Research Article

The Gradient Perfusion Model Part 2: Substantiation of the GPM with clinical cases

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ABSTRACT

Introduction: In Part 1 of this three-part series, we provided an explanation as to why and at what sites decompression sickness (DCS) occurs, using the Gradient-Perfusion Model (GPM). In this part, we provide information to substantiate the concept and present clinical cases that were initially labeled as "unexplained DCS," but later disordering events were identified to explain the clinical presentations.

Materials and Methods: Among 500 cases of DCS we have managed for over 50 years, a cohort of these patients was initially diagnosed as unexplained DCS. However, some have shown that disordering events are the likely cause of their DCS.

Results: By pairing the tissue involved with the patient's dive history, a gradient-perfusion imbalance connection was identified. In all serious (Type 2) presentations of DCS, alterations in perfusion of the fast tissues were able to account for the clinical findings. The consequences demonstrated that the gradients overwhelmed the ability of altered perfusion to offgas/offload the inert gas. Pain-only and peripheral neuropathy presentations involved both intermediate and slowly perfused tissues. Rather than perfusion, gradient limitations were the reasons for the clinical presentations of these patients.

Conclusions: The GPM accounts for signs and symptom presentations in DCS. This provides the basis for appropriate treatments and logical recommendations for return to diving. We recommend that the label "unexplained DCS" be discontinued and that the GPM be used to determine the cause. Once the cause is established, "DCS due to disordered decompression" becomes the appropriate term.

KEYWORDS: Gradient Perfusion Model; perfusion; DCS; disordered decompression

INTRODUCTION

In the majority of cases the causes of decompression sickness (DCS) in divers can be explained by table/computer violations or uncontrolled ascents. When readily apparent causes are not found, the label "unexplained DCS" is typically given as the cause. We ascertain that in the majority of cases, the cause of the event can be explained by pairing a careful analysis of the dive profile with our Gradient-Perfusion Model (see preceding paper: "The Gradient Perfusion Model Part 1: Why and at what sites decompression sickness can occur").

The essence of the GPM is that tissues have variable perfusion requirements and this, coupled with gradients that develop with decreases in ambient pressure, determines whether DCS occurs. The model is supported by substantiating information, which considers six components from a decompression science perspective and shows how this information applies to our GPM (Table 1).

Perfusion is extremely variable in the human body. Blood flows where it is needed. This allows the 5-liter blood volume of the human to meet perfusion, oxygenation and nutrition demands for a variety of tissues that have different and changeable metabolic requirements in a circulation system that has greater than a 20-liter capacity [1]. The sympathetic nervous system and chemical mediators control where the blood flows. For fast tissues, such as the lungs, blood, heart and brain, perfusion is constant; any perturbations in perfusion can have disastrous consequences. For example, the brain, a fast tissue, receives 15 percent of the cardiac output but comprises only 2 percent of the body mass [1].

TABLE 1. INFORMATION SUPPORTING THE GRADIENT-PERFUSION MODEL

Component	Decompression Science Perspective	Applications to GPM
Perfusion	Responsible for ongassing and offgassing of inert gas secondary to changes in ambient and/or partial pressures (i.e. switching gas mixtures)	With decompression, at a certain point the gradient can exceed the ability for perfusion to carry-off the inert gas in the tissues and autochthonous bubbles form. For the Haldane model, the ratio was 2:1; for the Workman "m" value, i.e. amount of allowable supersaturation varied with the depth of the dive
Silent Bubbles	With adequate perfusion and "safe" gradients, dissolved and even small bubbles can be carried safely by the bloodstream to the lungs where they can be offloaded to the outside environment	Adequate perfusion and safe gradients allow inert gas to offgas after exposures to increased ambient pressures
Interruption of Circulation (e.g. placing a tourniquet on the arm before ascending)	Bubbles form, coalesce, and enlarge with ascent (Boyle's law), generating signs and symptoms of DCS	Adequate perfusion is necessary to transport inert gases from tissues to the lungs for exhalation
Overwhelming Gradients	Autochthonous bubbles form in situ	Signs and symptoms of DCS are a function of the type of tissue in which they present e.g. fast tissues, intermediate tissues, or slow tissues
Environments	Areas of slow moving blood flow (e.g. sinusoids, venous plexuses, blood reservoirs etc.) provide an environment for silent bubbles to coalesce and enlarge	Bends paraplegia attributed to bubble formation in Babson's plexus of veins in the spinal cord circulation
Pain only DCS and Non-Anatomical Neuropathies	Remains undefined; bubbles have not been discovered in symptomatic tissues	Microscopic bubbles in stretch sensitive organelles (Ruffini type 2 corpuscles) and adventitious (lining) tissues presumed to account for clinical presentations

