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SECURING THE HYPERBARIC TREATMENT OF DECOMPRESSION SICKNESS IN THE POLISH NAVY

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ABSTRACT

Recently, the Polish Navy has extended its capability for the implementation of underwater works with autonomous dives conducted beyond the saturation zone to a depth of $80\ mH_2O$. In the near future it is also planned to introduce long-term dives within typical depths of the saturation plateau.
One of the activities resulting from the analysis of the risk associated with the extension of these competences is the need to conduct a critical review of the system for securing hyperbaric treatment of cases of decompression sickness[1].
Keywords: decompression sickness, saturation diving.

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INTRODUCTION

Currently, the Polish Navy utilises the system of recompression tables (TT)[2] proposed by the US Navy [1]. They were developed as a result of analysis of possible, effective TT for use in the hyperbaric treatment of various forms of decompression sickness, with particular emphasis on the acceleration[3] of the inert gas desaturation process using oxygen $oxy - TT\ US\ Navy$. It provides coverage of hyperbaric treatment for a significant proportion of DCS cases. The use of these procedures in place of traditional treatment methods allows to shorten the treatment time as compared to saturation methods [2]. It also requires less hyperbaric equipment, thus reducing the investment costs. This makes it possible to popularize the methods of recompression and subsequent prophylactic, rescue and later treatment decompression at the place of an occurrence of a case of DCS [3,4].

However, with the currently planned increase of competences in relation to underwater works, the Polish Navy should consider returning to some traditional TT, including saturation, i.e. $sat - TT$.

For severe DCS cases, e.g. caused by explosive decompression[4], during $Trimix \xrightarrow{Tx} Heliox\ Hx$ independent deep dives outside the saturation zone, $sat - TT$ are still the most effective rescue method.

DECOMPRESSION

As the diver ascends, the ambient pressure decreases, and the inert[5] gases dissolved in the tissues are redistributed in each direction towards places where the pressure is lower. What is desirable for the desaturation process is the flow of the inert gases towards the lumen of the blood vessel from where they can be transported to the lungs and eliminated from the body by means of gas exchange. This unidirectional flow occurs following saturation[6] – fig. 1.

The reduction of external pressure results in the removal through the blood of inert gases from the part of the tissue adjacent to the blood vessels. At this time, inert gases from parts of the cells[7] in which their concentration is higher, move to the cells surrounding the blood vessels, as well as to others in which their concentration is lower. The content of the inert gas accumulated in various tissues and organs depends largely on its solubility and tissue perfusion[8] by blood. The example values of solubility and perfusion contained in Table 1 come from studies on the elimination of nitrogen from the human body during breathing with pure oxygen[9] after dives at a medium depth, assuming that the body fat does not constitute more than 10% of the body mass [5,6].

Depending on their perfusion, tissues can tolerate the excess of accumulated inert gases without generating symptoms of DCS. This allows to increase the diffusion drive module[10] by creating an increased pressure of dissolved gases in tissues in relation to their partial pressures in the respiratory atmosphere, thus intensifying the decompression processes.

The course of decompression under isobaric conditions is also shown in Fig. 1. Isobaric decompression is most often applied when resting on the surface. The decompression process usually results in the diver being left in a phase of safe saturation.

