

Case reports

Inner-ear decompression sickness in nine trimix recreational divers

Silvia Guenzani, Diego Mereu, Mark Messersmith, Diego Olivari, Mario Arena and Andrea Spanò

Abstract

(Guenzani S, Mereu D, Messersmith M, Olivari D, Arena M, Spanò A. Inner-ear decompression sickness in nine trimix recreational divers. *Diving and Hyperbaric Medicine*. 2016 June;46(2):111-116.)

Introduction: Recreational technical diving, including the use of helium-based mixes (trimix) and the experimentation of new decompression algorithms, has become increasingly popular. Inner-ear decompression sickness (DCS) can occur as an isolated clinical entity or as part of a multi-organ presentation in this population. Physiological characteristics of the inner ear make it selectively vulnerable to DCS. The inner ear has a slower gas washout than the brain thus potentially making it more vulnerable to deleterious effects of any bubbles that cross a persistent foramen ovale (PFO) and enter the basilar artery, whilst the inner ear remains supersaturated but the brain does not.

Methods: A questionnaire was made widely available to divers to analyse the incidence of inner-ear DCS after technical dives. One-hundred-and-twenty-six divers submitted completed questionnaires, and we studied each incident in detail.

Results: Nine (7.1%) of the 126 responders reported to have had at least one episode of inner-ear DCS, of which seven occurred without having omitted planned decompression stops. Of these seven, four suffered from DCS affecting just the inner ear, while three also had skin, joint and bladder involvement. Five of the nine divers affected were found to have a PFO. All affected divers suffered from vestibular symptoms, while two also reported cochlear symptoms. Three divers reported to have balance problems long after the accident.

Conclusions: This small study is consistent with a high prevalence of PFO among divers suffering inner-ear DCS after trimix dives, and the pathophysiological characteristics of the inner ear could contribute to this pathology, as described previously. After an episode of DCS, vestibular and cochlear injury should always be examined for.

Key words

Decompression illness; helium; scuba/open circuit; rebreathers/semi-closed circuit; rebreathers/closed circuit; persistent (patent) foramen ovale (PFO); case reports

Introduction

During the last 20 years, recreational divers interested in exploring deep wrecks, caves and reefs have understood the importance of improving their diving techniques to avoid injury and to increase their enjoyment during these deeper dives. These objectives have been achieved using enhanced diving equipment and techniques and breathing gases different from air. These so-called recreational technical dives utilizing helium-based mixed gases (trimix) have become increasingly popular.¹ Compared to nitrogen, helium is non-narcotic and its lower density makes it easier to breath at high pressures. Gas-switches to hyperoxic nitrox/trimix mixes are often used during ascent to make decompression more efficient.^{2,3}

Technical diving carries a greater risk of decompression sickness (DCS), mainly related to the greater depths and prolonged bottom times associated with this type of diving. The vestibulo-cochlear end organ of the inner ear can be damaged by inert gas bubble formation in DCS.² For a useful summary of the potential mechanisms of inner ear DCS, readers are referred to a recent review published in this

journal,⁴ based on previous theoretical and epidemiological studies.⁵⁻⁸

Reports describing inner-ear DCS in technical trimix divers are limited, so in this paper we present the case histories of nine divers who responded to a widely distributed, on-line questionnaire sent to technical divers in Europe to assess the occurrence of inner-ear DCS in this form of recreational diving.

Methods

The study was conducted between September 2014 and February 2015. An on-line questionnaire* in English and Italian was distributed to recreational trimix divers using mailing lists and social networks (GUE-mailing list, DIR-Germany mailing list, DIR-Italy mailing list, Facebook). Eligibility criteria for respondents were recreational technical divers having a trimix diving certification and at least five dives after their training course. Divers were asked to provide anthropometric data, number of dives in their career and any occurrence of inner-ear DCS after trimix dives. Divers who had suffered from inner-ear DCS were

* **Footnote:** Questionnaire available on request to the author <silvia.guenzani@gmail.com>.

