Decompression illness treated at the Geneva hyperbaric facility 2010–2016: A retrospective analysis of local cases

Julian Thaler[1], Rodrigue Pignel[2], Marie-Anne Magnan[2], Michel Pellegrini[2], Pierre Louge[2]

[1] *Department of Intensive Care Medicine, Valais Hospital, Sion, Switzerland*
[2] *Hyperbaric Medicine, Geneva University Hospitals, Geneva, Switzerland*

Corresponding author: *Dr Julian Thaler, Department of Intensive Care Medicine, Valais Hospital, Avenue du Grand-Champsec 80, 1951 Sion, Switzerland*
julianmatthias.thaler@hopitalvs.ch

Key words
Diving; Decompression illness; Arterial gas embolism; Epidemiology; Scuba; Recompression

Abstract
(Thaler J, Pignel R, Magnan M-A, Pellegrini M, Louge P. Decompression illness treated at the Geneva hyperbaric facility 2010–2016: A retrospective analysis of local cases. Diving and Hyperbaric Medicine. 2020 December 20;50(4):370–376. doi: 10.28920/dhm50.4.370-376. PMID: 33325018.)
Introduction: The Geneva hyperbaric chamber is the main treatment centre for decompression illness (DCI) in Switzerland. The characteristics, symptomatology, treatment and short-term outcome of divers treated at this chamber have not previously been investigated.
Methods: This was a retrospective study of patients treated with hyperbaric oxygen (HBO) for DCI from 2010 to 2016. Data were analysed to provide a description of the cases and statistical analysis for possible factors associated with an unfavourable outcome.
Results: One hundred and thirty-five patients were treated for DCI. Ninety-two were included in the study. Sixty-four presented with neurological and 28 with mild DCI. One hundred and thirty-five patients were treated for DCI. Ninety-two were included in the study. Sixty-four presented with neurological and 28 with mild DCI. Patients with mild DCI mainly had musculoskeletal symptoms (79%). Patients with neurological DCI mainly had spinal (55%), followed by vestibular (36%) symptoms. Arterial gas embolism was diagnosed in 30% of cases. Diving depths ranged between 15 and 142 metres, and dive times between two and 241 min. Median time to treatment was 6 h. Patients with neurological DCI had a high rate (25%) of persisting deficits after treatment. Older age was associated with an unfavourable outcome in univariate but not in multivariate analysis. No adverse effects of HBO were observed. For spinal DCI, a high Boussuges score was associated with persisting deficits after treatment.
Conclusions: Our findings are consistent with other series. Severe DCI was associated with a high rate of persisting deficits. No single factor was associated with a negative outcome. A Boussuges score > 7 had sensitivity of 90% and positive predictive value 53% for predicting an unfavourable outcome in spinal DCI.

Introduction

Despite its landlocked geographical situation, scuba diving is a popular sport in Switzerland with an overall low number of reported injuries. However, cases of decompression illness (DCI) requiring recompression therapy are reported regularly (10–29 cases per year).[1] In Switzerland, there are currently only two hyperbaric facilities: the main chamber (altitude 375 metres above sea level) located at HUG (University Hospitals of Geneva) with another chamber in Basel in private practice. Most of the patients with DCI are treated at the chamber in Geneva, but sometimes divers are treated in Basel (about 0–6 cases/year). The purpose of this study was to describe the cases of DCI treated at the HUG chamber and to investigate the influence of potential risk factors associated with a poor outcome. This study was the first investigation of a case series of patients treated at the HUG chamber and the first one in Switzerland looking at outcomes and potentially associated risk factors.

Methods

The study was approved by the local ethics committee (swissethics). A letter of non-objection to the use of their data for the purposes of this research was sent to each patient.

This was a retrospective, descriptive study of DCI cases treated with hyperbaric oxygen (HBO) at the HUG chamber. Patients that were treated for DCI between 2010 and 2016 and who did not refuse consent were included in the survey.

