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DIFFERENTIAL DIAGNOSIS OF DECOMPRESSION ILLNESS

Workshop Proceedings

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Differential Diagnosis of Decompression Illness Workshop

Wednesday, June 27, 2018 Schedule

TIME	LECTURE	SPEAKER
0800-0815	Welcome and Introductions	Petar Denoble, MD
0815-0900	Diagnosis of DCI: Current State of the Art	Richard Moon, MD
0900-0945	Neurological exam of injured divers	Wayne Massey, MD
0945-1030	Decompression illness and coincidental acute post-dive conditions in recreational and commercial divers	Matias Nochetto, MD
1030-1045	BREAK	
1045-1130	Diagnostic algorithms for DCI	Ian Grover, MD
1130-1215	French approach to differential diagnosis of DCI	Jean Eric Blatteau, MD
1215-1315	LUNCH	
1315-1400	Cardiorespiratory post dive conditions	Bruce Derrick, MD
1400-1445	Abdominal post-dive issues	Aaron Heerboth, MD
1445-1500	BREAK	
1500-1545	ENT issues	Nick Vandemoer, MD
1545-1630	Trauma, aches and pains	Jim Chimiak, MD
1630-1700	Q & A - Concluding remarks	Petar Denoble, MD and Alessandro Marroni, MD

Overall Goal

The aim of this workshop is to review the most common coincidental post-decompression conditions that could be confused for DCI and discussion of when and what additional testing is needed to avoid misdiagnosis. The topics will include serious neurological conditions, neurological conditions resistant to HBO_2 treatment, serious cardiorespiratory conditions, post-dive abdominal discomfort, cutaneous manifestations, and osteomuscular aches and pains.

List of Speakers

Richard Moon, MD

Anesthesiologist and pulmonary/critical care specialist at Duke University Medical Center. Medical Director, Duke Center for Hyperbaric Medicine & Environmental Physiology. Research endeavors include diving, altitude hypoxia, physiological and pathological effects of immersion. Current research projects include development of decompression procedures for diving at altitude, development of hypoxia warning system for rebreather divers.

Wayne Massey, MD, FAAN, FACP

Professor of Neurology Duke University Medical Center, Board of Directors DAN.

Matias Nochetto, MD

Director of Medical Services and Programs at Divers Alert Network (DAN). He received his medical degree in 2001 at Universidad de Buenos Aires (UBA), in Argentina and completed a 3-year clinical and research fellowship in hyperbaric and diving medicine from National Autonomous University of Mexico (UNAM) in Mexico City. Nochetto is also a diving instructor since 1999 and a National Oceanic and Atmospheric Administration's (NOAA) trained Dive Medical Officer.

Ian Grover, MD, FACEP

Medical Director, UCSD Division of Hyperbaric Medicine and Wound Healing. He is the co-chair of the 24/7 Critical Care Hyperbaric Committee and he also is a member of the UHMS Education Committee. His research interests include hyperbaric oxygen therapy for preconditioning, critical care in the hyperbaric chamber, and diving medicine.

Eric Blatteau, MD, PhD

Professor of diving and hyperbaric medicine, director of the department of diving and hyperbaric medicine at the Sainte-Anne military hospital in Toulon, South of France.

Bruce Derrick, MD, FACEP

Associate professor and program director, Undersea & Hyperbaric Medicine Fellowship; Division of Emergency Medicine, Center for Hyperbaric Medicine & Environmental Physiology at Duke University.

Aaron Heerboth, MD

HBO and emergency medicine physician in San Diego, CA. Attended Cornell University medical school and completed his residency in Emergency Medicine at UCSD.

J. Nicholas Vandemoer, MD

Otolaryngology and Diving and Hyperbaric Medicine physician. He is Board Certified in Diving and Hyperbatic Medicine. He has been an instructor in the UHMS and DAN Fitness for Diving course from 2004 to 2015. His medical work continues as a Utilization Management consultant. He continues his passion for diving and sailing from his home on Cape Cod, Massachusetts

James M. Chimiak, MD

Medical Director at Divers Alert Network. He is a Navy flight surgeon and diving medical officer. Dr Chimiak holds board certifications for Anesthesiology, Undersea & Hyperbaric Medicine in addition to Pain Management.

Diagnosis of DCI: Current State of the Art

Richard E. Moon, MD

Decompression illness (DCI) is a group of syndromes caused by bubble formation due to an acute reduction in ambient pressure. DCI may be due to decompression sickness (DCS) or arterial gas embolism (AGE). The diagnosis of DCI is based upon (a) the setting (e.g., diving, acute altitude exposure) and (b) recognition of clinical manifestations. DCI is a clinical diagnosis, in which lab testing or imaging are not helpful.

DCS, due to in situ bubble formation caused by inert gas supersaturation, requires a sufficient inert gas load. A review of DCS incidence after direct ascent to the surface following saturation dives to various depths revealed that DCS did not occur at a depth of 20 fsw or shallower.[1] Therefore, DCS is extremely unlikely if a diver's maximum depth did not exceed 20-25 fsw or greater. On the other hand, AGE can occur after rapid ascent from depths as shallow as 3 fsw. Patterns of symptoms observed in decompression illness are helpful. Over 60% of recreational divers with decompression illness experience either pain, numbness or paresthesias. More severe symptoms such as dizziness or vertigo, motor weakness, incoordination or loss of consciousness are less common although more specific.[2] Onset time of symptoms is relatively rapid. Median onset time of pain and paresthesias in recreational divers is 2 hours after surfacing.[3] More serious symptoms such as cerebral or spinal cord manifestations occur more rapidly, with 90% presenting within 2 hours.[4] In recreational divers joint pain and paresthesias occur more frequently in the arms compared with the legs. Motor weakness is equally common in arms and legs.[5]

AGE typically occurs after rapid or breath-hold ascent. Classically, patients exhibit manifestations similar to acute stroke. Loss of consciousness often occurs, although more subtle manifestations can also occur. Onset is usually more rapid with AGE compared with DCS, typically within a few minutes of surfacing. Manifestations of venous gas embolism (VGE) occur after a significant depth-time exposure and may include cough, dyspnea, and pulmonary edema. The right-to-left shunt can result in AGE.