Any decrement in brain perfusion (and oxygenation), that is to say a "steal syndrome," will lead to almost immediate loss of consciousness. If the fast brain, blood and heart tissues – in terms of ongassing and offgassing – do not maintain their baseline perfusion, gradients of gases in these tissues become so great that autochthonous bubble formation occurs, with occlusion of blood vessels in the brain that can have potentially devastating effects. These are the tissues that will be affected by rapid ascents.

The intermediate tissues such as muscles, skin, the viscera and subcutaneous tissue, and perhaps bone, have highly variable perfusion rates depending on their metabolic needs. When inactive, their perfusion and oxygenation requirements are minimal. Muscle blood flow varies as much as 50-fold between rest and high exertion levels [1-7]. A similar number is probably needed for wound healing and managing wound infections [8]. Their minimal requirement for perfusion while at rest is the reason harmful consequences do not typically occur with significant decompression stresses since on- and offgassing of tissues would be minimal in their low metabolic demand "resting" states.

Slow tissues, such as ligaments, tendons, joint capsules, and similar types, ongas and offgas slowly with changes in ambient pressure. This is because almost all gas exchange occurs via diffusion through tissue fluids of connective tissues, which have minimal perfusion and metabolic requirements. These are the tissues that develop symptoms with longer, deeper divers, even if the ascents are slow.

Incipient observations

Long before we formulated the Gradient-Perfusion Model, several striking observations led the first author to appreciate how important gradients and perfusion were in critical organs when subjected to decreases in ambient pressure (Table 2).

TABLE 2. GPM AND DISORDERED DECOMPRESSION

Case #	Site	Gradient Consideration	Perfusion Consideration	Comments
Incipient Observations				
1	Lungs, blood	Massive	Overwhelmed by gradients	Divers dead on reaching surface
2	Blood	Moderate	Substantial	CPR inadequate for offgassing inert gas load
3	Vestibular system	Moderate, secondary to 46 hour ongassing. With NAVY treatment Table 4	Moderate	Resolved with pure oxygen treatment table
Fast Tissue Observations				
4	Brain, spinal cord	Substantial	Substantial	Temporary "steal" situation after loss of consciousness and sympathetic nervous system vasomotor control
5	Brain	SAA	SAA	SAA
6	Brain	Minimal	Substantial	"Silent" venous gas bubbles to arterial circulation through a patent foramen ovale
Intermediate Tissue Observations				
7	Spinal cord, viscera	Moderate	Significant	Blood volume insufficient to meet needs of digestive system and spinal cord simultaneously
8	Spinal cord	Moderate	Substantial	Disc bulge interfered with venous outflow
9	Vestibular system	Substantial	Minimal	Four rapid ascents and no safety stop in 38 minutes, 52 maximum depth working dive
Slow Tissue Observations				
10	Peripheral nerve	Moderate	Substantial	Flexed elbow interfered with offgassing. The increased gradient from altitude was sufficient to generate bubbles from the residual inert gas in the tissue
11	Peripheral nerve	Minimal	Substantial	Raynaud's phenomenon prevented adequate offgassing of nerve innervation to hands
12	Joints and peripheral nerve	Moderate	Substantial	Perfusion compromised enough by dehydration plus altitude excursion that gradients were sufficient to generate bubbles

CASE 1: Explosive decompression

Two helmeted surface-supplied divers breathing air lost communications with topside at near the time they reached their 180-foot depth. The decision was made to surface the divers as quickly as possible and immediately recompress them with surface decompression. Upon reaching the surface, the divers were unconscious and unresponsive to resuscitation. Autopsies revealed extensive intrapulmonary and intravascular bubbles.

GPM considerations

Obviously overwhelming gradients occurred in the ultra-fast tissues of the lungs and blood with the precipitous ascent, even though the divers were only momentarily at bottom depth. Diagnoses included the "chokes" (bubbling in the pulmonary vasculature) and decompression illness (intravascular bubbles). Even though ultra-fast tissues were involved, the massive gradients exceeded the ability of the blood and lungs to offgas and offload the inert gas.