The year 2010 was chosen as a starting point as it was the first year with full implementation of an electronic medical record system, and 2016 was the end point for internal logistical reasons. Medical records were reviewed and the following data extracted: age, gender, month and place of incident, delay between surfacing and first symptoms, delay between first symptoms and hyperbaric treatment, breathing gas used, maximum depth, dive time, single or repetitive dive, emergency events during the dive, symptoms and classification of DCI (mild or severe neurological).

The presumed pathophysiological mechanism was evaluated for each case: inadequate elimination of nitrogen (classically regarded as 'decompression sickness' [DCS]) versus the introduction of bubbles to the arterial circulation by pulmonary barotrauma (arterial gas embolism [AGE]). We calculated the Boussuges score of severity for neurological DCI with spinal involvement.[2] We extracted the following data on treatment and outcome: treatment applied before HBO (if any), type and number of hyperbaric treatment sessions applied, response after the first HBO treatment, total treatment days and early outcome (in particular, any persisting neurological deficits after the final HBO treatment).

Cases in the final dataset were divided into two groups: The first group consisted of patients with mild DCI presenting with fatigue, joint/muscle pain ('bends') or skin rash. Divers with isolated patchy tingling in the skin as well as mottled or marbled skin rash without an objective neurologic change were also included in this group given their good prognosis even if possibly of 'neurological' origin.[3] The second group consisted of patients with severe DCI, all of them presenting with neurological deficits. A similar classification was used in a previous case series.[4]

Cases per year, the geographic location of dive sites, seasonal occurrence and dive parameters were considered. Mistakes or procedural errors or events contributory to the 'probability of injury' were reviewed. The ratio of mild and severe DCI, the most frequent main symptoms and whether pharmacologic treatment was performed prior to arrival at the chamber were evaluated. Univariate analyses for associations between dive and diver characteristics and mild or severe DCI were performed.

At the end, we analysed the cases for 'outcome' by type of DCI (mild vs. severe), the 'Boussuges score' in cases of spinal neurological DCI and the percentage of persisting neurological deficits after the last treatment session.

Logistic regression analysis was applied for a negative outcome (persistent symptoms after final treatment session) in the group of neurological DCI cases for the following covariables: sex, age, single or repetitive dive, interval from surfacing to first symptoms as well as interval from first symptoms to treatment. All mild DCI cases had a complete resolution of symptoms and were therefore not analysed further.

STATISTICAL ANALYSIS

The Shapiro-Wilk test was used for assessing the assumption of normality. Data were expressed as mean (normally distributed data) or median with a range for non-normal data. Fisher's exact test and the Mann Whitney U-test were used for group analysis. We performed univariate as well as multivariate analysis of several risk factors for a negative outcome. Statistical significance level was set at $P < 0.05$. Statistical analyses were performed using jamovi for MAC, version 1.08, retrieved at https://www.jamovi.org/ using R: A language and environment for statistical computing by R Core Team (2018).

Results

One hundred and thirty-five patients with suspected or diagnosed DCI were treated in a range from 10 cases (2011) to 30 cases (2015) per year (Figure 1). Forty-three were excluded from the final analysis. In 18 patients treatment was conducted in a rather speculative manner after hazardous hyperbaric exposures without symptoms of DCI. These were excluded from further analysis as no clear diagnosis of DCI was made. Twenty travellers who had suffered from DCI abroad and were referred to our unit on return were also excluded from further analysis as most of the patients had already been treated with HBO. Two professional tunnel workers were excluded. Finally, there were 3 refusals of data disclosure (Figure 2).

Figure 1
Number of divers treated with HBO for DCI per calendar year over the study period

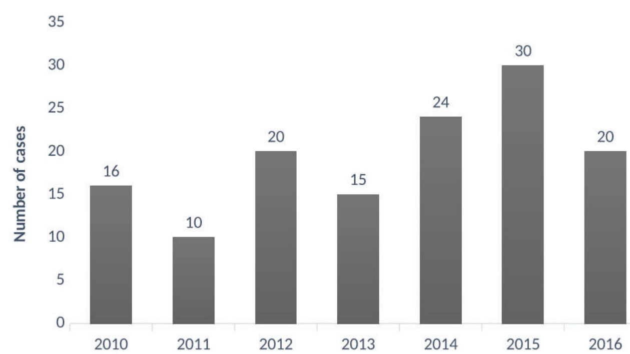


Figure 2
Flowchart explaining selection of patients presented in this study; DCI = decompression illness