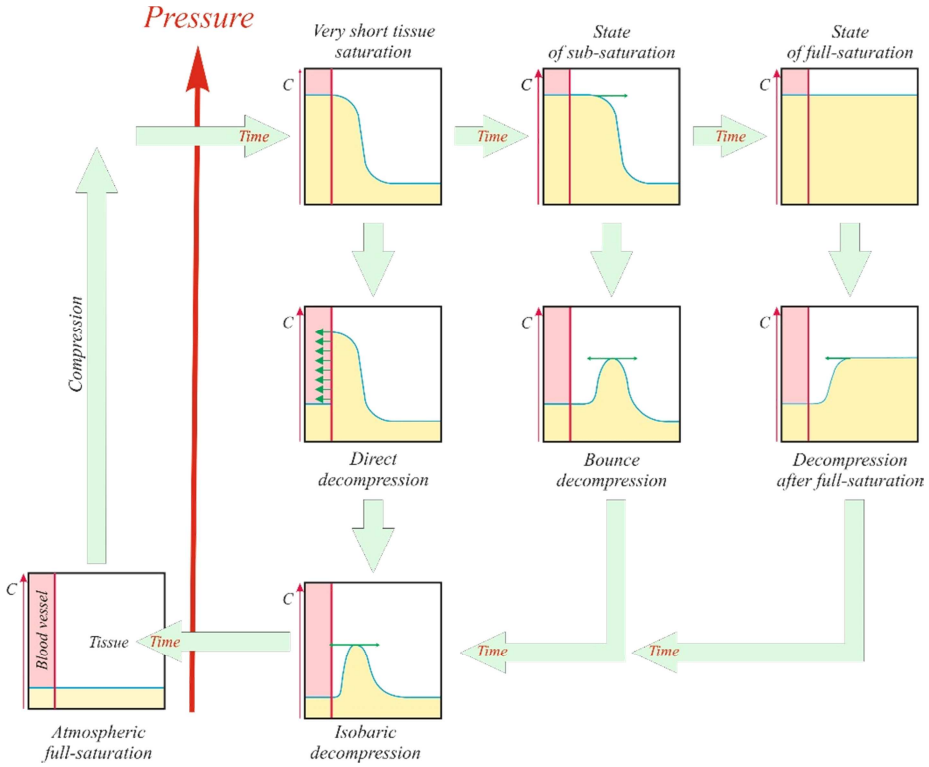


Fig. 1 The scheme of distribution of an inert gas near a blood vessel during the compression and decompression processes.

Tab 1

Selected parameters for different tissues and organs during nitrogen elimination when breathing oxygen after air diving at medium depths [5,6].

Specification	Groups of tissues and organs				
	Blood, brain, heart, kidneys	Muscles, skin, spinal cord, nervous system	Cellular bones (no fat)	Marrow bones (rich in fat)	Adipose tissue
mass $[g]$	15000	37000	3500	1500	9500
water content $[g]$	12000	30000	2000	240	2000
fat content $[g]$	350	100	—	1200	7000
nitrogen content $[cm^3]$	126	275	18	63	368
blood perfusion $[cm^3 \cdot min^{-1}]$	4000	1200	80	50	375
nitrogen transport in the first minute $cm^3 \cdot min^{-1}$	40	12	0.8	0.5	3.75
desorption half time $[min]$	1.9	16	16	85	69

In some cases, an inert gas deposit left in tissues is potentially dangerous. The utilised decompression tables should specify the principles of carrying out isobaric decompression during mandatory rest on the surface. Physical activity following decompression may result in the dynamic extrusion of inert gases contained in muscle tissue, thus causing imbalance and the release of a free gas phase, potentially increasing the risk of DCS symptoms. After saturation, making the least effort can cause loss of homeostasis[11] owing to different reasons, such as lack of quick adaptation from hyperoxic to normoxic conditions. A physical activity following decompression may cause rapid acidification of the blood, thus reducing the efficiency of oxygen transport by haemoglobin. This leads to difficulties in the process of isobaric decompression on the surface, although the pressure and composition of the respiratory atmosphere is favourable.

The atmospheric pressure change during a flight in an aircraft[12] or helicopter violates the state of balance after decompression[13] due to a relatively rapid pressure drop at flight altitude[14]. This may cause an imbalance of the established equilibrium[15] in relation to atmospheric pressure and initiate a free gaseous phase, leading to the development of DCS symptoms.

The replacement of one breathing mix with another activates the phenomenon of counterdiffusion, which, with large differences in the solubility of diffusible inert gases, may result in an increase in tissue pressure even under constant external pressure. Counterdiffusion may then lead to the initiation of a free gaseous phase, which in turn may produce symptoms of DCS. Counterdiffusion phenomena occur not only when the breathing mix changes in hyperbaric conditions, but also in normobaric conditions[16].

A specific type of breathing mix change is the use of oxygen, or mixtures with increased oxygen content[17], to accelerate decompression processes. After removing inert gases[18] from tissues, the oxygen becomes quickly metabolised in them, thus creating a pressure gap commonly referred to as an "oxygen window" [4]. The use of oxygen and oxygen-rich mixtures is not only the basis for decompression acceleration, but also for the treatment of certain forms of DCS.

DCS

In the paper the DCS[19] division proposed by Wienke was adopted – tab. 2. The most common cases include: I-DCS and II-DCS. It is believed that they are primarily caused by the release of a significant amount of free gas in to tissues. Their early symptoms can be effectively treated with hyperbaric methods using TT, leading to a reduction in the size of the free gas phase bubbles, enabling their resorption and elimination by inhalation.

Tab. 2

Decompression sickness (DCS) division [7].

$I-DCS$	**Decompression sickness of the limbs** is manifested by local limb pain, itching of the skin, local redness of the skin, swelling or weakness of the knees, hips, elbows, muscles or skin.
$II-DCS$	**Decompression sickness of the central nervous system** manifests itself in confusion, anxiety, paralysis, shortness of breath and chest pain, difficulty breathing, loss of consciousness, difficulties in focusing attention, problems with maintaining balance and an upright posture, especially maintaining the spine.
$III-DCS$	**Decompression sickness of the inner ear** is manifested by impaired hearing, dizziness, ringing and tinnitus or nausea and is the result of pressure affecting the balance of the organs in the inner ear.
$IV-DCS$	**Sterile bone necrosis** is manifested by mechanical bone damage, structural damage, local mineralisation, in particular by attacking long bones.

