

Initial Severity Scoring and Residual Deficit in Scuba Divers with Inner Ear Decompression Sickness

Emmanuel Gempp; Pierre Louge; Sébastien de Maistre; Jean-Baptiste Morvan; Nicolas Vallée; Jean-Eric Blatteau

- BACKGROUND:** Inner ear decompression sickness (IEDCS) in scuba diving results in residual vestibulocochlear deficits with a potential impact on health-related quality of life. The aim of this study was to determine the predictive factors for poor clinical recovery and to try to establish a prognostic score on initial physical examination.
- METHODS:** The medical records of injured divers with IEDCS treated in our facility between 2009 and 2014 were retrospectively analyzed. The clinical severity of the deficit was evaluated on admission using a numerical scoring system taking into account the intensity of vestibular symptoms and the presence of cochlear signs. The clinical outcome was assessed at 3 mo by telephone interview. After multivariate analysis of potential risk factors for sequelae, the discriminating value of the score and these prognostic reliability indices were calculated.
- RESULTS:** Among the 99 patients included in the study, 24% still had residual symptoms. Statistical analysis revealed that only a high clinical score [OR = 1.39 (95% CI 1.13-1.71)] and a delay in hyperbaric recompression >6 h [OR = 1.001 (95% CI 1-1.003)] were independently associated with incomplete recovery. The advantage of the score lay in its highly specific nature (92%) rather than its sensitivity (48%) for a threshold of 10.
- CONCLUSION:** Results suggest that the severity of IEDCS can be easily determined by a clinical score during the acute phase. Recompression treatment should not be delayed.
- KEYWORDS:** inner ear, decompression sickness, scuba diving, hyperbaric oxygen therapy.

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The usual formation of tissue and vascular bubbles during the ascent from a dive exposes the diver to the risk of decompression sickness (DCS) after surfacing when their presence becomes excessive and aggressive for the body. The incidence of DCS is estimated at 1–3 cases/10,000 dives,¹⁵ of which 25% are vestibulocochlear in origin.^{2,7} In fact, the inner ear is a target organ which is particularly sensitive to this type of injury, either by in situ degassing in the endolymphatic and perilymphatic fluids or by a gaseous embolism in the arterial vascularization. The symptoms are mainly characterized by vestibular disorders,^{7,9} with an impairment preferentially on the right side.^{7,12} Bilateral lesions are exceptional.¹⁰ The clinical manifestations occur early after the dive, usually in the hour after surfacing and, in 15% of cases, there may be associated neurological or cutaneous symptoms.^{7,9,12} The presence of a large right-to-left circulatory shunt (mainly patent foramen ovale) is found in three-quarters of cases.^{7–9} Despite early, appropriate treatment with hyperbaric oxygen therapy (HBOT) and good functional recovery by central vestibular compensation in the 6 mo following the injury,

audio-vestibular sequelae persist in 70–90% of cases after laboratory evaluation.^{7,10,13} To date, the few scientific reports dealing with the conditions of occurrence, treatment, and follow-up for this type of DCS are mainly based on a limited number of patients with relatively unknown predictors for sequelae.^{8,10,13} The aim of our study was to assess the potential risk factors associated with a poor clinical recovery in a large cohort of divers who were referred for inner ear DCS (IEDCS) in a single institution. At the same time, we also tried to establish a predictive score for residual deficit from the initial clinical presentation.