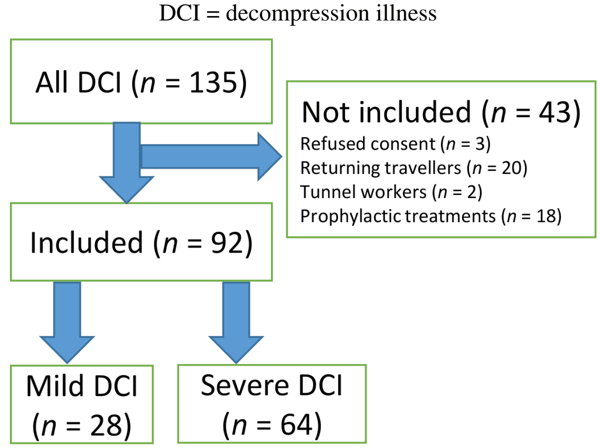


Table 1
Dive parameters for 92 divers presenting with decompression illness

Dive parameters	
Maximum depth (mfw), Median (range)	45.5 (18–142)
Dive time (min), Median (range)	41 (2–241)
Dive modality (scuba/rebreather/apnoea) *n*	89 / 2 / 1
Breathing mixture (air/nitrox/trimix) *n*	80 / 4 / 8
Normal dive/Emergency event *n* (%)	66 (71%) / 26 (29%)
Single dive/Repetitive dive *n* (%)	72 (78%) / 20 (22%)

Table 2
Diver and dive characteristics with divers stratified into mild or severe DCI. Data are median (range) unless otherwise indicated. mfw = metres' freshwater

Parameter	**Mild DCI (*n* = 28)**	**Severe DCI (*n* = 64)**	**All (*n* = 92)**	***P***
Age, mean (range)	41 (16–68)	45 (21–81)	44 (16–81)	0.09
Male sex, *n* (%)	24 (85%)	59 (92%)	83 (90%)	0.44
Maximum depth (mfw)	45 (15–130)	46 (18–142)	46 (15–142)	0.99
Dive time (min)	44 (10–120)	40 (2–241)	41 (2–241)	0.42
Repetitive dive, *n* (%)	7 (25%)	13 (20%)	20 (22%)	0.59
Time surfacing – symptoms (min)	60 (0–600)	15 (0–780)	30 (0–780)	0.39
Time symptoms – HBO (min)	360 (30–7,200)	310 (76–5,760)	360 (30–7,200)	0.64

There were 92 divers retained in the final dataset. The mean age of males (*n* = 83) was 44 y (range 16–81) and females (*n* = 9) 41 y (range 21–68). Most injuries occurred during summer months in Switzerland (85%), with a further 8% in France (7% of dive sites unknown). Dive parameters are shown in Table 1.

Self-reported emergency events were noted in 27 (29%) of cases, most commonly omission of decompression obligations in 13 (14%). Unplanned rapid ascents, often due to loss of buoyancy control, malfunctioning or free flowing regulator/dry suit inflator, or while helping distressed dive buddies, were less common (10 divers, 11%). The other four divers reported stress events (lost buddy, cold and equipment problems) without rapid ascents.