Table 1

Summary of nine divers with inner-ear DCS injury and their symptoms; BMI – body mass index; CCR – closed-circuit rebreather; DCS – decompression sickness; OC – open-circuit scuba; PFO – persistent foramen ovale; SCR – semi-closed circuit rebreather;

Case	Age (y)	BMI (kg·m ⁻²)	Total dives	Trimix dives	Equipment	Decompression model Gradient factors	Omitted stops	Isolated inner-ear DCS	Side	Vestibular symptoms	Cochlear symptoms	Long-term symptoms	PFO	Restart diving
1	41	23.3	2,500	350	OC	Buhlmann; 20/80	No	Yes	?	Yes	No	No	Yes	Yes
2	50	26.1	500	60	SCR	Buhlmann; 20/80	Yes	Yes	?	Yes	No	Imbalance	Yes	No
3	33	29.3	900	150	SCR	Buhlmann; 20/80	No	No	?	Yes	No	No	?	Yes
4	46	27.4	1,200	250	OC	Ratio method	No	No	?	Yes	No	No	Yes	Yes
5	55	31.5	405	68	OC	Buhlmann; 10/90	No	Yes	?	Yes	Yes	Imbalance	Yes	Yes
6	61	23.7	1,500	400	OC	Buhlmann; 20/80	No	Yes	Right	Yes	No	No	Yes	Yes
7	46	24.4	5,000	2,000	OC	Ratio method	No	Yes	Right	Yes	Yes	Imbalance	No	Yes
8	57	28.1	1,500	1,000	CCR	VPM 2	No	No	Right	Yes	No	No	No	Yes
9	37	21.8	2,000	350	SCR	VPM 2	Yes	No	Left	Yes	No	No	No	Yes

subsequently contacted directly by the authors to gather further information about their accidents. We analysed every case report and considered as inner-ear DCS only those episodes that were diagnosed and treated by hyperbaric physicians. Written permission was obtained from all the divers with diagnosed inner-ear DCS to publish their data and details of each case report.

Data analysis was performed using Prism 6 Graph Pad. Data is presented as mean (range) for continuous variables and as frequency for discrete variables.

Results

One-hundred-and-twenty-six divers completed the questionnaires (89.6% male, 10.4% female). Mean age was 44 (range 25–69) years and mean body mass index 24.7 (range 17.6–41) kg·m⁻². The median total number of dives in their career was 1,000 (range 153–6,500), and the median number of trimix dives 200 (range 5–4,000). Nine divers (7.1%), all male, reported having suffered from inner-ear DCS after a trimix dive (diagnosis confirmed by a hyperbaric physician). A summary of the cases appears in Table 1.

Seven divers suffered from inner-ear DCS without having omitted planned decompression stops. Of these seven subjects, four suffered from isolated inner-ear DCS, whilst three also had skin, joint and bladder involvement. Of the four divers with isolated inner-ear DCS, three were discovered to have a PFO after the accident, whilst the fourth diver's test was reported as negative but was performed without bubble contrast.

All nine divers suffered from vestibular symptoms, but only two reported tinnitus or hearing loss. The right ear was affected in three cases, the left in one whilst the other subjects' diagnosis did not specify the side of the injury. Considering long-term follow up, no subjects were examined subsequently for vestibular function,⁸ despite three divers complaining of on-going balance problems. Hearing damage was not reported in the four divers who underwent a hearing test, this was normal post treatment. All but one subject has started diving again; the ninth diver is awaiting closure of his PFO.

CASE 1

This 41-year-old diver who had logged 350 mixed-gas dives suffered inner-ear DCS on two occasions using open-circuit (OC) trimix. The first dive was for a bottom time (BT) of 45 minutes (min) to a maximum depth of 84 metres of water (mw) using a bottom mix of 13% oxygen and 67% helium (13/67, residuum nitrogen) and, on ascent, nitrox 50 % (nitrox50) from 21 mw and oxygen (O₂) from 6 mw for decompression. The diver followed a Bühlmann algorithm with gradient factors of 20/80. The second dive, some months later, was for a 50 min BT to a maximum depth of

45 mw, using a bottom mix of 21/35 and nitrox50 from 21 mw on the ascent using the 'ratio deco' method. In neither dive were decompression obligations missed. No problems with equalisation occurred during either descent.