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METHODS

Subjects

The local Ethics Committee (Sainte Anne's Military Hospital, Toulon, France) approved the study design and all the patients gave their written consent to participate. Between 2009 and 2014, all the cases of IEDCS treated in our hyperbaric facility were retrospectively analyzed using a computerized database which contains standardized information on the diver's characteristics, the parameters and conditions of the dive, the treatment undertaken, and the additional investigations performed. The inclusion criteria were the appearance of acute vestibular symptoms (rotary vertigo, nausea, vomiting) and/or cochlear symptoms (hypacusia, tinnitus) occurring in the 6 h after leaving the water. Patients for whom the clinical presentation was ambiguous and described incompletely in the medical records, or also for whom questioning gave the impression of otalgia or pressure equalization problems of the middle ear when diving, with tympanic abnormalities on otoscopic examination, were excluded from the study. One hundred and six divers presented a clinical picture compatible with an IEDCS during the period considered. After excluding 2 ambiguous cases, which could also have been inner ear barotrauma, and 5 other cases which either presented mild symptoms or did not answer the telephone survey, 99 patients (82 men, 48 ± 11 yr) were finally included in the analysis.

Materials and Equipment

On arrival at the hyperbaric center, the injured divers were clinically assessed by a diving physician from the department using, in particular, a rating scale for the symptoms and clinical signs presented on admission, leading to the determination of an initial severity score (Table I). The diagnosis and topography of the inner ear impairment were confirmed afterwards by pure tone audiometry, dynamic posturography, and videonystagmography with bicaloric testing in the following week. All the patients were given a recompression treatment using oxygen-enriched mixtures (table at 4 ATA for 4 to 6 h) or pure oxygen (table at 2.8 ATA for 2.5 to 5 h) with intravenous methylprednisolone (80 to 120 mg) and, if necessary, acetyl leucine (500 mg), metoclopramide (10 mg), or even aspirin (250 mg) if this had not been administered early before transportation to the hospital. In a very large majority of cases when symptoms persisted after the first hyperbaric treatment, additional sessions of HBOT (1 to 2 sessions per day at 2.5 ATA for 70-90 min) were programmed until the disappearance or distinct improvement in the deficit with a maximum of 10 sessions. In cases of gait instability and residual vertigo, vestibular rehabilitation (rotary chair, unstable platform) was started routinely in hospital in the 2-3 d following the insult, followed by a prescription of additional sessions after discharge.

Procedure

Follow-up clinical evaluation was performed in all subjects by telephone interview 3 mo post-DCS. In 41% of cases, the absence of additional audiometric or videonystagmographic

Table I. Severity Score for Inner Ear Decompression Sickness on Admission.

SYMPTOMS AND SIGNS	COEFFICIENT
Rotary vertigo	
Absent	0
Variable or single vertiginous sensation	1
Permanent	2
Nystagmus	
Absent	0
Present in eccentric gaze in the direction of the fast saccade	1
Present in centered gaze	2
Present in eccentric gaze in the direction of the slow saccade	3
Autonomic symptoms	
Absent	0
Isolated nausea	1
Nausea and rare vomiting, particularly after mobilization	2
Uncontrollable vomiting	3
Gait instability	
Absent	0
Present, eyes closed	1
Present, eyes open	2
Impossible	3
Cochlear symptoms	
Absent	0
Present	8

The coefficients corresponding to the intensity of the signs and symptoms for each item have been determined arbitrarily depending on clinical experience. They are added together to give a maximum score of 19.

testing meant it was not possible to accurately determine the magnitude of the deficit and, consequently, to identify the sequelae solely on the findings of the laboratory investigations. Thus, the main outcome measure of this study was based on the presence of subjective residual symptoms potentially affecting daily living, i.e., auditory disorders (hypo/hyperacusia, tinnitus) and/or vestibular deficit assessed by the European Evaluation Vertigo scale, a validated physician-administered questionnaire analyzing the intensity of five items: motion intolerance, instability, neurovegetative signs, illusion of movement, and duration of illusion.¹¹ The items scaling ranged from 0 (absence) to 4 (severe or very often) and subjects with a score equal or greater than 2 out of 20 over the last 8 d before the interview were considered symptomatic.