$I\text{-}DCS$ is usually caused by the precipitation of a free gaseous phase in adipose and connective tissue and II-DCS in nervous tissue. The presence of a free gaseous phase in the nervous tissue, especially its location in the neurolemma[20], may cause obstruction of nerve conduction and result in neurological symptoms: II-DCS. Blockage of blood flow through the bubbles generated by the free gaseous phase may cause local ischaemia leading to necrosis. Gas emboli are particularly dangerous for the brain. It is also believed that they may develop into IV-DCS in the bones and joints.

Mechanical destructive actions are not the only pathological symptoms induced by the free gaseous phase. The body's defensive reactions induced by the free gaseous phase need not be directly related to its mechanical action, but they also have a biochemical basis for pain formation. This theory, which is referred to as complement activation[21] appears to be well documented [8]. There are several theories on the probable mechanism of inducing pain for the symptoms of I-DCS [9]. It appears when the bubbles of the free gaseous phase reach a size large enough to:

- irritate the nerve endings by causing their displacement.
- block capillaries causing ischaemia and death of tissues which by excreting active substances warn the brain by means of chemical warnings, inducing a feeling of pain generated by the brain.
- activate the biochemical reaction of antibody production, which also stimulates pain signalling their presence in living cells.

$II-DCS$ may be the result of blockage of blood flow to the spinal cord resulting in the release of stimulation or inhibition of higher brain functions[22].

$III-DCS$ may be caused by counter-diffusion and/or osmosis[23] of dissolved inert gases causing an increase in their pressure and displacement of bodily fluids in the inner ear. Bodily fluids aim to compensate for the concentration of the gases contained there, causing dilution of a more concentrated solution by their migration. This may result in an increase in the volume of fluids in the organs of the inner ear and, as a result, an increase in pressure causing abnormal functioning of the labyrinth [10].

Theoretically, the symptoms of III-DCS may also be caused by anomalies appearing on the surface of cell membranes, for which counter-diffusion caused by gradients of content of various inert gases is responsible. When a gas is diffused in one direction and the other gas in the opposite direction, their counterflows cause disruptions to the gaseous exchange at the borderline of the phases. In addition, differences in their natural solubility in tissues[24] may cause the accumulation of gases beyond their solubility limits also under isobaric conditions [11]. Their mutual competition for a place in the solution may result in the displacement of one of them[25] [12].

It is difficult to say unequivocally what is the connection between IV-DCS and decompression processes. Statistically, this disease is of importance for professional divers and caisson workers. Both professional groups are repeatedly exposed to high pressures. It is probable that repeated rapid compression and carbon dioxide content promote the induction of symptoms of sterile bone necrosis. Also long exposures to oxygen partial pressures above 60 kPa may contribute to the development of the symptoms of sterile bone necrosis, although this mechanism is difficult to determine.

Dehydration symptoms similar to IV-DCS are observed in racing horses and alcoholics. Symptoms are local bone necrosis without being induced by infection[26]. If pathological sclerosis occurs in the central area of the bone, it does not hinder vital functions. If pathological changes appear in the joints, it may cause splinters and joint damage. It is assumed that sterile bone necrosis is a symptom of IV-DCS. Induction of IV-DCS symptoms may be triggered by the free gaseous phase causing embolia[27] and thus ischaemia caused by the plugging of small blood vessels in bone tubules. The mechanisms of the development of IV-DCS symptoms have no clear explanation. As mentioned above, a probable mechanism is the blocking of blood supply to the bone marrow and living bone tissue by bubbles of the free gaseous phase localised in capillary vessels [3]. A correlation was observed between the occurrence of pain symptoms in joints and the level of "yellow" bone marrow content in bones. "Yellow" marrow is less irrigated with blood[28] and contains more fat than its "red" variety. In combination with the generally known good solubility of inert gases in fats, it can be assumed that difficulties in transporting gas from the inside of the bones may be the cause of disease symptoms [5].

Treatment of III-DCS and IV-DCS with hyperbaric methods is not recommended. In fact, treating III-DCS with therapeutic recompression using TT outside the saturation zone results in a significant deepening of

disease symptoms [13]. This remark is important as II-DCS and III-DCS are difficult to distinguish.