Symptoms included severe nausea and vomiting, vertigo, balance problems and nystagmus appearing within 10 min of the dive in the first incident and 90 min in the second. The victim was given high concentration O₂ and transferred to the nearest recompression chamber. On both occasions, he was recompressed on a US Navy Treatment Table 6 (USN T6) with a good response. After the second incident, he tested positive for a persistent foramen ovale (PFO) and decided to undergo surgery, following which he resumed diving. There does not appear to be any residual damage to cochlear or vestibular function, but he has not undergone any specific testing.

CASE 2

This 50-year-old diver, with 60 trimix dives, dived for a BT of 30 min to a max. depth of 60 mw, using a semi-closed rebreather (SCR); bottom mix was 21/35, 50/25 from 21 mw and O₂ from 6 mw. Decompression was planned according to a Bühlmann algorithm using gradient factors 20/80. The subject noticed something wrong during his gas switch at 21 mw, as indicated by his oxygen partial pressure (PO₂) monitor, which showed a PO₂ lower than expected, but he assumed the sensor was providing an erroneous output. At 15 mw, he switched momentarily to pure oxygen to test the sensors, noting a PO₂ of 2.1 bar (213 kPa) and concluding the sensor was reading correctly. The decompression stops were carried out as planned. On reaching the surface, he experienced vomiting and vertigo and was taken to the nearest recompression chamber where he underwent a USN T6 with a good response. Subsequent to the incident, the subject had an echocardiogram which showed a PFO and he is awaiting surgery. He reports on-going imbalance issues at times but has never undergone a specific medical exam to test vestibular function.

Comment: Following the incident, gas analysis showed that the 21-m cylinder had a 33/45 mix not nitrox50, so less O₂ than planned. The diver had not analysed the cylinder himself before the dive. Using Decompression Planner software and a Bühlmann algorithm with gradient factors 20/80, the same as used by the diver for his profile but with the actual gases used, 20 min of decompression had been omitted in the 21–9 mw range.

CASE 3

This 33-year-old diver, with 150 mixed gas dives was diving in a lake using a SCR. The diving profile was a V-shaped bounce dive with a BT of approximately 2 min to a max. depth of 140 mw using a bottom mix 12/60 and decompression mixes of 20/50 from 75 mw, 38/30 from 39

mw, 55/15 from 24 mw and oxygen from 6 m depth. At a depth of 30 mw, he had a strange feeling in the elbow which persisted during the ascent. At 9 mw, he started feeling nauseated but this went away after a short time on oxygen at the 6 mw stop. The subject followed the algorithm suggested by his dive computer (Bühlmann gradient factors 20/80) with an extension of the decompression time because of the pain he experienced at depth.

Approximately 10 min post dive, he developed vertigo and was unable to walk without holding on to fixed objects for stabilization. He was rapidly transferred to a hyperbaric chamber and recompressed following a COMEX 30 table. The following day, he underwent a second hyperbaric treatment at 253 kPa for a total treatment time of 150 min. At the end of this treatment he was asymptomatic. Some months later, he started to dive again. He claims to be asymptomatic concerning both hearing and balance but has not undergone any specific medical examination. Neither was echocardiography performed to examine for the presence of a PFO.

CASE 4

A 46-year-old diver, with 250 trimix dives, undertook a wreck dive using OC-scuba for a BT of 45 min to a max. depth of 57 mw. The bottom mix was 18/45 and decompression gases nitrox50 from 21 mw and O₂ from 6 mw. Following the 'ratio deco' method, no decompression stops were omitted and the other team members did not suffer any symptoms post dive. After 20 minutes on the surface the subject experienced vertigo, difficulty with balance, a skin rash and hypersensitivity to sunlight. He was treated with surface O₂ and subsequently taken to the nearest hyperbaric chamber. In the hospital, the diver had a chest X-ray, which was normal, and subsequently underwent a USN T6, resulting in complete resolution of his symptoms.