Statistical Analysis

The data are expressed as mean \pm SD or the median + interquartile range depending on its distribution evaluated by the Kolmogorov-Smirnov test. The severity of IEDCS (presence or not of residual symptoms) has been treated as a dependent variable, whereas the predictive factors for incomplete recovery, analyzed as quantitative or qualitative explanatory variables, were the following: age, gender, the parameters and circumstances of the dive, the time of onset of symptoms after surfacing, the administration of oxygen during evacuation, the clinical score on admission, the time to recompression, the regimen of therapeutic table used initially, the number of additional sessions of HBOT, and the total number of vestibular rehabilitation sessions. Initially, the variables for each group were compared using the Fischer, Mann-Whitney, or

Student *t*-test, where appropriate, with a significance threshold of $P < 0.05$. The variables with $P < 0.20$ were then retained as covariables in a logistic regression model to take into account potentially confounding factors and to better identify the independent variables for predicting sequelae. As a final analysis, the identification of the threshold value for the severity score to be considered for the prognosis of IEDCS was carried out by establishing a receiver operating characteristic curve combined with the calculation of the area under the curve, sensitivity, specificity, and negative and positive predictive values for the test. All the calculations, particularly the determination of the adjusted odds ratios (AOR) and the confidence intervals at 95% (95% CI) when necessary, were performed using Sigmapstat 3.0 software (SYSTAT Inc., Richmond, CA).

RESULTS

On admission, isolated vestibular impairment was predominant (79% of cases), followed by mixed vestibulocochlear (16%) and then pure cochlear (5%) forms of IEDCS. In 11% of cases, there were additional symptoms evocative of combined DCS with cutaneous, spinal cord, or cerebral involvement. The median time to hyperbaric treatment was 3 h.

At 3 mo, 25 divers (24% of 99 subjects) had incomplete clinical recovery, including 15 out of 25 (60%) with residual vestibular disorders, 8 (32%) with isolated cochlear symptoms,

and 2 (8%) who presented a mixed deficit. Median European Evaluation Vertigo score was 4 (2-5) for those with vestibular impairment. In the subgroup of 58 divers who underwent follow-up investigations, 19 (33%) had totally recovered, 31 (53%) still showed abnormalities on videonystagmography testing, 6 (10.5%) still had an audiometric deficit, and 2 (3.5%) presented a vestibulocochlear injury.

Among the many parameters studied, the univariate analysis showed that a high clinical score on admission, particularly when there were hearing disorders, was significantly associated with the existence of sequelae (Table II). As the intercorrelation between the severity score and the “presence of cochlear symptoms” variable was too great, this has not been included in the multivariate analysis. After adjustment, average age, additional HBOT, and rehabilitation sessions did not reach statistical significance, but the results confirmed the importance of the initial score in the prediction of incomplete recovery [AOR = 1.39 (1.13–1.71); $P = 0.002$] and also the negative influence of a delay to recompression >6 h in the resolution of IEDCS [AOR = 1.001 (1–1.003); $P = 0.008$]. The threshold value of the severity score which best distinguished the divers with deficits for maximum sensitivity and specificity was 10 on the receiver operating characteristic curve (Fig. 1), with an area under the curve of 72% (59–85) ($P = 0.001$) and the following prognostic reliability indices were: sensitivity = 48% (27–68), specificity = 92% (83–97), positive predictive value = 66% (41–86), and negative predictive value = 84% (71–91).

Table II. Univariate Analysis of Variables Potentially Predictive of Residual Deficit in Divers with Inner Ear Decompression Sickness ($N = 99$).

VARIABLES	CURE	DEFICIT	P-VALUE
Age (yr)	49 ± 11	46 ± 10	0.16
Gender (M/F)	60/14	22/3	0.55
Total dive time (min)	41 ± 11	44 ± 12	0.35
Maximum depth (msw)	37 ± 13	35 ± 10	0.40
Omitted decompression, fast ascent			1
Yes	3	1	
No	71	24	
Successive dive			0.63
Yes	25	10	
No	49	15	
Time onset of symptoms (min)	30 [14-60]	30 [8-55]	0.37
Initial cochlear disorder			0.0004*
Yes	9	12	
No	65	13	
Clinical score on admission	6 [4-9]	9 [8-12]	0.0016*
First-aid oxygen during evacuation			0.33
Yes	65	20	
No	9	5	
Pharmacological treatment [§]			0.27
Yes	68	21	
No	6	4	
Initial recompression table			1
Table at 2.8 ATA	66	23	
Table at 4 ATA	8	2	
Additional HBOT sessions (nb)	4 [2-7]	6 [4-8]	0.06
Rehabilitation sessions (nb)	3 [0-7]	6 [0-14]	0.07