CASE 2: Loss of consciousness at depth

An experienced salvage diver was doing a bottom search at a 145-foot depth using an umbilical air supply from a diving bell/personnel transfer capsule (PTC). After a brief exploration, less than five minutes, the diver poked his head into the open bottom hatch of the PTC and told the tender he was OK. The diver then resumed his search, but a couple minutes later did not respond to a line tug signal. Upon instructions from topside, the tender pulled the diver into the hatch, but was not able to bring him into the PTC.

The PTC was then brought to the 30-foot depth, where three scuba divers were able to push the diver into the bell with the aid of the tender. After this, the patient and tender were transferred to a deck decompression chamber (DDC). The DDC was pressured to 100 feet of seawater; a doctor was inside and initiated cardiopulmonary resuscitation (CPR) for the unconscious diver. The chamber was decompressed on an omitted decompression schedule. Intravenous normal saline was started, and CPR was continued.

At the 30-foot level and after more than two hours of CPR, the victim's veins began to bulge. When aspirated, frothy pink blood was returned but there was no return of cardiac activity. Cardiopulmonary resuscitation efforts were discontinued and the victim was pronounced dead. An autopsy failed to establish a cause of death. The supposition was that the patient developed a fatal arrhythmia and lost consciousness at depth.

GPM considerations

Obviously overwhelming gradients developed as the DDC was slowly decompressed with the omitted decompression table. The CPR efforts were not sufficient to offgas the inert gas load. This resulted in autochthonous bubble formation in the diver's bloodstream in the absence of any cardiopulmonary function. The tender who assisted with CPR during the omitted decompression management had no signs or symptoms of DCS.

CASE 3: Inner ear decompression sickness

A Navy Medical Officer "rode out" a 46-hour U.S. Navy Treatment Table 4 as an inside attendant for a victim of decompression illness. About 45 minutes after completing the treatment, he took a hot shower and immediately experienced vertigo, dizziness and nausea. He was diagnosed with inner ear (vestibular system) DCS. Rather than repeating another 46-hour Table 4 treatment within an hour, a monoplace hyperbaric chamber was started. He underwent a one and one-half hour hyperbaric oxygen treatment without air breaks. His symptoms resolved during treatment.

GPM considerations

In this situation the hot shower, coupled with presumed bubble enlargement (Charles' Law) in the bloodstream, interfered with the circulation to the vestibular system. We hypothesize that ongassed nitrogen in this organ, generated from the 46-hour long treatment table, then produced a gradient large enough to generate autochthonous bubbles and cause the inner ear presentation of DCS. The excellent response to the pure oxygen treatment table was attributed to bubble size reduction with pressurization (Boyle's law) and inert gas washout with breathing pure oxygen.

FAST-TISSUE PERFUSION CASES OF DCS AND THE GPM

CASE 4: Transient loss of consciousness on the surface after a decompression stop requiring dive

A commercial diver who was a previous high-functioning U.S. Navy SEAL completed a working dive. The dive, which required a decompression stop, was completed without incident. Later, while working topside, a high-tension cable parted and struck the diver's head, rendering him unconscious for a few moments. After regaining consciousness, the diver showed significant cognitive function impairment (equivalent to a U.S. fourth-grade performance level in primary school). In addition, he experienced diffuse non-anatomical head and spine pain that required narcotic analgesics for management. After multiple hyperbaric oxygen treatments, rehabilitation efforts and neurological assessments, the diver remained totally disabled.

GPM considerations

The transient loss of consciousness from the head injury led to disruption of autoregulation enough that it no longer provided adequate perfusion to the brain and spinal cord. We postulate that with blood flow insufficient to offload the residual inert gas in these two nervous system structures, offgassing was impaired. This caused diffuse autochthonous bubble formation in the brain and spinal cord, with nervous system injury not conforming

to anatomical parameters. In essence, it was a "steal syndrome" of blood being diverted from the brain and spinal cord during the transient loss of sympathetic nervous system vasomotor control and best explained by the GPM.

CASE 5: Transient loss of consciousness on the surface after an emergency ascent

A female scuba diver with several hundred previous dives experienced acute onset of a severe headache after reaching a 90-foot depth. She aborted the dive and made a controlled ascent, but upon reaching the surface lost consciousness. Dive support personnel immediately retrieved her. On the surface, after emerging from the temporary loss of consciousness, she manifested diffuse neurological involvement. This included weakness, balance problems, visual disturbances, speech impairment and deficits in thought processing. These signs and symptoms improved only incompletely with hyperbaric oxygen (HBO_2) treatments, rehabilitation, walking aids, and special eyeglass prescriptions.