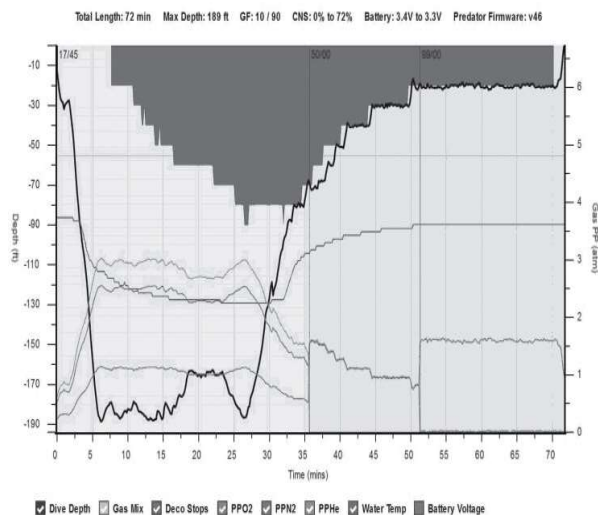
On two separate occasions after this incident, he reported DCI symptoms (skin rash) after trimix dives, so he decided to undergo an echocardiography which showed a PFO. This was repaired and he subsequently resumed diving. He also underwent a hearing test, which was normal, and has no balance problems.

CASE 5

This 55-year-old diver, with 68 trimix dives, undertook a wreck dive in a lake using OC-scuba. The dive profile was a BT of 25 min to a max. depth of 63 mw, with a total run time of 75 minutes. Bottom mix was 17/45, then nitrox50 from 21 mw and O₂ from 6 mw for decompression. No planned decompression was omitted according to the Bühlmann algorithm with gradient factors 10/90 (Figure 1). The subject claims to have started feeling sinus pressure during the 6 mw stop on O₂. Then 15 minutes after surfacing, severe vertigo, tinnitus and hypersensitivity to light and sound

Figure 1

Case 5 dive profile (downloaded from the diver's Shearwater™ computer)



developed. The Coast Guard happened to be on board for a routine inspection, so the diver was taken by their vessel to the nearest harbour where an ambulance was waiting to take him to the nearest hyperbaric chamber. He was recompressed with a USN T6. He received six more daily 2-h hyperbaric oxygen treatments, by which time he had improved considerably.

He resumed recreational diving, but decided to stop trimix technical diving activity. He also underwent echocardiography which showed the presence of a PFO. His vestibular or cochlear function were not tested, but he reports that, under certain circumstances, he still gets occasional episodes of imbalance, like riding a bike across a narrow bridge or going through a tunnel.

CASE 6

This 61-year-old diver, with 405 trimix dives, was on a wreck dive using OC-scuba with a BT of 20 min to a max. depth of 95 mw. Bottom mix was 15/65 and decompression gases 35/25 from 36 mw, nitrox50 from 21 mw and O₂ from 6 mw. During ascent, no decompression stops were omitted according to a Bühlmann algorithm with gradient factors 20/80. He experienced sudden onset of vertigo post dive while hauling up the boat's anchor. He was immediately administered surface O₂ until eventually arriving at the recompression chamber. He remained at the first hospital, which did not have an operational chamber, for about four hours whilst undergoing various tests including a brain CT scan before being transferred to a hyperbaric chamber. He was recompressed on a USN T6 with a two-hour extension, at the end of which he was asymptomatic. The diagnosis was right-sided inner-ear DCS.

Subsequent transthoracic echocardiography revealed the presence of a PFO. He chose not to have this closed and resumed diving as he was asymptomatic and a hearing test was normal. About one year later, he experienced sudden hearing loss in the right ear, which resolved spontaneously. Both a hyperbaric medicine physician and otolaryngologist excluded the possibility that this could have been related to the DCS incident.

Comment: This dive had the same decompression profile as another one a year before on the same wreck. The differences were that he had to haul up the anchor post dive on the incident dive and that he was colder because some water had entered his drysuit. It is not uncommon to see the onset of DCI symptoms at the time of or shortly after post-dive exertion.

CASE 7

This 46-year-old diver, with over 2,000 trimix dives using OC-scuba, dived to a max. depth of 45 mw for a 20 min BT using 21/35 as bottom mix and nitrox50 for decompression from 21 mw. No decompression stops had been omitted following the 'ratio deco' model. He did not "feel right" just after surfacing and developed tinnitus and heard crackling in the ear(s). After using a nasal spray to try to clear the ear, symptoms quickly deteriorated and as he was walking to obtain O₂ he collapsed from vertigo.

