Freediving Medical and Scientific Commission

Medical Risk Awareness in Freediving: Safety Recommendations - DCS/DCI

2026/01

1. PURPOSE AND SCOPE

Freediving has grown rapidly as a competitive and recreational activity worldwide, and breath-hold decompression illness (DCI) is increasingly recognized as a genuine, if still under-reported, hazard of the sport rather than a historical curiosity confined to traditional pearl and shellfish divers (Blogg, Tillmans, & Lindholm, 2023). Neurological forms of DCI, including presentations that closely resemble stroke and the condition historically known as Taravana syndrome, are of particular concern because they can affect young, otherwise healthy athletes and may leave lasting damage if not recognized quickly.

This document brings together current peer-reviewed evidence together with existing guidance from the Divers Alert Network (DAN) and the Undersea and Hyperbaric Medical Society (UHMS) on patent foramen ovale (PFO) and fitness to dive (Divers Alert Network & Undersea and Hyperbaric Medical Society, 2015), in order to alert freedivers to the range of factors that can influence their individual risk of neurological decompression illness.

Scope and limitations. This recommendation describes risk factors and warning signs only. It deliberately does not address treatment, hyperbaric protocols, or in-water management of suspected decompression illness, as these are matters for qualified medical professionals to determine on a case-by-case basis. The document concludes by directing athletes toward the specific types of medical consultation appropriate to their individual circumstances.

2. BACKGROUND: HOW NEUROLOGICAL INJURY CAN OCCUR IN FREEDIVING

2.1 From "negligible risk" to a recognized hazard

For many years it was assumed that a single breath-hold dive carries essentially no decompression risk, because the time spent with compressed gas at depth is so brief. This assumption was first challenged in the 1960s, when repeated breath-hold dives were shown to be capable of producing decompression-sickness-type symptoms (Paulev, 1965). Subsequent research has confirmed that repetitive deep breath-hold dives performed with short surface intervals can allow nitrogen to accumulate in blood and tissue to a degree sufficient to cause decompression sickness, including neurological forms (Lemaitre, Fahlman, Gardette, & Kohshi, 2009). A 2023 systematic review of the available case literature concluded

that both decompression sickness (DCS) and arterial gas embolism (AGE) are documented, plausible mechanisms of neurological injury in breath-hold divers, and that both should be regarded as genuine risks for this group, just as they are for divers breathing compressed gas (Blogg et al., 2023).

2.2 Taravana syndrome

"Taravana" is the term historically applied to the neurological illness observed in Polynesian breath-hold pearl divers who performed many repetitive deep dives in a single day; it is increasingly understood as a specific, breath-hold-diving presentation of decompression illness (Kohshi, Tamaki, Lemaître, Morimatsu, Denoble, & Ishitake, 2021). Its hallmark is that symptoms are predominantly cerebral and stroke-like — weakness, visual or speech disturbance, vertigo, or confusion — while the spinal cord, which is frequently affected in classical compressed-gas decompression sickness, is typically spared (Kohshi, Denoble, Tamaki, Morimatsu, Ishitake, & Lemaître, 2021). The precise pathophysiology remains debated. The most widely accepted explanation treats Taravana as a specific form of decompression sickness driven by nitrogen micro-bubbles. One hypothesis claims bubbles formation in the venous bloodstream, and from there, through a shunt, migrate to the arterial side, and the complementary hypothesis under active investigation proposes that some cases arise from micro-bubbles forming directly within cerebral vascular structures (Druelle, Castagna, Roffi, Louge, Faivre, & Blatteau, 2024).