During saturation the symptoms of I-DCS may occur after a trip to depths shallower than the plateau of saturation or during decompression following saturation completion. It is manifested by typical sores on the borderline of skeletal muscles and tendons, in the vicinity of large joints, in particular knee joints. It may be preceded by skin symptoms such as itching, rash, spottiness or marbling of skin. The most common initial symptom is increasing stiffness in the knee joints, which makes it difficult to move. Next, pain appears in the joints and intensifies within the lapse of a few hours. However, divers should be sensitised to differentiate between pain caused by minor injuries or effort[29] and such that may be due to decompression. For this purpose, you should carefully consider the history of the development and exacerbation of pain. If it is unlikely that it arose before the beginning of the decompression process, and the pain was unchanging over time, it was caused by DCS.

I-DCS which developed during a trip from the saturation plateau and up to 60 minutes after a trip above the saturation plateau depth, should be treated in the same way as II-DCS, because in this case I-DCS symptoms most frequently are only a first sign of more serious symptoms.

Treatment of DCS symptoms during saturation is different from the treatment applied after a completed saturation diving and operational dives outside the saturation zone [4]. Hyperbaric treatment of DCS in saturation may also be used as a hyperbaric DCS treatment method following dives performed outside the saturation zone [14]. The effectiveness of such methods should be higher than that of other methods, but these are long-term methods. Often, treatment with oxy-TTT treatment tables is more effective, the most popular of which is the oxy-TTT US Navy table system [15,1].

Treatment of I-DCS and II-DCS cases should be based on the quickest possible implementation of hyperbaric treatment using TT in order to prevent the induction of the body's defence mechanisms.

The first therapeutic parameter is the influence of pressure, which causes compression of the precipitated free gaseous phase in the tissues. Compression, first of all, leads to the reduction of the mechanical effects of gas on tissues, unblocks the blood flow in capillaries and prevents the described reaction of the immune system.

The second parameter is the time needed to restore homeostasis[30], resorption of gas in tissues and body fluids, and then transport the excess dissolved gas to the blood. Through the blood perfusing tissues of the body and the pulmonary tissue, the excess gas is eliminated from the body through the respiratory tract. The factor intensifying the process of hyperbaric treatment is the use of oxygen or mixtures enriched with oxygen[31]: $oxy - TT$.

The process of therapeutic decompression can also be supported pharmacologically by dilution of blood, appropriate hydration, administration of analgesics, anticoagulants, antithrombotic and anti-inflammatory agents, etc. The freedom in applying different pressures, time spans and compositions of the breathing mix is often limited by the phenomenon of oxygen toxicity, counter-diffusion, respiratory resistance, etc. The problem of oxygen toxicity has been omitted in this paper, as it is too extensive and has already been discussed in separate articles [4,16].

Counter-diffusion modelling has not yet resulted in a coherent theory, although the phenomena of counterdiffusion are relatively frequent in dives [17,18]. The occurrence of DCS symptoms associated with counterdiffusion can be divided into two forms: cutaneous SICD[32] and intraorganic DTICD[33]. The cutaneous forms accompany the situation when the diver breathes with a different breathing mixture from the surrounding one [11]. For example, the Polish Navy utilises deep diving technology using Tx as a breathing mix while the suit is filled with air.

During the decompression phase, the diver replaces the breathing mix with air during transfer under pressure and then undergoes oxygen decompression. Most divers exhibit slight skin symptoms of I-DCS in the form of pruritus. DTICD[34] symptoms accompany sequential changes in the breathing mix. Both the sequential changes in the breathing mix from lighter to heavier, and vice versa, may cause DCS symptoms even if not accompanied by a change in depth – ICD[34]. The combination of the phenomena of gas diffusion through cell membranes and their solubility in tissues may result in the exceeding of the saturation limit and the release of a free gaseous phase in tissues causing symptoms of DCS, which is particularly the case with helium/nitrogen counterdiffusion [19].

Counterdiffusion may occur in the course of oxy-TT US Navy treatment after an accident occurring during Tx/Hx deep-water or saturation dives. With the use of TT COMEX proposed by the French company COMEX, it is possible to minimise the likelihood of counterdiffusion by including in the treatment the breathing mix that was used when the symptoms of DCS occurred [14].

Divers' decompression tables, commonly used around the world, usually only allow the diver to move around in the depths, without being burdened with additional effort caused by hyperbaric conditions. For example, divers must wear suits which result in more effort being needed to carry out activities under water, they must also carry at least a small reserve of the breathing mixture on their backs, which requires an extra amount of effort associated with carrying it around during performance of the dive, etc. Only part of the decompression tables allow for additional effort[35] in performing useful work underwater. This limitation is due to the need to undergo decompression later. The decompression process will be then disturbed by metabolic products resulting from the effort. For example, the acidification of the body with lactic acid as a result of anaerobic metabolism results in blood acidification.