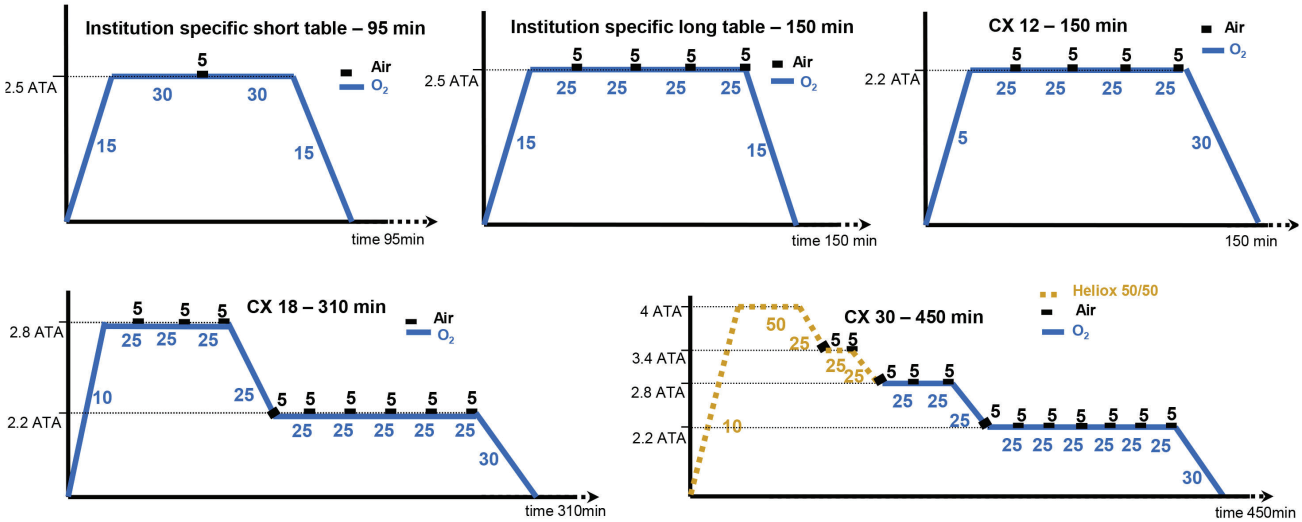
Patients presented with mild DCI in 28 cases (30%) and severe neurological DCI in 64 cases (70%). Patients with mild DCI presented mainly with diffuse muscular and/or periarticular pain (76%) and, to a lesser extent, with cutaneous symptoms (16%) and fatigue (8%). Neurological DCI patients presented mainly with spinal symptoms (55%), vestibular symptoms (34%) or cerebral/cerebellar symptoms (11%). AGE as probable pathophysiological mechanism was diagnosed in 19 (30%) of the latter cases presenting with neurological deficits. The diagnosis of AGE was made in patients with a combination of low gas load, rapid ascent and early onset of symptoms. Computed chest tomography scanning for the detection of a pulmonary barotrauma, blebs or bullae was not systematically performed in these cases.

Median time to first symptoms after surfacing was 30 (range 0–780) min. Median time between first symptoms and hyperbaric treatment was 360 (range 0–7,200) min. Delays between surfacing and first symptoms as well as delays between first symptoms and hyperbaric treatment were shorter in the group presenting with severe DCI but this was not statistically significant. Furthermore, there was no association between age, sex, dive time, maximum depth, and type of DCI (mild versus severe) in univariate analysis. Divers and dives characteristics in regard to the type of DCI are shown in Table 2.

Concerning pre-hospital treatment, either oral or intravenous hydration was used in 48 (52%) of cases (mild as well as neurologic DCI). Twenty-nine (32%) of the patients received acetylsalicylic acid.

Treatment protocols utilised at our institution at the discretion of the responsible hyperbaric physician were

Figure 3
Recompression tables used at HUG. ATA = atmospheres absolute pressure (1 ATA = 101.3 kPa)



Comex (CX, Compagnie maritime d`expertises) tables: CX30 Heliox (7 h 30 min overall run time with a maximal pressure of 405 kPa breathing Heliox 50%), CX18 (5 h 10 min overall run time with a maximal pressure of 284 kPa), CX12 (2 h 30 min overall run time with a maximal pressure of 223 kPa). Additionally, institution specific HBO tables (short tables of 95 min at 253 kPa or long tables of 150 min at 253 kPa with air breaks) were used, mainly for consolidation treatment sessions (treatment tables used are shown in Figure 3).