2.3 Not the same condition as hypoxic blackout

Freediving carries a separate, better-known neurological risk in the form of hypoxic blackout, caused by a fall in blood oxygen rather than by gas bubble formation. The two conditions can produce overlapping symptoms, such as confusion or loss of consciousness, but they arise through different mechanisms. This distinction matters because physiological resilience to hypoxia does not equate to protection against decompression-related neurological injury. Research on elite breath-hold divers has documented marked cardiac and pulmonary-vascular adaptation to repeated, severe hypoxic stress during maximal apnea (Kjeld et al., 2021; Kjeld et al., 2024); however, gas bubble formation and right-to-left shunting across a PFO (Patent Foramen Ovale) operate independently of an individual's tolerance to low oxygen. In other words, being a strong, well-adapted breath-hold athlete does not, by itself, reduce the decompression-related risks addressed in this document.

3. FACTORS THAT MAY INCREASE THE RISK OF NEUROLOGICAL DECOMPRESSION ILLNESS

Research and accumulated clinical experience point to several largely independent factors that can each raise an individual freediver's risk of neurological decompression illness, and which may compound one another when present together. They are summarized below, each followed by a corresponding caution.

3.1 Dive Profile: Repetition, Depth, Surface Intervals and Ascent Rate

Across the available case literature, the most consistently identified risk factor for breath-hold DCI is the dive profile itself: repetitive deep dives performed with inadequate surface intervals, dives exceeding roughly 40 metres, and rapid ascent rates (Blogg et al., 2023). Field studies

of commercial Ama breath-hold divers in Japan show the same pattern, with neurological, stroke-like events concentrated among divers performing many repetitive dives with short recovery periods (Kohshi, Tamaki, Lemaître, Morimatsu, Denoble, & Ishitake, 2021). Prolonged, multi-day high-load training or competition blocks without adequate recovery extend this same risk over time (Kohshi et al., 2021).

Underwater propulsion devices (diver propulsion vehicles, or "scooters") introduce a related and increasingly recognized concern: by allowing divers to reach greater depths, complete more repetitions, and ascend faster than is achievable by unaided finning, they can intensify exactly the profile-related factors described above. A documented case of severe neurological injury, with features overlapping both Taravana syndrome and posterior reversible encephalopathy syndrome, occurred in a freediver using such a device (Druelle, Castagna, Roffi, Louge, Faivre, & Blatteau, 2024).

┌─ CAUTION
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──────┐ │ Freedivers who perform repetitive deep dives with short surface │ │ intervals, who train or compete across multiple consecutive days without │ │ adequate recovery, or who use underwater propulsion devices to extend │ │ depth and repetition, should recognize that this combination of factors │ │ is specifically and repeatedly associated with neurological decompression │ │ illness, including Taravana-type presentations. │
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3.2 Patent Foramen Ovale and Other Right-to-Left Shunts

A patent foramen ovale (PFO) is a small, naturally occurring opening between the upper chambers of the heart that fails to fully close after birth, present in roughly 27% of people, with larger, higher-grade openings present in around 6% (Divers Alert Network & Undersea and Hyperbaric Medical Society, 2015). When pressure in the right side of the heart exceeds that on the left, a PFO can allow venous gas bubbles to bypass the lungs and reach the arterial circulation, including the brain — a right-to-left shunt. DAN/UHMS guidance summarizes that divers with a PFO carry roughly 2.5 times the overall decompression sickness risk of divers without one, and around four times the risk of neurological decompression sickness, with the largest absolute risk concentrated among the minority who carry large, high-grade defects (Divers Alert Network & Undersea and Hyperbaric Medical Society, 2015). The same guidance notes that PFO shows a particularly strong association with cutaneous and inner-ear forms of decompression sickness, and that detection of a PFO after a decompression event does not, by itself, prove that the PFO caused it.

Several independent cohort and case-control studies in compressed-gas divers reinforce this picture: high-grade PFO is over-represented among divers who experience unprovoked or neurological decompression sickness compared with controls (Germonpré, Balestra, Obeid, & Lafère, 1998; Honěk et al., 2019; Lee, Moon, Park, Kim, & Cho, 2023). Although most of this evidence originates from compressed-gas diving, it is relevant to freediving because repetitive deep breath-hold dives can themselves generate venous gas emboli capable of crossing such a shunt.