Under these conditions, haemoglobin loses its normal oxygen transfer efficiency. This hinders gas exchange and thus interferes with the decompression process[36]. Accurate assessment of the impact of workload on the planned decompression is difficult. Only saturation dives give divers the opportunity to make relatively large efforts, as the decompression process is postponed by a properly selected, relatively long rest period for the purpose of stabilisation[37].

PROCEDURES

A distinction should be made between two different moments of implementation of recompression[38] or therapeutic decompression, use: immediate and deferred.

Postponement of therapeutic decompression procedures usually causes complications associated with

the activation of the body's natural defence mechanisms, which in these conditions can sometimes even be considered as autoaggressive. Deferring the application of hyperbaric treatment procedures may cause such significant changes in the diver's body functioning that the treatment has to be based primarily on the best life-saving techniques, carried out in a specialist hospital[39], in which TT may only play a supportive role.

Decompression studies and analysis of historical emergencies show that the postponement of recompression, without risk of exposure to DCS symptoms, for an air surface decompression procedure cannot exceed 7 minutes [21]. In the case of air and Nitrox Nx saturated decompression it is up to approx. 5 minutes after the surface is reached [22]. It has been reported that it is possible to withstand a practical DCS risk for up to 15 minutes, which is expressed by the probability of an occurrence of severe symptoms of II-DCS at the level $p_{II-DCS} \triangleq 0.2$, whereas after the time of 30 min at the probability level for a mass accident reaches $p_{II-DCS} \gg 0.5$ [23].

The above analysis shows that military diving operations undertaken in conditions of peace should be secured by the possibility of initiating hyperbaric treatment of DCS symptoms on site, as transport to a specialist hyperbaric medical centre becomes problematic owing to the limited time for evacuation of all II-DCS cases. The current consensus within NATO is as a minimum to commence treatment of II-DCS and, if possible, to treat all DCS cases on site [24]. Through the implementation of the STANAG 1432 standard, this view is also officially binding for the Polish Armed Forces.

The exception is oxygen I-DCS, which is not required to be treated because the symptoms disappear spontaneously and the likelihood of their occurrence is limited to theoretical predictions [25,26]. This applies to procedures where the oxygen content of the breathing mix should be maintained at $x_{oxy} \geq 98\%_v$. Currently, following the example of the US Navy and Bundesmarine, a thorough ventilation of the breathing space of a closed-circuit diving apparatus is being abandoned, hence in the circulation there is $Nitrox\ \ Nx \in [0{,}85; 0{,}90]\%_v O_2$. In such dives the probability of developing I-DCS symptoms is higher, however all mild cases of I-DCS can be treated with hyperbaric *TT* with a considerable deferral, without a significantly increased risk of lasting repercussions[40].

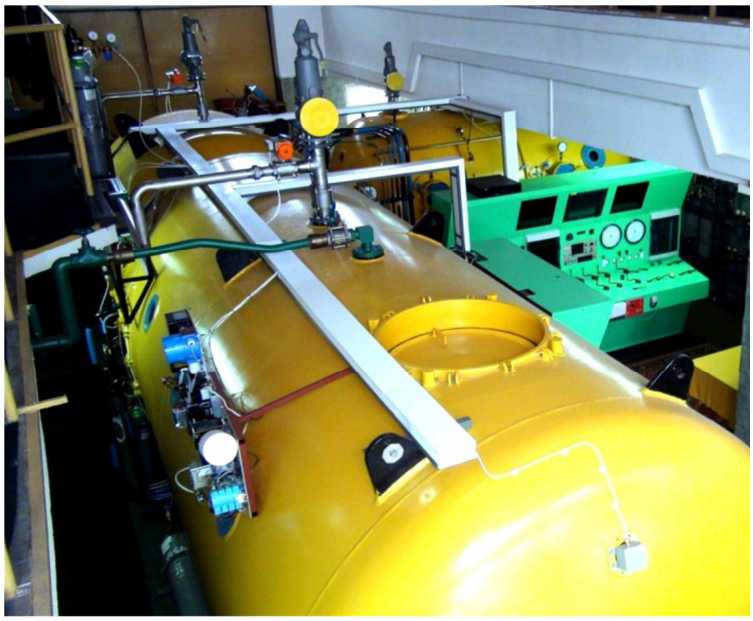
It can be argued that the immediate implementation of TT in relation to an injured person with no additional complications[41], is not a therapeutic procedure, but only a continuation of diving procedures. In many countries, procedures using TT can be entrusted to a diving supervisor. A problem situation consists in determining whether the postponement has already caused significant disruption of life processes. Hence, in countries where TT procedures are implemented by the diving supervisor, back-up is practiced in the form of remote medical consultations and, if possible, transport of a diving specialist to the place of initiation of hyperbaric treatment. Continuous presence of a physician at diving works, although formerly practiced in the Polish Navy and now desirable, is in many cases an excessive burden in terms of finances and organisation.