Hyperbaric treatments were run until there was no further benefit over two consecutive treatments (in severe cases two times daily, in less severe cases once a day, weekends included). For patients with mild DCI initial recompression tables used were predominantly CX12 (21, 75%) or the institution specific tables. CX18 and CX30 Heliox were used for more symptomatic patients who had conducted deeper dives (7, 25%). Thirty patients (47%) with severe DCI were treated with CX18, institution specific long tables (9, 14%) or CX30 Heliox tables (25, 39%). Initial worsening despite hyperbaric treatment occurred in seven of the severe DCI patients. All of them had neurologic DCI occurring after deep (mean 70 mfw) bounce dives. Four of these patients were initially treated with a CX18 table. For these four patients with worsening of symptoms, the CX18 table was interrupted after 15–30 minutes and escalated to a CX30 Heliox table with success. The other three patients were already on a CX30 table.

Severe DCI cases had a significantly higher number of treatment sessions (median 5, range 1–55) than patients with mild DCI (median 2, range 1–4) due to severity and longer time to resolution of symptoms. There was no relation between longer dive time or deeper depth and the number of treatment sessions needed for either group.

As expected, there was a strong and significant correlation between the severity of DCI and outcome as all mild DCI cases had a complete recovery (OR for negative outcome: 19.8; $P = 0.002$) whereas 16/64 (25%) of patients in the severe DCI group had a negative outcome with persistent deficits after the final treatment session. Persistent deficits included hypaesthesia/dysaesthesia, residual vestibular syndrome, urinary sphincter troubles and severe disabilities such as limb paresis, paraparesis, para- and tetraplegia. Most commonly reported were motor weakness/paresis (7/16) and sphincter troubles (6/16 cases). For severe DCI with spinal medullary injury, a Boussuges score of > 7 was significantly associated with a negative outcome in univariate analysis (Table 3).

In univariate analysis of the severe DCI group, older age was significantly associated with worse outcome (OR 3.86); however, in logistic regression analysis this could not be reproduced. In multivariate analysis no single factor was significantly associated with a negative outcome in severe DCI (Table 4). No adverse effect of recompression therapy occurred during any of the treatment sessions.

Table 3
Boussuges scale analysis in spinal cord DCI. When stratified by scores > 7 or ≤ 7 this system exhibited the following characteristics: sensitivity 90%, specifity 58%, positive predictive value 53%, negative predictive value 92%

Boussuges score	Sequelae	No sequelae	Total
≤ 7	1	11	12
> 7	9	8	17
Total	10	19	29

Table 4

Correlation of risk factors and adverse outcome in severe DCI cases. CI = confidence interval; HBO = hyperbaric oxygen; mfw = metres' freshwater; OR = odds ratio

Comparison	Univariate analysis		Multivariate analysis	
	OR (95% CI)	*P*	OR (95% CI)	*P*
Age (≥ 45 vs. < 45 y)	3.86 (1.09–13.70)	0.04	1.03 (0.97–2.82)	0.27
Female vs. male	0.57 (0.06–4.80)	0.60	0.58 (0.5–5.88)	0.65
Repetitive vs. single dive	0.42 (0.08–2.19)	0.48	0.46 (0.08–2.44)	0.36
Surface to first symptoms (> 60 vs. ≤ 60 min)	0.21 (0.04–1.03)	0.06	0.97 (0.95–1.00)	0.08
Symptoms to HBO (> 360 vs. ≤ 360 min)	0.27 (0.06–1.10)	0.07	1.0 (0.99–1.00)	0.49
Depth (> 40 vs. ≤ 40 mfw)	0.83 (0.25–2.70)	0.76		
Dive time (> 60 vs. ≤ 60 min)	1.22 (0.32–4.61)	0.74		
HBO treatments (> 6 vs. ≤ 6)	24.5 (5.6–100.5)	< 0.01		
Mild vs. severe DCI	19.8 (1.2–336)	< 0.01		

Discussion

The number of DCI cases treated in this seven-year period is comparable to other centres situated in Central Europe.[4] In our local catchment area, more patients were treated during the summer months, likely due to the favourable weather conditions in Swiss lakes and the summer holiday season. Most accidents took place in Switzerland. As Geneva is situated at the French border with popular diving sites nearby (Lac Leman, Lac d`Annecy) several French divers were treated as well. Switzerland is a mountainous country: all diving done is done at > 300 m altitude and is formally 'altitude diving'; however, our divers mostly dived at moderate altitude (for example Lac Leman 372 m, Thunersee 558 m, Lac Neuchâtel 429 m). It is unknown if this affects the incidence of DCI compared to other locations.