PFO is also recognized more broadly, outside diving, as a structural cardiac condition associated with cryptogenic stroke (Kjeld, Jørgensen, Fornitz, Roland, & Arendrup, 2018). Of particular interest for freedivers, that same review notes that cryptogenic stroke onset has been statistically associated with vigorous or strenuous exertion and with abrupt intrathoracic pressure changes such as coughing and sneezing (Kjeld et al., 2018). Forceful equalization techniques and heavy exertion around a dive involve broadly comparable abrupt pressure changes in the chest and right heart. This parallel does not establish that equalization technique causes paradoxical embolism in freedivers, but it is a biologically plausible additional consideration for PFO-positive athletes, alongside the diving-specific decompression mechanism already documented above.

The same review also illustrates that closing a PFO is not a simple or universally proven fix: the efficacy of closure devices has not been conclusively demonstrated when randomized trials are taken individually, residual shunting can persist in a substantial proportion of cases after closure, and the procedure carries its own risk of new-onset arrhythmia and device-related complications (Kjeld et al., 2018). DAN/UHMS guidance correspondingly cautions that closure after a decompression event cannot guarantee that decompression sickness will not recur, and specifies that diving should not resume until closure is confirmed by repeat bubble-contrast echocardiography at least three months after the procedure, with potent antiplatelet medication discontinued (Divers Alert Network & Undersea and Hyperbaric Medical Society, 2015).

┌─ CAUTION
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──────┐ │ Athletes with a known or suspected large or spontaneously-shunting PFO │ │ — particularly those who have already experienced an unprovoked, │ │ neurological, cutaneous, or inner-ear decompression event — carry a │ │ meaningfully elevated risk and should be evaluated by a cardiologist │ │ together with a diving-medicine physician before continuing repetitive or │ │ deep freediving. A negative PFO test does not eliminate future risk, and │ │ identifying a PFO after an event does not prove it was the cause of that │ │ event; both situations call for individualized medical interpretation, │ │ not self-diagnosis or self-management. │
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3.3 Equalization Technique and Lung Overexpansion (Glossopharyngeal Insufflation)

Many competitive freedivers use glossopharyngeal insufflation ("lung packing") to increase lung gas volume before a dive. The technique can raise transpulmonary pressure substantially, creating a documented risk of pulmonary barotrauma (Schiffer & Lindholm, 2013). Experimental work measuring airway pressure during glossopharyngeal insufflation found that some divers reach pressures associated with this risk, and breath-hold divers have reported transient, severe neurological symptoms consistent with arterial gas embolism following the technique (Schiffer & Lindholm, 2013). A separate published case describes a breath-hold diver who developed transient paresthesia and reduced sensation on one side of the neck within a minute of performing glossopharyngeal insufflation, a presentation the authors attributed to arterial gas embolism (Liner & Andersson, 2010).

This is mechanistically distinct from classical decompression sickness, since it does not require a deep or repetitive dive profile: the injury arises from lung overexpansion at or near the surface. The resulting symptoms can nonetheless closely resemble Taravana syndrome or stroke.

┌─ CAUTION ────────────────────────────────────┐ │ Glossopharyngeal insufflation (lung packing) and other techniques that │ │ substantially raise lung volume or intrathoracic pressure before a dive │ │ carry a documented, technique-specific risk of pulmonary barotrauma and │ │ arterial gas embolism, producing neurological symptoms that can closely │ │ resemble Taravana syndrome or stroke even without a classic deep, │ │ repetitive decompression profile. Athletes using these techniques should │ │ be trained and supervised by a suitably qualified freediving instructor │ │ and should discuss the practice with a diving-medicine physician. │ └──┘

3.4 Thermal Stress (Cold Water Exposure)

Cold exposure during and after decompression has been shown, in controlled experimental models, to alter cardiovascular function in ways that are mechanistically consistent with increased decompression-related risk, including changes in cardiac output and stroke volume during cold decompression (Gaustad, Kondratiev, Eftedal, & Tveita, 2021). This is consistent with long-standing diving-medicine observations that intrapulmonary shunts — an additional pathway through which venous bubbles can reach the arterial circulation — tend to open with exercise, hypoxia and adrenergic (stress) stimulation, and to close with breathing supplemental oxygen, making it physiologically plausible that exertion, hypoxia and cold-related adrenergic stimulation around a dive could contribute to decompression sickness that might not otherwise have occurred (Divers Alert Network & Undersea and Hyperbaric Medical Society, 2015).