TECHNICAL REQUIREMENTS

The implementation of long-lasting hyperbaric treatment procedures does not require the involvement of a full-size, typical hyperbaric complex for conducting saturation dives, but only the introduction of a few additional subsystems. Successive modernisation of hyperbaric facilities currently in operation in the Polish Navy has led to the incorporation of reasonably accurate and precise measurement systems. The question of their unification and standards for automatic data collection and documentation remains to be resolved. It seems that serial production of systems for video observation, audio recording and controlling the composition parameters, as well as the hyperbaric atmosphere used in the SERCÓWKA sets, would be a good solution – fig. 2.

The automation of processes of recompression/decompression and maintenance of partial oxygen pressure seems to be a reasonable solution to improve the comfort of treatment procedures. The prototypical technical applications are excellent when conducting saturation training at the DGKN-120 facility - Fig. 3.

a)



b)

Fig. 2 a) Experimental Deepwater Diving Complex DGKN-120; b) The SERCÓWKA set with visible data collection, visualisation, video and communication system.

These solutions have the important advantage that they operate in the same IT system as the measurement systems used in the SERCÓWKA set. Their manufacture, as elements of a test series consolidated with the measuring system used in the SERCÓWKA set for serial production, should not be long-lasting nor expensive. Such a system would become an element of deliveries carried out by the Polish Navy within the framework of commissioned modernisations or building of new hyperbaric facilities in the Polish Navy.

a)

b)

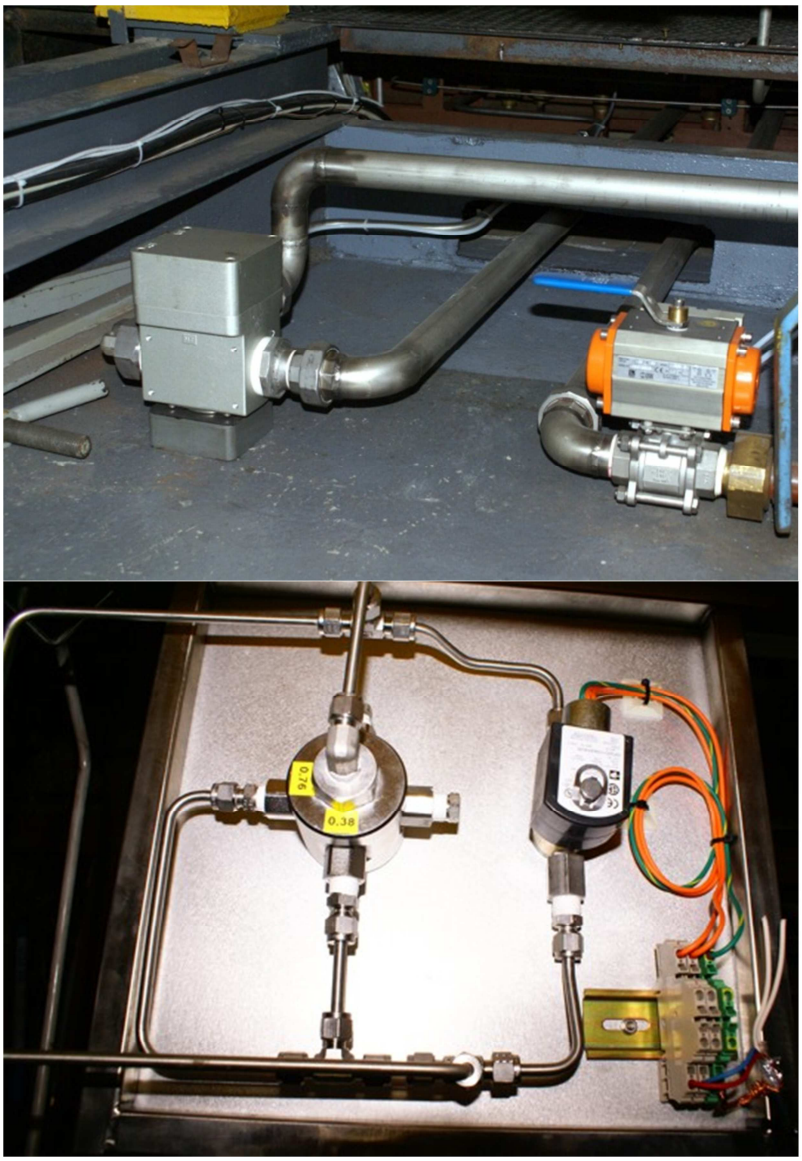


Fig. 3 a) Elements of the pressure maintenance and modification system during the compression and decompression process at the DGKN-120 complex; b) Elements of the oxygen partial pressure maintenance system at the DGKN-120 complex.

