . The majority of remaining cases have been related to rapid excursions from saturation storage depth in helium-oxygen and have exhibited vestibular symptomatology (Gardette et al. 1977 [24] ; Berghage 1980 [19] ; Greene & Lambertsen 1980 [21]). In these situations, high Doppler bubble grade was

associated with an increased risk of vestibular and central nervous system non-spinal cord decompression sickness, suggesting an analogy to helium-oxygen bounce dives and their consequences (Gardette et al. 1977 [24] ; Masurel et al. 1977 [25]). Spinal cord symptomatology, commonly observed in air diving situations, is a relatively infrequent happening in saturation decompressions."

Hallenbeck and Andersen (1982) [18] reflect generally: "Probably because of the gradual nature of most saturation decompression profiles, cardiopulmonary and central nervous system forms of decompression sickness are observed most frequently following bounce dives, altitude decompressions and occasionally excursions from saturation storage depth. Following decompression, bubbles of undissolved gas appear in the venous circulation, exert their surface effects and accumulate in the lung. Their number, size and time of appearance are dependent upon the depth and time of the inciting exposure and the inspired inert gas. Following equivalent exposures, *helium bubbles are smaller, more numerous and more rapidly appearing than those of nitrogen* (Chapter 14, Vann (1982) [26]). When the decompression profile has been marginally adequate and the diver's physiological state is sufficiently stable, intravascular bubble production may soon subside. Local changes in the lung secondary to bubble and bubble-product entrapment may then be reversed by pulmonary endothelial synthesis of bronchodilatory and vasodilatory substances and the removal of cellular debris by phagocytic cells. Such a process may be subclinical or attended by only minimal symptomatology: a sensation of pulmonary 'tightness' due to a transient bubble and cellular aggregate-induced increase in pulmonary artery pressure and possibly an increase in airway resistance. It is superficially analogous to subclinical thromboembolic disease."

Also according to Vann (1989) [27] , spinal symptoms are relatively rare during decompression from saturation dives.

Berghage (1976) [28]: having analyzed all "available Navy saturation diving data", states that: "None of the cases of decompression sickness recorded during saturation dives involved more than musculoskeletal or joint pain". Adding to this, Berghage (1980) [19] states that most CNS cases that occurred in connection with excursions, were vestibular. This is in line with Greene and Lambertsen (1980) [21] who identified 10 DCS cases following heliox excursion decompressions, including 7 inner ear / vestibular, 2 spinal and 1 chest / pruritus.

Inner ear problems

Hallenbeck and Andersen (1982) [18] considerate also 'vestibular' decompression sickness in deep helium diving (p. 449): "The pathogenesis of 'vestibular' decompression sickness and its relationship to decompression events involving the brain stem remain complex and incompletely understood. It seems likely that mechanisms similar to those operative elsewhere in the central nervous system may be involved. There is experimental evidence suggesting that antagonism of procoagulant enzymes by heparin potentiation of antithrombin III may be of benefit in preventing cochlear damage by haemorrhage (McCormick et al. 1973 [29]). In Gardette's series, *vestibular decompression sickness following saturation excursions was preceded by elevated Doppler bubble scores at rest, in contrast to limb-bends occurring during saturation decompression where Doppler monitoring generally revealed no circulating bubbles* (Gardette 1979) [20]. Since bounce dives breathing air or mixed gas rarely produce vestibular symptoms despite elevated Doppler bubble scores, it is possible that the vestibular apparatus and its CNS connections may be 'sensitized' by high-pressure helium exposure to the early ravages of undissolved helium."

Under "audiovestibular manifestations" Francis and Mitchell [15] say (p. 543) "It is notable that 'pure' inner ear DCS is uncommon following air diving within the recreational diving range, but

became well recognized as deep diving using oxygen-helium mixtures became prevalent [30] (Farmer 1993). This has been attributed to expansion of ‘silent’ vestibulocochlear helium bubbles by inward diffusion of nitrogen following gas switching from oxygen-helium mixes to air during decompression [31] (Farmer et al 1976).”