He was taken to a hospital and upon arriving was diagnosed with complete loss of hearing in the right ear. Despite this, he was sent home even though he had difficulty walking and maintaining a straight line without vomiting. The following day, he visited an ear specialist who confirmed there was no ear barotrauma. The diver then insisted on being referred to a hyperbaric chamber for treatment. A diagnosis of right-sided inner-ear DCS was made and, after receiving a USN T6, he felt much better. His tinnitus subsided completely over two weeks and a hearing test was normal. A transthoracic echocardiography, but without bubble contrast, did not demonstrate a PFO. He still experiences intermittent episodes of imbalance.

Comment: A past history that might be relevant included a bicycle crash a few months earlier, in which he fractured his right cheekbone. He also reported that he had slipped on a wet floor a few days before this dive, again striking the right side of his head.

CASE 8

This 57-year-old diver, with 1,000 trimix dives, undertook a wreck dive using a closed-circuit rebreather (CCR) with a BT of 30 min to a max. depth of 90 mw. Bottom mix was 8/60, with the PO₂ held at 1.2 bar (121 kPa) during the BT and 1.4 bar (143 kPa) during the ascent. Decompression stops were calculated using the VPM 2 algorithm, and no

planned decompression was omitted. During the descent the diver did not have any trouble equalizing his ears. Soon after surfacing, he experienced vertigo, nausea and was unable to micturate. The diver was diagnosed with right-sided inner-ear DCS and neurological involvement of the bladder. He was promptly treated with a USN T6 in the nearest recompression chamber. He improved but at the end of the treatment balance problems remained. The day after he did a second treatment according to a US Navy Treatment Table 5 (USN T5) and was discharged home. He continued to have some balance problems for three days but is now asymptomatic, and audiometry is normal. Transthoracic echocardiography with bubble contrast did not demonstrate a PFO.

CASE 9

This 37-year-old diver, with 350 trimix dives, commented that prior to the day of the incident he was very tired following two weeks of hard work. He and his dive buddies were all diving with SCRs during the incident dive. The profile was a BT of 40 min to a max. depth of 88 mw, with a bottom mix of 10/70. At 43 min of run time, the subject and his team reached 57 mw and switched to a 21/35 mix. Some delay occurred during this gas switch and at 50 min run time they recommenced their ascent at a rate of 3 m·min⁻¹ until reaching 42 mw. The subsequent deco stops were prolonged by 2 min every 3 mw until 27 mw, reaching 24 mw at 67 min run time. At 24 mw, the team did a 3-min gas break breathing the bottom mix, and then ascended to 21 mw, where the entire team switched to trimix 50/20. A few minutes after this gas switch, the diver experienced severe vertigo and loss of orientation. He decided to immediately switch to open circuit and signalled the team members of his situation. From 21 mw to the surface, no deco stops were omitted according to algorithm VPM 2. For the remaining two hours of the decompression, the subject needed constant assistance by his team in gas switches and position reference. Due to frequent vomiting the subject became dehydrated during the dive. Upon exiting the water, the subject reported pain in both knees and that he was unable to micturate. He was rapidly transferred to the nearest hyperbaric chamber and recompressed following a USN T6. During the next few days he was recompressed three times using a USN T5. The diagnosis was musculo-skeletal and inner-ear DCS, probably affecting the left ear. The difficulty with micturition was ascribed to severe dehydration. One month after the incident he had completely recovered and started diving again. Transoesophageal echocardiography with bubble contrast excluded the presence of a PFO. He declined vestibular or audiometric testing.

Comment: Some days after the incident, he and his dive buddies analysed the decompression profile and realised they had omitted about 20 minutes of deep deco stops according to the VPM-2 algorithm by not taking into account as bottom time the extra period spent at 57 mw to complete the gas switch.