GPM considerations

It appears that the severe headache with transient loss of consciousness at the surface disrupted offgassing from the brain enough to significantly change the normal gradient-perfusion responses. The transient loss of autoregulation of cerebral perfusion resulted in a "steal syndrome" with respect to the brain. As in the previous example, diffuse bubbling in the brain caused disparate brain injury. Although strongly motivated, the patient was advised not to scuba dive again, in accordance with our GPM's labeling of the problem as a "deserved" (expected) DCS occurrence with severe neurological residuals (See Figure 4 in Part 1 of this series).

CASE 6: Post -dive Valsalva-like effect and immediate loss of consciousness

A recently certified young male completed an uneventful 20th recreational scuba dive that was well within dive table no-decompression limits. During the surface interval he began laughing hysterically after hearing a joke and immediately collapsed. An immediate HBO2 recompression treatment resulted in full recovery. The patient wanted a clearance to resume scuba diving, saying it was good for his emotional health. A cardiology consultation and resultant bubble study demonstrated a patent foramen ovale (PFO).

GPM considerations

The hysterical laughter presumably increased the diver's intrathoracic pressure sufficiently to force the "silent" bubbles in his venous blood to pass through the PFO, enter the arterial circulation and be carried to his brain. While not specifically a gradient-perfusion problem, perfusion was, in fact, altered by his PFO, with movement of "silent" bubbles into the left side of the heart, travel ,to the brain and resultant loss of consciousness rather than instantaneously offloading the "silent" bubbles through the lungs. Although the episode was initially cataloged as an unexplained DCS event, the discovery of the PFO justified labeling it as disordered decompression. Repair of the PFO was advised before resuming scuba diving.

INTERMEDIATE PERFUSED TISSUE CASES OF DCS AND THE GPM

CASE 7: Extraordinary case of visceral and lower-extremity paralysis

A healthy septuagenarian female underwent a "pep-up" exposure in a home-constructed hyperbaric chamber breathing air. After a 30-minute pressurization (slow due to the small air compressor), a bottom time equivalent to a depth of 72 feet of seawater was maintained for 30 minutes. Following a 20-minute ascent, she felt "great" following the exposure. After eating a "hearty" meal and four hours after the exposure was completed, she experienced abdominal pain and was unable to feel sensation or move her lower extremities. After transport to a hospital, a computerized tomography scan showed gas in her abdominal and pelvic veins (See case report in Part 3 of this three-part series). While breathing oxygen over the next couple of hours, her symptoms improved and resolved completely with a delayed HBO_2 treatment.

GPM considerations

This case of unexplained DCS was clarified by our GPM. The increased perfusion requirements of the patient's finite blood volume was insufficient to offgas simultaneously the inert gas accumulated during the dive from the spinal cord tissues and digest her "hearty" meal. In this example, deficient/insufficient perfusion and sufficient gradients allowed autochthonous bubbles to form and generate symptoms in the symptomatic areas.

CASE 8: Disc protrusion interfering with spinal cord venous outflow

A healthy male septuagenarian who was a very experienced scuba diver developed bilateral lower-extremity numbness after a series of dives, none of which violated his dive computer. With oxygen breathing, his symptoms cleared rapidly. The diver required medical clearance after the above incident in order to continue diving as a volunteer at a local aquarium. His physical examination demonstrated an amazingly fit male with an entirely normal neurological exam. Collaboration with a neurologist resulted in obtaining a magnetic resonance imaging study. The study demonstrated a markedly bulging thoracic disc. The problem was resolved with back surgery, and the patient returned to diving.

GPM considerations

The patient's post dive symptoms corresponded to bubble formation in the Babson plexus of veins surrounding the spinal cord. The disc protrusion apparently impeded venous outflow in this intermediate perfusion category of tissues to interfere with sensory transmission of the spinothalamic tracts of the spinal cord. This disordered DCS event was resolved with discectomy.

CASE 9: Concussion trauma and repeated ascents leading to inner ear DCS

A 45-year old commercial diver with more than 400 uneventful dives was doing very hard work at a maximum depth of 52 feet for 38 minutes. Challenges that made the dive strenuous included concussion waves from nearby underwater demolitions associated with the bridge construction project on which he was working as well as the need to surface four times in order to change locations for his bottom work. The ascents were made without safety stops. After surfacing from the final dive, the diver experienced tingling in his fingers and toes, nausea, vomiting, tinnitus, vertigo and marked disequilibrium. No focal neurological deficits were identified. After the third hyperbaric oxygen recompression treatment, his symptoms resolved completely. An extensive neurotology workup was normal and a transesophageal echocardiogram bubble study did not demonstrate a patent foramen ovale. The patient has returned to commercial diving without incidents.