Farmer (1993) [30] goes into this quite detailed and his “Inner Ear Injury Occurring During or Shortly After Ascent or Decompression (Otological Decompression Illness)” (p. 289) is cited fully here;
“Diving inner ear injuries related to decompression were noted by several nineteenth- and early twentieth-century writers. Smith (1873) noted severe deafness and vestibular problems among other injuries in compressed air workers and described these injuries as part of ‘Caisson Disease’. Heller et al. (1895), Alt et al. (1897) and Vail (1929) suggested that inner ear injuries during decompression were related to intra-labyrinthine nitrogen bubble formation or interference with the inner ear blood flow from intravascular nitrogen bubble formations. In the 1930s and 1940s, safety procedures for air diving improved, and the frequency of inner ear injuries in diving apparently decreased. Indeed, most of the diving literature concerning decompression illness noted symptoms suggestive of inner ear injury only in association with what was thought to be central nervous system decompression illness, where the inner ear symptoms were relegated to secondary importance and, in many cases, were possibly related to centrally located lesions. Otological symptoms were not felt to occur during decompression without other manifestations of decompression illness. Tinnitus, hearing loss and/or vertigo occurring without other signs of decompression illness during or shortly after decompression often were not evaluated or treated. Most of the diving literature concerning otological injury was devoted to middle ear barotrauma, felt to be reversible and not usually resulting in permanent or serious disabilities. During the 1960s and 1970s, exposures to deeper depths using mixed helium atmospheres became more frequent and isolated symptoms of inner ear dysfunction occurring during or shortly after decompression from dives in which decompression illness was possible were described. Thus, the syndrome of ‘inner ear decompression sickness’ (illness) became recognized.

Bühlmann and Waldvogel (1967) [32], reporting on 82 decompression accidents in a series of 211 dives, noted that the only neurological symptoms in the entire series consisted of vertigo, nausea, vomiting and tinnitus in 11 cases. Hearing losses were noted in two of these cases. These symptoms appeared only with decompressions from the deepest dives, depths of 485 and 726 ft (148 and 221 m). Nine of these 11 cases required recompression treatment, whereas a smaller proportion (49 out of 71) of the remaining non-inner ear accidents required treatment. Bühlmann and Gehring (1976) [33] described 12 cases of inner ear symptoms consisting of vertigo, nausea and vomiting, after 24 decompressions from depths ranging from 140 to 1000 ft (43 to 305 m). In four cases, there were associated hearing losses and tinnitus. With dives using longer decompression schedules, such inner ear symptoms were not noted. Barnard (1967) [34], Hempleman (1976) [35] and Spaur (1976) [36] have noted that inner ear decompression illness is a principal manifestation of overall decompression illness in deep subsaturation diving and has been best managed by immediate recompression to the depth of the dive.

Farmer et al. (1976) [31] reported on 23 cases of vestibular and/or cochlear injuries occurring during or shortly after decompression. These cases were selected from reports of military and civilian diving accidents on file at NEDU and from cases referred to the authors. Ten of these cases had been previously described by Rubenstein and Summit (1971) [37]. Cases were excluded in which the divers had difficulty clearing their ears during compression, described ear symptoms while at the maximum depth, or were exposed to uncontrolled or rapid emergency ascents, with possible air emboli. Excluded also were cases with insufficient information or with signs of hypoxia, hypercarbia or other neurological symptoms suggestive of central nervous system decompression illness. Ten cases presented with vestibular symptoms only; seven cases with auditory symptoms only; six cases with auditory and vestibular symptoms. Four of the cases were air dives requiring staged decompression; the remaining 19 cases were helium-

oxygen dives. In this series, the 11 divers who were recompressed within 42 min after symptom onset during or shortly after ascent experienced relief during recompression and had no residual inner ear dysfunction. Three divers were recompressed within 60-68 min after symptom onset; only one of these divers experienced relief of symptoms. The remaining two divers had only partial or no relief and exhibited significant residual sensorineural hearing loss or vestibular dysfunction. No cases in which recompression treatment was delayed longer than 68 min after symptom onset, exhibited relief or lack of residual inner ear dysfunction. Thus, a significant correlation between prompt recompression treatment and recovery was indicated. This finding, plus the fact that the symptoms all began either during or shortly after decompression and the absence of otological symptoms at other phases of the dives, indicated that these cases represented true decompression illness. Otic barotrauma was an unlikely contributing factor in this series, in that no diver noted difficulties with ear-clearing during compression.

Thirteen of the 19 helium dives in this series involved a change to an air atmosphere at depths ranging from 60 to 150 ft (18.5 to 46 m) during the latter stages of decompression. In one of these cases, the symptoms began before the air change but became more severe after the diver had entered the air atmosphere. This sudden change in gas composition was felt possibly to contribute to the tendency for nitrogen molecules to enter into ‘silent’ helium bubbles sufficiently to achieve a critical size and cause symptoms. This phenomenon may be similar to that described above for isobaric counterdiffusion inner ear injuries, in that the counterdiffusion of the two different inert gases between inner ear fluid compartments may have resulted in bubbling at tissue interfaces. Thus, the inner ear seems to be particularly susceptible to injury during changes in inert gas composition, not only at stable deep depths, but also during decompression.