When adapting hyperbaric facilities for long-lasting therapeutic procedures, it is important to equip them with elements of life support systems[42]. Most of the hyperbaric complexes used in the Polish Navy are located in rooms or containers, hence heating systems based on hyperbaric radiators seem to provide sufficient thermal comfort in the decompression chambers.

a)  b) 

Fig. 4 a) LSS for the system of $DGKN - 120$, b) internal scrubber for removing carbon dioxide from the atmosphere of the hyperbaric chamber.

In addition, these systems can be equipped with integral, emergency power supply from UPS systems or external support, for example from an additional unit that can be taken on board a ship or connected to the ship's network.

Hyperbaric complexes operated in the Polish Navy are equipped with internal regeneration systems in the form of scrubbers with sodium lime - fig.4b. Hyperbaric facilities intended for long-lasting therapeutic procedures should have at least the possibility of connecting external regeneration systems or have them in stock. They may be a reduced version of typical LSS[43] used in saturation systems. LSS prototypes were produced at the Naval Academy and multiplied to three series – fig. 4a.

The design of faeces removal systems poses more technical difficulties for hyperbaric facilities already in operation due to the lack of space for them. Where appropriate retrofitting cannot be carried out, the faeces removal process should be carried out by sluicing.

Long-term hyperbaric treatment of DCS symptoms occurring during or after saturation dives does not require additional technical equipment due to the presence of a full-size saturation diving complex at the site of the incident.

It seems that all hyperbaric complexes used in the Polish Navy should be adapted to include various types of portable medical equipment dedicated to hyperbaric conditions, such as: respirators, defibrillators, ECG monitoring, etc.

Attempts can be made to develop our own simple elements of hyperbaric medical equipment, such as vacuums or automatic respirators. In addition, attempts may be made to adapt existing typical medical technical equipment to hyperbaric conditions, such as ECG monitoring systems.

Long-term hyperbaric treatment procedures additionally require securing sufficient supplies of therapeutic gas mixtures. This may be based on exchangeable cylinder units connected to hyperbaric gas system installations.

Securing carbon dioxide sorbents could be based on a regular storage management with regard to utilisation and rotation.

CONCLUSIONS

If a decompression accident does not cause significant changes in homeostasis[44] in addition to gas imbalances, or they are marginal, the processes leading to restoring the equilibrium are based on a correct selection of time and pressure for the recompression process. Pressure should lead to compression of the free gaseous phase, thus reducing its mechanical impact on tissues, unblocking blood flow in capillaries and minimising the response of the body's systems to stimuli such as pathogenic gas bubbles[45]. The exposure time should allow for repeated dissolution of the free gaseous phase in body fluids, transport of the dissolved gas through blood to the lungs and its evacuation outside the respiratory tract.

The assumption is that the combination of pressure and time should lead to the restoration of homeostasis and enable adequate decompression. Oxygen can be used as a factor intensifying homeostasis restoration processes and accelerating the decompression process. Appropriate hydration, pharmacological reduction of blood coagulation or administration of symptomatic drugs are conditions conducive to obtaining homeostasis. In the opinion of some organisations, the administration of standard drugs may be carried out by a diving supervisor or the divers themselves, including injections.

The TT system of therapeutic tables proposed for the Polish Navy will not be quoted here, as it has been described earlier [27].

Increasing the operating capacity of the Polish Navy in the area of underwater works is connected with numerous intersecting projects in the following areas: investment, training, logistics, etc.

Investments must concern both diving equipment for underwater works and diving safety equipment, the selection of which is based on a risk analysis based on the established quality standard. Necessary training is related to the acquisition by divers and security personnel of appropriate knowledge and related qualifications, and, as before, they result from the risk analysis based on the preset quality standard. The same standard results in a range of training activities to ensure continuous combat readiness, which includes maintaining divers in good condition and the fitness of security personnel to carry out underwater operations.

Risk analysis based on the assumed quality standard is also the starting point for securing the appropriate logistic chain for all projects: investment, training and practice.

In order to establish and maintain the capacity to conduct independent deep and saturation dives, it is necessary to significantly expand the existing system for conducting underwater works in the Polish Navy.

The above simplified analysis of the identified problem shows that before commencing the investment process, a preliminary multi-optional risk analysis should be carried out in relation to the planned expansion of operational capabilities, related to the development of competencies based on a defined minimum quality standard. Otherwise, in the investment process related to the development of a complex system, there is a high probability that incompatibilities will occur, which will result in wastage associated with unnecessary redundancy of system elements or the emergence of system weaknesses, threatening to complicate the process of maintaining the capacity to carry out underwater works.