Emergency events (mostly omitted decompression obligations or rapid ascents) were reported in 27 (29%) of the 92 cases. Other analyses have found a comparable number of human errors associated with diving accidents.[5] In contrast to other dive sites, most dives were performed using a drysuit because of the cold water temperatures year-round, which is a risk factor for rapid ascents as drysuit diving increases task loading. The rapid ascents reported by several divers were mostly due to drysuit issues. However, we could not collect enough data for a specific analysis. In those cases with technical problems, it remains unclear whether redundant equipment was carried, which is of high importance when diving in cold water as there may be an elevated risk of free flowing or freezing regulators.

Arterial gas embolism (AGE) was diagnosed in 28 (30%) of divers, mostly in divers who did not have a high tissue gas load to explain the symptoms such as in most of the cases with sudden rapid ascents. However, making a firm diagnosis of AGE or DCS as causative mechanisms based on symptoms is often difficult, and the two frequently coexist.[6] Additionally, as explained before, systematic chest CT scanning or other investigations to help differentiate between the two were not consistently performed.

Of the cases treated 64/92 (70%) were severe DCI cases, in contrast to other publications where mild DCI was the predominant type.[4] At the moment, we cannot discern the reasons and there are no data available on under reported untreated cases of mild DCI in Switzerland. Univariate analysis of mild versus severe DCI cases revealed no significant differences between the dive or divers' profiles.

The number of treatment sessions differed significantly between mild and severe DCI reflecting our policy of repeated treatment sessions until no significant improvement occurred on two consecutive treatments (in concordance to international practice).[7] This reflects our knowledge that during the treatment of DCI pain typically resolves much faster than neurological symptoms.[8] Treatment schedules for mild and neurologic DCI vary between institutions and individual hyperbaric physicians. Our approach (shorter and shallower tables for mild DCI, deeper and longer tables ± heliox for severe DCI) seems to be in accordance with international practice, even if there is a lack of scientific evidence as to the optimal selection of therapeutic tables.[9] For mild illness or follow-on treatments, tables like our institution specific tables, with frequent variations, are used in other European institutions as intermediate protocols between CX12 and CX18 tables.

No adverse effects of recompression therapy were reported in our series whereas in recent literature, up to 17.4% of patients experienced adverse events.[10] Strict operational protocols and in-chamber monitoring (gas pressures, camera observation, highly qualified attendants) are applied in our centre and may have contributed. One third of the patients received aspirin preclinically, usually given by French emergency providers as this is a part of their treatment algorithm.[11] At HUG no specific pharmacologic treatment was used, as good scientific evidence is lacking.

As expected, there was a strong and significant correlation between the severity of DCI and outcome as all mild DCI cases had a complete recovery (OR for negative outcome if severe:19.8, $P = 0.002$) and 16/64 (25%) of neurologic DCI patients had sequelae after the last hyperbaric session. For different reasons, we did not consistently follow up or interview the patients at a later time, especially those with sequelae, which is a limitation of the study. In the recent literature, a quite similar percentage (33%)[12] of incomplete resolution after spinal cord DCI was reported with other authors finding between 22% and 61%.[13,14] In univariate analysis of risk factors, older age (> 45 years) was associated with a negative outcome (Table 4). Older age is often considered to be a risk factor for decompression sickness itself.[15,16] However, in multivariate analysis applied to reveal variables associated with a poor outcome in severe DCI, none, including age, reached significance.