Discussion

Among serious cases of DCI in recreational divers, the incidence of inner-ear symptoms is reported to be around 24 to 34%,^{7,9,10} whilst in a reported case series from a single recompression centre, inner-ear DCS represented 11.7% of all DCI occurring after trimix dives.¹¹ However, these data cannot be considered to reliably describe a true incidence, because the number of analysed cases was quite small. In our case series, about 7% of technical recreational divers completing the questionnaire had suffered from inner-ear DCS after a trimix dive during their career. It is likely the 'true' incidence would be lower because of a reporting bias to our questionnaire. Also, as it was specific for the incidence of inner-ear DCS, we cannot assign an incidence of inner-ear DCS among all DCI in trimix divers. In recreational diving, we can usually determine if a diver exceeds decompression limits. The evaluation of decompression exposure is more challenging with technical diving because of the array of algorithms and implementations that are used. None of the decompression techniques documented in these nine cases could be regarded as 'conservative' from a DCS-risk perspective.

Our data are in keeping with the reported association of inner-ear DCS with the presence of a PFO.^{6,7,12} Venous gas emboli (VGE) are commonly detected after diving, often as 'silent' bubbles in asymptomatic divers. The presence of a PFO allows arterialization of venous bubbles, especially during particular manoeuvres such as a Valsalva or coughing. A high prevalence of arterial gas emboli has been reported after trimix diving, even without omission of decompression stops.¹³ In that study, arterialisation of VGE was detected in five of seven divers who completed 21 trimix dives without protocol violations, although a PFO was detected in only one diver. The authors ascribed this to possible intrapulmonary shunts when a high venous bubble grade was detected in the right heart.

Another possible mechanism described for inner-ear DCS is the phenomenon of isobaric counter-diffusion.¹⁴ This refers to the isobaric formation of bubbles as a result of supersaturation induced by the steady-state counter-diffusion of two gases with different solubilities and rates of diffusion. After a gas switch from a helium-rich mix to a poorer one, the vascular compartment of the inner-ear could be supersaturated because of the rapid transfer of helium from the perilymph into blood and the slower transfer of nitrogen into the perilymph from the vascular compartment. This mechanism could explain isolated inner-ear DCS occurring at depth.⁴ Of our cases, only one diver suffering vertigo and nausea underwater and, in this case, this was more likely because of missed decompression earlier in the ascent, rather than to the gas switch per se.

In our series, all divers had vestibular symptoms, whereas only three suffered from cochlear symptoms. A greater

prevalence of vestibular symptoms in inner-ear DCS has been reported in other studies.^{11,12} This could be explained by the fact that cochlear blood flow is three times greater than vestibular flow,¹⁵ so inert gas 'wash-out' is faster in the cochlea than in the vestibular organ.

Two studies have reported preponderance of inner-ear DCS to the right side.^{9,10} We observed that of the four divers who were diagnosed with unilateral inner-ear DCS, three were on the right side. This has been ascribed to a narrower diameter of the right vertebral artery compared to the left.¹⁰ However, bubbles are reported to reach the left ear 0.3 sec before the right, so time for their dissolution should be shorter and the left ear should be more susceptible than the right.¹⁴ We believe this topic is worthy of more detailed analysis.

Three of the nine divers reported on-going balance problems. Unfortunately none of the divers underwent specific vestibular or audiometric testing. Residual vestibular damage after DCI has been reported to affect 62 to 78% of divers, even if less than 20% were symptomatic.^{10,16,17} This emphasizes the need for thorough vestibular function evaluation after inner-ear DCS and before returning to diving activities. About 40% of divers have also been reported to have audiometric sequelae after inner-ear DCS.^{10,16}

Conclusion

Nine of 126 (7.1%) trimix divers responding to a questionnaire reported suffering inner-ear DCS. The presence of a right-to-left shunt and poorly validated decompression profiles are important risk factors for inner-ear DCS, as are anatomical and physiological characteristics of the inner-ear. Inner-ear DCS may occur in isolation or be associated with other symptoms of DCS. Prompt recompression treatment should resolve symptoms. Subsequent to inner-ear DCS, residual vestibular and cochlear damage should be assessed even if the diver appears to be asymptomatic.