[§]Corresponds to the administration of intravenous acetyl-leucine and metoclopramide. *Denotes variables that reached statistical significance. Values are given as mean ± SD or median [IQR].

DISCUSSION

In a large series of divers, our study showed that the prevalence of patients with residual symptoms in the medium term after an IEDCS was 24%, and that they mainly corresponded to feelings of instability in some situations (working at a height, movement), imbalance in the dark or when changing position, and even tinnitus associated with hypoacusia of varying intensity. These results are consistent with those found in a series of 38 divers after hospital discharge (21%)⁵ and are also similar to those of an American study where the proportion of residual functional disorders that had an impact on quality of life was 32% after an IEDCS.¹⁴ Moreover, among the 58 patients who underwent audiometric and vestibular testing, we observed that 67% of cases still had inner

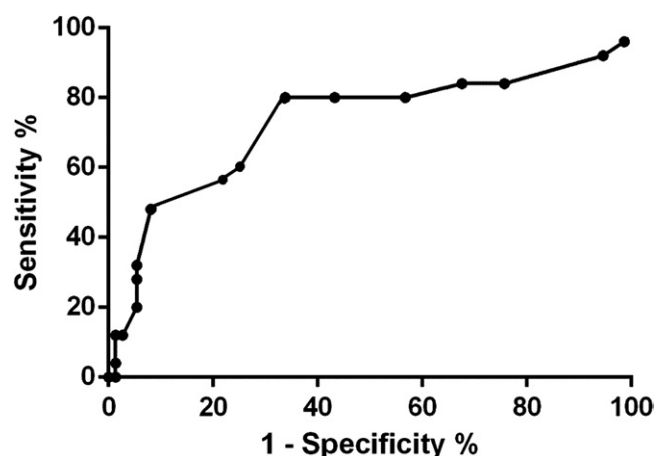


Fig. 1. Receiver operating characteristic curve of the severity score in predicting residual deficit after inner ear decompression sickness. The area under the curve is 72% (59–85).

ear damage shown by laboratory investigations later on. Although lower, this rate corresponds to that found by some authors.^{10,13} The difference could be linked to the shorter median delay in treatment in our series (3 h versus 5 to 10 h).

Based on our review of the existing literature, we believe that this is the first time that a study sought to determine if there were predictive elements for poor recovery after an episode of IEDCS by identifying two independent factors for poor prognosis, i.e., the intensity of the vestibulocochlear symptoms present before hyperbaric treatment and the delay in recompression. It is interesting to note that neither the initial treatment (oxygen during transportation, first recompression table) nor the secondary treatment (additional HBOT, rehabilitation) differed between the two groups, thus limiting the risk of measurement bias. In two previous studies, it was noted that cochlear impairment was a negative condition in recovery from IEDCS, with 40–50% of cases still having functional auditory sequelae.^{7,14} This particular characteristic has justified arbitrarily allocating a weighted value equivalent to 8 in our clinical score in the event of initial hearing loss. The results of our series seem to show that this choice was appropriate, with significant influence of this anatomical impairment in the prognosis for these injuries if reference is made to the results of the univariate analysis. We have shown that a simple severity score, developed from symptoms presented on admission and the initial clinical examination, appeared to be useful in predicting the risk of incomplete recovery with, in particular, a strong probability (84%) of not having sequelae if the score was <10 . However, if one looks at the sensitivity of this score, it must be noted that an injured diver with a value of ≥ 10 has a significant chance of progressing favorably, thus making this score less advantageous as a diagnostic tool. In any case, the prognostic validity of our scoring system devised in this preliminary work should be further confirmed in another cohort of IEDCS divers.