GPM considerations

This otherwise unexplained (i.e., well within dive table safe limits) case of DCS is another example of disordered decompression. It was attributed to the combination of hard work while underwater, four ascents without safety stops and concussion stresses from underwater explosions occurring during a relatively short dive. We believe the repeated, rapid ascents and lack of safety stops exceeded the ability for perfusion to offgas the inert gas accumulated in the diver's vestibular system. The resultant gradient in the vestibular system was sufficient for autochthonous bubbles to form and generate symptoms. The rapid improvement with HBO_2 treatments supports our conclusion that this was a disordered decompression event.

SLOW TISSUE PERFUSION CASES OF DCS AND THE GPM

CASE 10: 'Tourniquet effect' plus altitude

A scuba instructor in her early 30s made several non-challenging dives (each shallower than 40 feet) as part of a Caribbean vacation. Some 36 hours later she returned to the United States. After falling asleep during the commercial flight, she awoke with numbness and total paralysis of her right hand. After landing she immediately sought a hyperbaric medicine consultation. Dramatic resolution of symptoms to full recovery occurred as soon as the patient was pressurized to three atmospheres absolute pure oxygen.

GPM considerations

Several deductions are apparent. First, the problem leading to her symptoms involved slow tissues with regard to offgassing, with apparent residual nitrogen in these tissues even 36 hours after completing scuba dives. Second, offgassing was apparently impaired by falling asleep with her right elbow in a hyperflexed position. Third, the hypobaric environment of the airplane cabin at altitude fostered bubble enlargement. Fourth, the non-anatomical distribution of the symptoms is best explained by bubble formation in the adventitial tissues surrounding nerves to the hands. Finally, the immediate response to pressurization confirmed that bubble formation was mitigated by compression alone and suggests they were entirely a mechanical problem and decreased in size, as predicted by Boyle's law. With this analysis, an otherwise case of unexplained DCS had identifiable disordering events.

CASE 11: Hand numbness in a diver with Raynaud's syndrome

A scuba instructor in her 40s was conducting checkout dives in 58°F water at a depth of 30 feet. Although she wore gloves they were not the usual neoprene ones she routinely used during her dives. After surfacing, her hands were cold and numb. Dry and later wet (warm water) rewarming warmed the hands, but numbness persisted. The patient contacted Divers Alert Network and was referred for a hyperbaric medicine consultation. With hyperbaric oxygen recompression, the symptoms improved with the initial treatment and resolved after two washout treatments.

GPM considerations

As in the previous case, the signs and symptoms developed because of interference with perfusion. In this diver's situation, vasoconstriction secondary to Raynaud's syndrome and precipitated by cold water slowed circulation enough that sufficiently large gradients developed for bubbling to occur. Again, we postulate that they occurred in slow, adventitial tissues surrounding the sensory nerves to the hands and account for the non-anatomical distribution. The diagnosis of disordered decompression was substantiated by the diver's response to HBO_2 recompression.

CASE 12: Dehydration with altitude excursion after a scuba dive

A young woman completed a supervised scuba checkout morning dive with a maximum depth of 40 feet for less than 30 minutes. To avoid needing to empty her bladder, she did not drink any fluids after the previous night's dinner. The dive was uneventful. Later in the day she joined friends to hike in a local mountain range, but did not hydrate herself. As she approached the 2,500-foot summit, she developed pain in one elbow, and non-anatomical paresthesias in both upper extremities. After contacting Divers Alert Network, she reported to our hospital emergency department. Intravenous hydration was initiated, but her symptoms persisted. We were consulted for a case of possible unexplained DCS. The diver's symptoms resolved after the first hyperbaric oxygen recompression treatment. The discharge diagnosis was Type I DCS secondary to dehydration and altitude exposure, resulting in disordered decompression.

GPM considerations

Dehydration interfered with the diver's circulatory system's ability to offgas adequately. The modest altitude excursion was sufficient to develop gradients large enough to allow autochthonous bubble formation in the elbow and the upper-extremity nerve sheaths. As described in Part 1, the Ruffini Type 2 corpuscle provides the best explanation for elbow pain symptoms and the formation of microscopic bubbles in the adventitial coverings of nerves for non-anatomical distribution of her upper-extremity paresthesias. Presumably, her lower extremities were spared due to a steal syndrome attributed to increased blood flow to the lower extremities required for hiking. The excellent response to the HBO_2 recompression treatment confirmed the diagnosis of DCS.