Delayed (i.e., > 6 hours) commencement of treatment was not associated with a worse outcome in this case series; however, our study was not designed for specifically answering the question of effect of delay to HBO. In other studies, delay to hyperbaric treatment was associated with a less favourable outcome in neurological DCI.[17,18] These findings may make the case for in-water recompression in remote locations.[19]

In this study, median time from first symptoms to HBOT was 6 hours due to a great many of our patients presenting late after the first symptoms and probably also as a result of the geographical position of our hospital at the southwestern edge of Switzerland, prolonging transport times even if helicopter evacuation to a hyperbaric facility can theoretically be achieved in a time frame of less than one hour all over the country.[20] A recent study showed that delayed treatment > 48 hours was nonetheless effective in reducing the symptoms of DCI.[21]

The Boussuges score takes into account objective neurological deficits and the course of illness (stability or deterioration) on admission.[2] For spinal DCI, there was a significant association between a high Boussuges score (> 7) and persistent neurological deficits after the completion of the final treatment session with 90% sensitivity, however specificity (58%) was lower than in other case series.[2,22] Because of the low number of cases of spinal DCI where data allowing a Boussuges classification was available (29 patients) no multivariate analysis was conducted.

Conclusions

Of 135 divers presenting to the Geneva hyperbaric centre over a seven-year period, 92 were analysed for risk factors for mild versus severe DCI and negative outcome defined as persisting neurologic deficits after the last treatment session. No differences in the dive parameters were associated with mild versus severe DCI. Median delay between first symptoms and treatment was 6 h. Comex treatment tables were successfully used alongside HUG tables instead of US Navy tables. No adverse effects of recompression therapy were reported.

All patients presenting with mild symptoms made a full recovery, whereas 16 of 64 (25%) of patients with severe DCI showed a persistent neurological deficit after the last HBOT session. Our results are comparable with those of other reported case series.

In univariate analysis, older age was significantly associated with a worse outcome. However, in multivariate analysis no single risk factor was significantly associated with a negative outcome. Because of the rather large number of variables analysed for a small sample of a selected patient group, conclusions should be drawn with caution. The study is not sufficiently powered to rule out risk factors, which is one of the limitations. Other limitations are the retrospective design with a selected group of subjects and poor control over the exposure factors, covariates and potential confounders. A Boussuges score > 7 was associated with an unfavourable outcome in spinal DCI in this series.

Finally, our survey shows that despite adequate HBOT, neurological DCI is still associated with significant morbidity and a high rate of persistent deficits.

References

1 FTU/DAN EUROPE Suisse. Tauchunfälle - Fallsammlunge 2017. Zürich: FTU/DAN; 2018. Available from: https://ftu.ch/de/downloads/statistiken/ftu-unfallberichte/test3.pdf. [cited 2018 Dec 10]. German.

2 Boussuges A, Thirion X, Blanc P, Molenat F, Sainty JM. Neurologic decompression illness: A gravity score . Undersea Hyperb Med. 1996;23:151–5. PMID: 8931282.

3 Germonpré P, Balestra C, Obeid G, Caers D. Cutis Marmorata skin decompression sickness is a manifestation of brainstem bubble embolization, not of local skin bubbles. Med Hypotheses. 2015;85:863–9. doi: 10.1016/j.mehy.2015.09.022. PMID: 26432631.

4 Kot J, Sićko M, Michałkiewicz M, Lizak E, Góralczyk P. Recompression treatment for decompression illness: 5-year report (2003–2007) from national centre for hyperbaric medicine in Poland. Int Marit Health. 2008;59:69–80. PMID: 19227740.