The importance of the shortest delay in recompression to limit the risk of sequelae after a neurological DCS is a concept that is commonly accepted in the literature, but the maximum period of time beyond which the likelihood of recovery is

reduced has not been clearly established.¹ In the specific case of IEDCS, there are very few studies that have assessed the influence of the delay in receiving hyperbaric treatment on the prognosis for these injuries. In a series of 23 injured divers (mostly military), half of whom had used a helium-oxygen breathing mixture during the dive with a change of gas (air) at the decompression stops, Farmer *et al.*⁶ observed that among those who were recompressed in 68 min (14 cases) only 2 cases retained a residual deficit, whereas none of the 9 divers who had later hyperbaric treatment (or even no treatment) were cured. In another study concerning 26 recreational divers who were victims of IEDCS and were treated with a U.S. Navy table 6 (oxygen at 2.8 ATA for 5 h), it was shown that a time to recompression <6 h significantly reduced the risk of audio-vestibular sequelae in the medium and long term.¹² More recently, in a cohort of 50 divers treated homogeneously and followed up for 3 mo after an IEDCS, Gempp and Louge⁷ did not observe any difference in the treatment delay between the group which progressed favorably and the group which still presented clinical disorders (150 min versus 180 min, respectively). In this study, we have shown that a delay in hyperbaric treatment with a threshold value close to 6 h was an independent predictive factor for functional disorders at 3 mo. Nevertheless, although this delay seems statistically significant, its influence in clinical practice seems limited in view of the relative risk (estimated by the AOR) which is close to 1. Other work is necessary to determine more accurately the optimum delay to be observed in order to significantly reduce the risk of incomplete recovery.

Our study had a certain number of limitations. A significant proportion of divers, notably those who had good functional recovery, did not have secondary ENT examinations to look for possible subclinical abnormalities, thus reducing the identification of predictive factors for sequelae solely on subjective data in our analysis. However, it is well established that the perception of vertigo and dizziness are poorly correlated with objective and quantifiable vestibular testing (caloric test, posturography) mainly due to central compensation.^{4,11} In this context, patient assessment of symptoms appears to have a greater value than objective measurements in terms of repercussions on daily life. In the present work, we are aware that the influence of emotional status on the extent of residual symptoms was not quantified, potentially decreasing the proportion of subjects with incomplete recovery. However, the psychological dimension of dizziness seems to be more prominent in nonorganic and chronic vestibular diseases,³ suggesting that the report of this item would not give additional value to the results. The majority of the injured divers (78%) were treated with a short initial recompression table using U.S. Navy table 5 (oxygen at 2.8 ATA for 2.5 h), which, on the one hand is a procedure that is little used for this indication in other countries, and on the other hand has not made it possible to analyze the influence of the type of regimen on the clinical outcome. However, our results showed that the proportion of patients who presented a residual deficit at the end of the follow-up period was generally lower than those of other series of divers treated with a longer table such as U.S. Navy table 6,^{10,13} thus excluding the possibility

of concluding that there is greater efficacy for this latter recompression procedure.

In conclusion, in one-quarter of cases, divers with IEDCS complain of persistent symptoms at 3 mo despite early HBOT. Nevertheless, the impact of this poor recovery on health-related quality of life remains to be demonstrated with a distinct questionnaire examining the degree of handicap associated with this injury.⁴ The establishment of a clinical severity score on admission appears useful in predicting the risk of functional sequelae. More specifically, a value <10 makes it reasonably possible to exclude an unfavorable outcome. In the future, it will be necessary to validate this score by prospective studies, particularly to assess the efficacy of the various existing therapeutic tables. Hyperbaric treatment after >6 h may also be associated with a poor outcome, but the real influence of time to recompression on recovery beyond this threshold has not been clearly demonstrated clinically in the present study.

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