One of the elements of risk analysis, for which a quality standard should be established, is a critical review of the system for securing hyperbaric treatment of cases of decompression sickness.

With current ambitions to significantly increase the capacity to carry out underwater works, the Polish Navy must consider returning to earlier hyperbaric treatments using saturation procedures. These methods should constitute the first series of hyperbaric treatment procedures in cases of sudden decompression[46] during Trimix Tx or Heliox Hx independent deep dives outside the saturation zone and cases of an occurrence of DCS symptoms following saturation diving. For example, the system of therapeutic tables TT can be modelled on that implemented by COMEX or NOAA [14,22]. These procedures are also extremely useful for rescuing the crews of submarines that have been subject to an increase in pressure and, as a result, saturation of the crew, as well as rescuing victims who are trapped in the wreckage of a sunken surface platform and have managed to survive in air pockets[47] [27].

To a great extent, the Polish Navy is technically prepared to re-introduce TT for long-term DCS treatment methods, thanks to the successive modernisation of the hyperbaric complexes we operate. Additional equipment improving the implementation and the use of long-term hyperbaric treatment methods is already prepared in the form of proven technology demonstrators or elements of a trial series which are ready to be implemented. In terms of technology, the production of uniform components can be launched through a spin-off company established by employees of the Naval Academy or in the form of a license transferred to the manufacturer preferred by the Polish Navy.

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[1] *Decompression Sickness,*
[2] Treatment Tables,
[3] speeding up,
[4] „diver ejection”,
[5] for example nitrogen,
[6] full saturation of tissues with gases remaining in dynamic balance with the hyperbaric respiratory atmosphere,
[7] not necessarily the same tissue,
[8] otherwise known as blood circulation, perfusion is the flow of blood through a tissue or organ, usually defined as a percentage of the volume of cardiac output in a time unit,
[9] nitrogen ventilation with oxygen,
[10] which is the increase in the gradient between the gas pressure in the blood and the partial pressure in the alveoli of the lungs,
[11] the ability of a living organism to maintain a relatively stable state of balance through appropriate coordination and regulation of life processes,
[12] even in passenger aircraft it is allowed to reduce the cabin pressure for reasons of durability - airplanes can be designed for smaller pressure changes, hence their structure may be lighter,
[13] unless this transport was planned in the decompression procedure,
[14] in accordance with generally accepted guidelines inside passenger aircraft cabins the pressure must be maintained at a level not lower than that corresponding to 2438 m above sea level [28],
[15] assumed as permitted saturation in the decompression programme,
[16] for example, after *Hx/Tx* dives within the saturation zone, the rest period on the surface needs to be relatively long, as counter-diffusion may increase the inert gas charge in the tissues under normobaric conditions,
[17] in relation to previously used breathing mixes,
[18] according to a mechanism similar to the saline effect in aqueous solutions,
[19] Decompression Sickness,
[20] neurolemma is produced by the cells surrounding the axons of nerve cells and fulfils the function of mechanical protection and electrical insulator,
[21] *complement* is a combination of plasma proteins participating in the body's defensive reactions,
[22] the central nervous system does not tolerate being deprived of the flow of information, therefore its action quickly fades, thus inducing the symptoms of II-DCS,
[23] solvent diffusion through a semi-permeable membrane separating two solutions of a different concentration,
[24] natural solubility is here understood as a state in which the gases occur independently,
[25] a mechanism similar to the 'saline effect' in aqueous solutions,
[26] hence the term ‘sterile bone necrosis’,
[27] gas embolism,
[28] perfused,
[29] resulting in the overburdening of joints or mechanical injuries occurring during work,
[30] the capacity of the living organism to maintain a relatively stable state of equilibrium through appropriate coordination and regulation of life processes,
[31] *oxygen acceleration,*
[32] *Superficial Isobaric Counterdiffusion,*
[33] *Deep Tissue Isobaric Counterdiffusion,*
[34] *Isobaric Counterdiffusion,*
[35] however this refers only to medium workload,
[36] this process has been earlier described in more detail [20],
[37] approx. $24\ hours$,
[38] the most common therapeutic procedures are associated with repeated compression,
[39] performing technically advanced therapeutic procedures,
[40] of course, depending on the patient’s condition,
[41] injuries, fractures, burns, etc.,
[42] including subsystems for atmosphere regeneration and temperature maintenance,
[43] *Life Support Systems,*
[44] this is true in most cases immediately after the accident, where no fractures, burns, wounds, etc. have occurred,
[45] recognised by the immunological system as an aggressor,
[46] „diver ejection”,
[47] „air cushions”,
[48] in alphabetical order.