Animal studies have increased the understanding of the pathophysiology of inner ear decompression illness. McCormick et al. (1973 [29], 1975 [38]), in studies designed to produce experimental decompression illness in guinea-pigs, actually observed bubble formations and haemorrhages in the labyrinth, with depressions of the inner ear electrophysiological responses to auditory stimuli. These investigators also observed that the deficits in inner ear electrical function in decompressed guinea-pigs could be reduced by treatment of the animals with heparin prior to the dive, suggesting that a mechanism of inner ear decompression illness may be lipid-platelet emboli and/or hyper-coagulation in the inner ear microvasculature similar to the coagulation changes noted in decompression illness (Philp 1974) [39].

Extensive studies of the pathophysiology of inner ear dysfunction in the squirrel monkey after rapid decompression have been reported from Toronto by Landolt et al. (1980) [40]. More than 90 squirrel monkeys underwent bilateral myringotomies and were rapidly decompressed in a hyperbaric chamber, using a special diving profile in which 35 % of the attempts produced decompression illness which appeared to be confined to the inner ear. Physical examinations and electro-nystagmographic recordings plus post-dive histological studies revealed that the symptoms of inner ear dysfunction occurred during the latter stages of decompression and were related to inner ear pathology. The animals were sacrificed at times ranging from 1 h to more than 12 months following inner ear decompression illness.

Temporal bone histological studies at 1 month or less after the injury revealed haemorrhage and/or a deep purple-staining precipitated material in the otic fluid spaces. In those animals killed more than 1 month after the injury, fibrosis and ectopic new bone growth in the canals was a common finding. Interestingly, new bone growth did not appear in the cochlea. In control animals, precipitated material was noted in the inner ears but was less than in those ears damaged by decompression; also, fibrosis and new bone growth did not occur. No apparent differences in the inner ear injuries were noted with different ambient gases, with switching of gases during the latter stages of decompression or with the type of diving profile.

Landolt et al. (1980) [40] concluded: (1) the inner ear in squirrel monkeys is unusually susceptible to damage from decompression, as it is in humans; (2) vestibulocochlear disorders

resulting from decompression are of peripheral origin unless the central nervous system involvement is extensive and obvious; (3) the cochlear disorders tend to be vascular lesions, mainly located in the stria vascularis in the spiral ligament; and (4) the vestibular lesions appear mostly along the arms of the semicircular canals and are more extensive and persistent than the cochlear lesions, with fibrosis and new bone growth noted in the semicircular canals. The cochlear lesions were felt to be likely to be related to microvascular bubble formations with blockage and/or rupture of microvessels causing haemorrhage or blood protein exudation into the labyrinthine fluid-filled spaces. This is likely to occur in the low-pressure venules or in the stria vascularis where the blood flow rate is slower despite a large number of vessels. Blockage of the venous ends of the capillary beds in an area where there is no collateral circulation, such as the vestibular and cochlear regions of the inner ear, results in a rise in intravascular hydrostatic pressure in the pre-capillary areas as well as arterioles, with a subsequent rupture and haemorrhage and/or transudation of fluids and macromolecules into the extravascular, intra-labyrinthine fluid spaces.

The likely reasons for the finding of precipitated material and haemorrhage in both the perilymph and endolymphatic spaces in the cochlear but only in the perilymphatic spaces of the vestibular apparatus was felt to relate to the larger microvascular blood supply in the vestibular perilymphatic spaces than in the endolymphatic spaces. The connective tissue formation and new bone growth found at a late stage after the injury in the semicircular canals is not an uncommon finding after labyrinthitis. Similar phenomena have been reported by Kimura and Perlman (1956) [41] in the perilymphatic spaces of the vestibular apparatus after permanent venous obstruction but not after arterial obstruction. In their experiments, haemorrhage into the semicircular canals occurred as early as 3 h after venous obstruction, and was followed by fibrosis in the perilymphatic spaces 2 weeks later, and then varying degrees of ossification for up to 6 months later.