5 O'Connor P, O'Dea A, Melton J. A methodology for

identifying human error in U.S. Navy diving accidents. Hum Factors. 2007;49:214–26. doi: 10.1518/001872007X312450. PMID: 17447664.
6 Neumann T. Arterial gas embolism and decompression sickness. News Physiol Sci. 2002;17:77–81. doi: 10.1152/nips.01370.2001. PMID: 11909997.
7 UHMS Best Practice Guidelines, 2011 April 04. [cited 2020 Feb 25]. Available from: https://www.uhms.org/images/DCS-AGE-Committee/dcsandage_prevandmgt_uhms-fi.pdf.
8 Moon RE, Gorman DF. Treatment of the decompression disorders. In: Brubakk AO, Neuman TS, editors. Bennett and Elliott's physiology and medicine of diving, 5th ed. Edinburgh: Saunders; 2003. p. 600–50.
9 Antonelli C, Franchi F, Della Marta ME, Carinci A, Sbrana G, Tanasi P, et al. Guiding principles in choosing a therapeutic table for DCI hyperbaric therapy. Minerva Anestesiol. 2009;75:151–61. PMID: 19221544.
10 Hadanny A, Meir O, Bechor Y, Fishlev G, Bergan J, Efrati S. The safety of hyperbaric oxygen treatment retrospective analysis in 2,334 patients. Undersea Hyperb Med. 2016;43:113–22. PMID: 27265988.
11 Bessereau J, Coulange M, Genotelle N, Barthélémy A, Michelet P, Bruguerolle B, et al. Aspirin in decompression sickness. Therapie. 2008;63:419–23. doi: 10.2515/therapie/2008067. PMID: 19236833.
12 Gempp E, Blatteau JE. Risk factors and treatment outcome in scuba divers with spinal cord decompression sickness. J Crit Care. 2010;25:236–42. doi: 10.1016/j.jcrc.2009.05.011. PMID: 19682840.
13 Aharon-Peretz J, Adir Y, Gordon CR, Kol S, Gal N, Melamed Y. Spinal cord decompression sickness in sport diving. Arch Neurol. 1993;50:753–6. doi: 10.1001/archneur.1993.00540070065017. PMID: 8323480.
14 Ball R. Effect of severity, time to recompression with oxygen, and retreatment on outcome of forty-nine cases of spinal cord decompression sickness. Undersea Hyperb Med. 1993;20:133–45. PMID: 8329940.
15 Desola J, Sala J, Bohe J, Garcia A, Abos R, Canela J. Prognostic factors of dysbaric disorders. Evidence-based conclusions after a multivariant analysis of 554 cases. In: Cali-Corleo R, editor. Proceedings of the 26th Annual Meeting of the European Underwater and Baromedical Society. Valetta, Malta: EUBS; 2000. p. 17–23.
16 Vann RD. Mechanisms and risks of decompression. In: Bove AA, editor. Bove and Davis' diving medicine, 4th ed. Saunders. 2004:127–64. doi: 10.1016/B978-0-7216-9424-5.50013-7.
17 Blatteau JE, Gempp E, Simon O, Coulange M, Delafosse B, Souday V, et al. Prognostic factors of spinal cord decompression sickness in recreational diving: retrospective and multicentric analysis of 279 cases. Neurocrit Care. 2011;15:120–7. doi: 10.1007/s12028-010-9370-1. PMID: 20734244.
18 Blatteau JE, Gempp E, Constantin P, Louge P. Risk factors and clinical outcome in military divers with neurological decompression sickness: Influence of time to recompression. Diving Hyperb Med. 2011;41:129–34. PMID: 21948497.
19 Doolette DJ, Mitchell SJ. In-water recompression. Diving Hyperb Med. 2018;48:84–95. doi: 10.28920/dhm48.2.84-95. PMID: 29888380. PMCID: PMC6156824.
20 Steffensmeier D, Albrecht R, Wendling J, Melliger R, Spahn DR, Stein P, Wyss C. Specialist advice may improve patient selection for decompression therapy following diving accidents: a retrospective observational study. Scand J Trauma Resusc Emerg Med. 2017;25:101. doi: 10.1186/s13049-017-0447-0. PMID: 29052534. PMCID: PMC5649053.
21 Hadanny A, Fishlev G, Bechor Y, Bergan J, Friedman M, Maliar A, et al. Delayed recompression for decompression sickness: retrospective analysis. PLoS One. 2015;10(4):e0124919 doi: 10.1371/journal.pone.0124919. PMID: 25906396. PMCID: PMC4408070.
22 Pitkin AD, Benton PJ, Broome JR. Outcome after treatment of neurological decompression illness is predicted by a published clinical scoring system. Aviat Space Environ Med. 1999;70:517–21. PMID: 10332950.

Acknowledgements

We would like to thank the hyperbaric technicians and nurses of the HUG.

Conflicts of interest and funding: nil

Submitted: 11 February 2020
Accepted after revision: 16 August 2020