References

- Mitchell SJ, Doolette DJ. Recreational technical diving part 1: an introduction to technical diving methods and activities. *Diving Hyperb Med.* 2013;43:86-93.
- Hamilton B. Mixed-gas diving. In: *Bove and Davis' diving medicine*, 4th ed. Philadelphia: Elsevier Inc; 2004.
- Jablonski J. *Getting clear on the basics: the fundamentals of technical diving*. High Springs FA: Global Underwater Explorers; 2001.
- Doolette DJ, Mitchell SJ. Biophysical basis for inner-ear decompression sickness. *J Appl Physiol.* 2003;94:2145-50.
- Mitchell SJ, Doolette DJ. Selective vulnerability of the inner-ear to decompression sickness in divers with right-to-left shunt: the role of tissue gas supersaturation. *J Appl Physiol.* 2009;106:298-301.
- Germonpré P, Dendale P, Unger P, Balestra C. Patent foramen ovale and decompression sickness in sports divers. *J Appl Physiol.* 1998;84:1622-6.
- Cantais E, Louge P, Suppini A, Foster P, Palmier B. Right-to-left shunt and risk of decompression illness with cochleovestibular and cerebral symptoms in divers: Case control study in 101 consecutive dive accidents. *Crit Care Med.* 2003;31:84-8.
- Wuytsa F, Furmanb J, Vanspauwena R, Van de Heyning P. Vestibular function testing. *Curr Opin Neurol.* 2007;20:19-24.
- Nachum Z, Shupak A, Spitzer O, Sharoni Z, Ramon Y, Abramovich A, et al. Inner-ear decompression sickness in sport compressed-air diving. *Laryngoscope.* 2001;111:851-6.
- Gemmp E, Louge P. Inner-ear decompression sickness in scuba divers: a review of 115 cases. *Eur Arch Otorhinolaryngol.* 2013;270:1831-7.
- Ignatescu M, Bryson P, Klingmann C. Susceptibility of the inner-ear structure to shunt-related decompression sickness. *Aviat Space Environ Med.* 2012;83:1145-51.
- Klingmann C, Benton PJ, Ringleb, PA, Knauth, M. Embolic inner-ear decompression illness: correlation with a right-to-left shunt. *Laryngoscope.* 2003;113:1356-61.
- Ljubkovic M, Marinovic J, Obad A, Breskovic T, Gaustad S, Dujic Z. High incidence of venous and arterial gas emboli at rest after trimix diving without protocol violations. *J Appl Physiol.* 2010;109:1670-4.
- Solano-Altamirano J, Goldman S. The lifetimes of small arterial gas emboli, and their possible connection to inner-ear decompression sickness. *Math Biosci.* 2014;252:27-34.
- Angelborg C, Larsen HC. Blood flow in the peripheral vestibular system. *J Otolaryngol.* 1985;14:41-3.
- Shupak A, Gil A, Nachum Z, Miller S, Gordon C, Tal D. Inner-ear decompression sickness and inner-ear barotrauma in recreational divers: A long-term follow-up. *Laryngoscope.* 2003;113:2141-7.
- Klingmann C, Praetorius M, Baumann I, Plinkert PK. Barotrauma and decompression illness of the inner-ear: 46 cases during treatment and follow-up. *Otol Neurotol.* 2007;28:447-54.

Conflicts of interest: nil

Acknowledgements

We are grateful to Fred Devos of Global Underwater Explorers and all 126 divers who completed our questionnaire.

Submitted: 22 April 2015; revised 11 May 2015, 03 January 2016 and 11 April 2016

Accepted: 16 April 2016

Silvia Guenzani¹, Diego Mereu², Mark Messersmith², Diego Olivari³, Mario Arena², Andrea Spanò²

¹ Hyperbaric Center, Niguarda Hospital, Milano, Italy

² GUE, Global Underwater Explorers, High Springs, Florida, USA

³ Nad Al Sheba Sports Complex Deep Dive, Dubai

Address for correspondence:

Silvia Guenzani
c/- Reparto di anestesia e rianimazione Bozza
Blocco DEA primo piano
Niguarda Hospital
Piazza dell'ospedale maggiore 3
Milano, Italy
Phone: +39-(0)34-9868-3498
E-mail: <silvia.guenzani@gmail.com>