┌─ CAUTION ────────────────────────────────────┐ │ Extended exposure to cold water, particularly across long training days │ │ or multi-stage competitions, can alter cardiovascular function in ways │ │ consistent with increased decompression stress. Athletes who experience │ │ pronounced cold-induced vasoconstriction, or who undertake long │ │ cold-water sessions, should factor this into how they plan surface │ │ intervals, session length and overall training load. │ └──┘

3.5 Hydration Status

Dehydration is a recognized, modifiable individual risk factor for decompression-related bubble formation. A controlled study in divers found that adequate pre-dive hydration measurably reduced post-dive venous bubble formation compared with a non-hydrated control condition (Gempp, Blatteau, Pontier, Balestra, & Louge, 2009). A large epidemiological analysis of the DAN Europe diving safety database likewise identified individual physiological

factors, alongside dive parameters, as contributing to bubble formation and decompression illness risk (Cialoni, Pieri, Balestra, & Marroni, 2017).

┌─ CAUTION
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──────┐ │ Inadequate fluid intake, heavy sweating, heat exposure, and alcohol │ │ consumption around a freediving session are recognized, modifiable │ │ contributors to decompression stress. Athletes should maintain good │ │ hydration before and after sessions, particularly during multi-day │ │ training camps or competitions. │
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3.6 Physical Exertion and Overall Condition

Heavy exertion, straining or lifting in the period immediately after surfacing is specifically flagged in DAN/UHMS guidance as a factor that can promote right-to-left shunting of venous bubbles in PFO carriers (Divers Alert Network & Undersea and Hyperbaric Medical Society, 2015). More broadly across diving disciplines, individual physical condition — including age, body composition and fitness — has been linked to the degree of venous bubble formation observed after diving (Carturan, Boussuges, Burnet, Fondarai, Vanuxem, & Gardette, 1999; Cialoni et al., 2017). The strength of these associations varies between studies and exposure types, but the underlying physiological mechanisms (slower nitrogen elimination from poorly perfused tissue, reduced cardiovascular reserve) are consistent across diving disciplines, including freediving.

┌─ CAUTION
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──────┐ │ Heavy exertion, straining, or lifting in the hours immediately following │ │ a freediving session, and poor physical conditioning more generally, are │ │ associated with increased decompression-related risk. Avoiding strenuous │ │ activity for a period after diving, and maintaining good overall fitness, │ │ are reasonable precautions for any freediver. │
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3.7 Individual Vascular and Hematological Predisposition

Case reports describe Taravana syndrome occurring in breath-hold divers with hyperhomocysteinemia, an underlying thrombophilic (clot-promoting) condition that the authors propose may have increased individual susceptibility (Cortegiani, Russotto, Montalto, Raineri, & Giarratano, 2013). Detailed neuroimaging work on breath-hold-diving-related decompression sickness with brain involvement similarly underscores that the vascular substrate of the individual diver, and not only the dive profile, contributes to how and where neurological injury manifests (Sánchez-Villalobos et al., 2022). This illustrates that an underlying clotting or vascular abnormality, even one previously unknown to the athlete, may compound neurological risk during freediving independently of, or in addition to, a PFO.