DISCUSSION

The evolution of our gradient-perfusion model (GPM) started with the first author's proximity to the cases described in the incipient observations section. The obvious massive gradients and/or overwhelming of the circulatory system to offload inert gas appeared to be the essential contributors to decompression sickness.

The second stage in the evolution of our model was the appreciation that the body has a finite blood volume, but the capacity of the vascular tree is a 4-to-5 multiple of blood volume. This has been especially appreciated in the healing of problem wounds, where perfusion and metabolism need to increase manyfold for healing to occur, but minimal needs exist in the healed, steady-stage condition [8]. This conclusion has obvious counterparts to ongassing and offgassing of the enormous variety of tissues in the human body in responses to changes in ambient pressure.

The final stage in generating the GPM was the appreciation that compartmentalization, no matter to what degree carried out, provided only generalizations as to bubble formation. However, it is obvious that there are three major tissue types with regard to perfusion. They include:

1) Fast tissues that have and require a continuous blood supply;
2) Intermediate tissues that have a blood supply proportional to their metabolic needs, which is highly regulated by the sympathetic nervous system and chemical mediators; and

3) Slow tissues that receive their oxygen and metabolic needs by diffusion from surrounding tissue, generally that have minimal perfusion needs such as connective tissues.

Although other factors may influence the occurrence of decompression sickness, such as carbon dioxide, obesity, disrupted circulation from old injuries, peripheral vascular disease and microparticles, we believe they are only a minor consideration in the total picture of gradient-perfusion causes of DCS.

With respect to the terminology "unexplained DCS," we believe the terminology "disordered decompression" is the preferred term and that disordering events should be sought to explain the occurrence of DCS when table/computer violations are not apparent. A prime example of seeking the cause of unexplained DCS is that of the patent foramen ovale. Once PFO is confirmed, the decompression "hit" is no longer unexplained, but rather an example of disordered decompression resulting from this anatomical defect. Immersion pulmonary edema, although not restricted to diving activities, may also have associations with the GPM.

Hypertension and associated reduced right ventricle diastolic relaxation during the cardiac cycle results in engorgement of the pulmonary vasculature. This causes pulmonary edema. This is precipitated by immersion and presumably aggravated by changes in ambient pressure and increased exhalation resistance with the scuba regulator [9,10]. The pathophysiology has relationships to the GPM since alterations of perfusion as a consequence of heart function and changes in ambient pressure, even with as little as immersion to the neck level, can generate the symptoms in susceptible individuals.

Finally, our GPM makes the evaluation and management of DCS logical. Once the tissue type or types – i.e., fast, intermediate or slow – is ascertained, the next question in the evaluation is to determine what disordering events could have altered the gradient-perfusion relationships to the tissues causing the symptoms. With having these two pieces of information for almost any case, the cause of the DCS hit is no longer unexplained but can be viewed as a result of the disordering event or events. This has important clinical applications. It justifies HBO_2 recompression treatment in patients who are symptomatic after a dive, even when exposure to increased ambient pressure is minimal and far removed from a table or computer violation. Our maxim is if the diver has symptoms that are not anatomically explainable, a hyperbaric oxygen recompression treatment should always be done. With improvement or resolution of symptoms, the diagnosis of DCS is confirmed. The other clinical application is that the determination of the disordering event and residuals symptoms after treatment provide a guide for logical recommendations as to whether the injured diver should resume diving activities or refrain from diving (see Figure 5 in Part 1 of this three-part series).

CONCLUSIONS

By appreciating the tissues involved in the presentation of DCS signs and symptoms as well as recognizing disordering events to perfusion and gradients generated with offgassing, the cause of DCS can almost always be established. Based on the above information we recommend that the term "unexplained DCS" not be used and that "disordered decompression" replace it. The understanding of the perfusion physiology of fast, intermediate and slow tissues provides the "why" in terms of location of the signs and symptoms that occur. While numerous theories have been formulated for bubble formation (See Table 1 of Part 1 in this three-part series), our Gradient-Perfusion Model explains why the signs and symptoms of DCS have occurred in the locations they do and provides logical guidelines for DCS management. In this respect, our GPM complements the bubble formation hypotheses and carries them to the clinical evaluation and management realm. ■

Conflict of interest statement

The authors declare that no conflicts of interest exist with this submission.

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