Chiappe (1939) investigated histologically the inner ear lesions resulting from decompression in guinea-pigs and cats with variable dive profiles. In these studies, the severity of the inner ear lesions was related to the speed of decompression, with rapid rates of decompression producing vascular lesions and extensive haemorrhaging and slow rates producing mild congestion of intact blood vessels with exudation of a protein-like material into the labyrinthine spaces. His histological observations were generally similar to those reported by McCormick et al. (1973 [29], 1975 [38]), Long et al. (1977) and Landolt et al. (1980) [40], except that the Landolt investigations noted that gross tissue damage was seldom produced in the nerve fibres, ganglia or neuroepithelia of the inner ear. Most of the inner ear damage from decompression appeared to be located in the fluid-filled spaces.

In later papers by the Toronto group, Venter et al. (1983) [42] and Fraser et al. (1983) [43] revealed an interesting pathological mechanism to explain some of the inner ear findings noted in the squirrel monkeys described by Landolt et al. (1980) [40]. Full fractures of the endosteal bone layers surrounding the semicircular canal walls was noted in some of the animals sacrificed a few days after decompression. Those sacrificed several months after decompression revealed that these fractures were infiltrated with new bone growth and fibrosis into the fluid spaces. These changes appear related to significant pressures from bubble enucleation and growth in the osteoclastic cell cavities of the endosteal bone layers developing during the later stages of decompression. The subsequent pressure differential between these cellular spaces and the adjacent perilymphatic spaces were postulated to result in a fracture of the endosteum into the perilymph. The implosive nature of these fractures apparently caused a pressure wave in the canal fluids with further teating of the endosteum, bleeding and disruption of the membraneous labyrinth, all of which are changes related to subsequent fibrosis and new bone growth as noted above by other authors. Indeed, Money et al. (1985) [44] described similar changes in the left temporal bone of a professional diver who was accidentally killed 56 days after suffering left inner ear decompression illness. He did not respond to

recompression and exhibited a persistent loss of left end organ vestibular function and left sensorineural deafness."

SUMMING UP

Reviewing publications with reports on symptoms from heliox saturation decompressions, a general trend is found. Symptoms are dominantly skin/musculoskeletal pain-only and in the few cases with other symptoms, these are practically exclusively vestibular [45, 46, 20, 21, 47-50, 33, 51-55, 28].

DISCUSSION

At moderately increased decompression speed one can expect pain-only, possibly audiovestibular symptoms. Neurological symptoms are unlikely.

If the decompression rate can be titrated by stopping /recompressing at symptom onset of pain and/or audiovestibular symptoms, the probability of serious neurological DCS is small.

We don't know what will happen if decompression that has triggered serious DCS symptoms, is carried on. It is not known how the symptoms may develop, since this has not been systematically studied. In Swann (2007) [56] however, examples of divers who have continued decompression with symptoms that went away after surfacing, are described. Moe and Bjelland (1994) [57] interviewed 15 saturation divers who lost their diving license between 1987 and 1992. 75% said that they had symptoms, mostly of DCS that they never reported [57]. In a questionnaire study, including 112 professional divers holding a bell diving certificate, 63 % of these saturation divers reported to have had decompression symptoms that never got treated [58] . Also there are descriptions where, probably based on experience, symptoms have been treated only with hyperoxic gas, not recompression [59] . Gardette et al. (1979) [20] report 25 cases of muscle or joint pains (23 during decompression from saturation) where only 2 were considered to "require therapeutic recompression". So there is reason to believe that even with symptoms of DCS and no recompression, there is a good chance for return to surface without acute intolerable problems.

One can imagine increasing the decompression speed till some divers get symptoms. What we don't know enough about, is how much it will delay subsequent decompression if one is to wait for divers with DCS to become sufficiently symptom-free to continue.

Anticoagulants like heparin, may possibly reduce the consequence of the audiovestibular DCS. But we don't know enough about if it can increase bleeding as we see it by spinal and cerebral DCS. Spinal, cerebral and audiovestibular DCS are characterized by petechial bleeding (respectively bleeding in the inner ear). Although heparin may be able to influence protein/lipids in the lymph of the ear, it can in parallel contribute to increased bleeding in the same places. We do not have sufficient knowledge to recommend heparin given prophylactic prior to emergency decompression.

CONCLUSIONS

If accelerated decompression from heliox saturation is carried out, the most probable medical problems will be pain only (limb) bends.

Next to this may be vestibular problems with dizziness (vertigo) and hearing loss (deafness) as probable symptoms.

Life-threatening conditions is not to be expected.

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