┌─ CAUTION
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───────┐ │ A personal or family history of unexplained clotting disorders, early or │ │ unexplained stroke, or recurrent unprovoked vascular events should be │ │ disclosed to a diving-medicine physician before undertaking deep or │ │ repetitive freediving, since such conditions may add to neurological risk │ │ through mechanisms separate from, or in combination with, a PFO. │
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3.8 Prior Neurological or Cutaneous Decompression Events

DAN/UHMS guidance specifically identifies a history of more than one episode of decompression sickness with cerebral, spinal, vestibulocochlear (inner-ear) or cutaneous manifestations as an indication for formal PFO testing; isolated headache after diving, or other "mild", non-specific symptoms, are explicitly not considered sufficient grounds on their own for PFO investigation (Divers Alert Network & Undersea and Hyperbaric Medical Society, 2015). Inner-ear symptoms deserve particular attention: in the studies reviewed by DAN/UHMS, a large majority of divers presenting with isolated inner-ear decompression symptoms were found to have a large, spontaneously shunting PFO, making this presentation a strong practical warning sign.

┌─ CAUTION
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───────┐ │ Any previous neurological, inner-ear, or unusual cutaneous symptom │ │ following a freediving session — even if it resolved completely, and even │ │ after what felt like a routine dive — should be reported in full to a │ │ diving-medicine physician before further deep or repetitive diving. │ │ Repeated episodes are a specific, recognized indication for formal PFO │ │ evaluation. │
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4. WARNING SIGNS: WHEN TO STOP AND SEEK HELP

Neurological decompression illness in freediving, including Taravana syndrome, can present suddenly and with symptoms that are easy to dismiss as fatigue, cold, or nerves. Athletes, buddies, safety divers and coaches should be alert to:

• weakness, heaviness, or numbness in a limb or on one side of the body; • sudden visual disturbance, double vision, or difficulty speaking; • severe or unusual dizziness, vertigo, loss of balance, or confusion; • hearing loss, ringing in the ears, or other inner-ear disturbance; • any other unexplained or unusual neurological or sensory symptom occurring during or after a freediving session.

Any of these symptoms, however mild or brief they may seem, should be treated as a possible medical emergency. The diver should stop diving immediately, not re-enter the water, and seek urgent medical evaluation without delay.

5. CONCLUSION AND RECOMMENDATION

Neurological decompression illness in freediving — including presentations resembling stroke and the historically described Taravana syndrome — remains rare in absolute terms, but its consequences can be severe, and it can affect young, fit, experienced athletes. As set out above, the evidence points not to a single cause but to a set of largely independent factors — dive profile, patent foramen ovale or other right-to-left shunting, equalization technique, thermal stress, hydration status, exertion and physical condition, individual vascular or hematological predisposition, and any prior neurological or cutaneous decompression event — each of which can raise an individual's risk, and several of which may compound one another when present together.

THIS DOCUMENT CANNOT DETERMINE WHETHER ANY INDIVIDUAL ATHLETE IS AT ELEVATED RISK, AND IT IS NOT A DIAGNOSTIC OR TREATMENT TOOL. Its role is limited to raising awareness of recognized risk factors and warning signs. Decisions about testing, training load, participation, and any medical or device-based intervention belong entirely to the athlete and the relevant treating physicians, on an individualized basis.

Accordingly, CMAS recommends that freedivers — particularly those engaged in deep, repetitive, or competitive freediving, or who recognize themselves in any of the factors described above — seek thorough, individualized medical consultation, including as relevant:

• a physician experienced in diving and hyperbaric medicine, for an overall fitness-to-dive assessment and an individualized risk-mitigation strategy;

• a cardiologist, for assessment of a known or suspected PFO or other right-to-left shunt, particularly following any unprovoked or neurological decompression-type event;

• a neurologist, following any neurological symptom occurring during or after diving;

• other specialists — for example in hematology — where personal or family history (unexplained clotting disorders, migraine with aura, unexplained vascular events) warrants further evaluation.

Responsible freediving begins with knowing one's own individual risk profile — something that can only be established through proper medical consultation, never through self-assessment.

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