On-site Doppler or transthoracic echo can be used to assess for the presence of VGE. However, its presence is not sufficiently specific to be useful diagnostically.[6] VGE tends to resolve quickly unless by the time of symptom onset VGE are likely to have resolved.

Inflation of a sphygmomanometer around joints afflicted with pain due to DCS has classically been described as a diagnostic test, with the resolution of pain if the diagnosis of DCS is correct. However, Rudge at all reported that pain is reduced with this maneuver in only 61% of patients.[7]

For any individual with suspected DCI, evaluation should include a complete neurological exam. Performing this in a supine patient on a stretcher is not usually adequate to exclude neurological DCI. The appropriate evaluation must include (if the patient is capable) of assessment of gait, heel-toe walking and sharpened Romberg. Heel-toe walking should be obtained with eyes open and closed, forwards and backwards. Inability to perform these maneuvers should be recorded as 'abnormal'. Sharpened Romberg testing requires placing one foot in front of the other (heel-to-toe), arms crossed and eyes closed. The patient should be able to maintain this position for 20 seconds or greater. These maneuvers should be performed with bare feet on a hard floor.

Occasionally, cortical abnormalities occur, and can be detected by asking the patient to do serial 7 subtraction, interpret proverbs (e.g. "a stitch in time saves 9", "a bird in the hand is worth 2 in the bush") or draw a clock. Inner ear DCS (IEDCS) most commonly affects the vestibular system and is associated with true vertigo, nausea, vomiting, and nystagmus. Onset is usually within 2 to 3 hours of surfacing from a dive deeper than 60 fsw (although it can occur after shallower dives).[8] Hearing loss can also occur in IEDCS, although in recreational divers it is less common than vestibular manifestations. IEDCS is the only form of DCS for which specific electrophysiological testing is helpful, and indeed recommended. After recompression treatment, audiometry and vestibular testing are used to assess the degree of resolution. Clinical assessment by itself is inadequate, as imbalance and vertigo will spontaneously resolve even in the presence of residual vestibular damage, which can only be detected by physiological testing. The main differential diagnosis for IEDCS is inner ear barotrauma (labyrinthine window rupture, IEBT). Manifestations of IEDCS and IEBT can be identical, but the treatments are quite different.[9] Onset of IEBT tend to occur more commonly during descent, while IEDCS occurs after the dive. IEBT generally has associated manifestations of middle ear barotrauma such as tympanic membrane redness and blood in the middle ear and is more frequently associated with hearing loss, vs. vertigo.

Intravascular bubbles tend to cause endothelial damage and extravasation of plasma. Severe DCS is therefore often associated with hypovolemia and hemoconcentration, for which intravenous fluid resuscitation is recommended.[10,11]

Except to exclude complications (e.g., pneumothorax) or unrelated condition (e.g. cerebral hemorrhage) radiographic imaging is not helpful for diagnosing DCI. Acute imaging of the brain is often ordered in an attempt to look for arterial gas bubbles in suspected cases of AGE. However, intravascular gas is observed in only a minority of AGE cases.[12] Similarly, attempting to use spinal cord MRI to confirm or exclude the diagnosis of DCS is not diagnostically helpful.[13] Therefore, most hyperbaric clinicians recommended not imaging suspected cases of DCI, but rather to initiate recompression treatment as soon as possible.

Not every clinical abnormality occurring after a dive is due to DCI. Coincidental onset of other conditions can be mistaken for DCI. Prior history, patient comorbidities or features that are not typical of DCI should, therefore, initiate consideration of other diagnoses. Some conditions that have been mistaken for DCI are listed in Table 1.

Table 1. Differential diagnosis of neurological events following a dive

Barotrauma (inner ear barotrauma, alternobaric barotrauma, facial baroparesis)
Immersion pulmonary edema
Other cerebral pathologies
Embolic stroke
Intracranial hemorrhage
Seizures
Migraine
Guillain-Barré syndrome
Other spinal cord conditions (e.g. epidural hemorrhage)
Transverse myelitis
Spinal cord compression (trauma, hemorrhage)
Seafood toxin ingestion (ciguatera, paralytic seafood)
Porphyria

Patients with a prior history of seizures, migraine or porphyria who presented with manifestations consistent with one of these diagnoses should be carefully evaluated assigning the diagnosis of DCI. Embolic stroke is likely to occur in patients with risk factors, such as older age, smoking, diabetes and hypertension. Rapid onset of stroke-like manifestations with a headache in the absence of rapid or breath-hold ascent may be associated with intracranial hemorrhage or spontaneous dissection of the carotid or vertebral arteries. Spontaneous epidural hemorrhage of the spine is often painful. Ingestion of seafood during the day before symptom onset may trigger consideration of ciguatera or paralytic seafood toxicity. The onset of neurological manifestations greater than 2-6 hours after surfacing from a dive should trigger consideration of diagnoses other than DCI.

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Neurologic Evaluation of DCI

Wayne Massey, MD

The primary purpose of this presentation is to review the neurological examination and to discuss features which will aid in making the appropriate diagnosis to aid in performing the accurate therapy for each suspected of Decompression Illness (DCI).

Diagnosis of DCS and AGE

When a diver develops neurological manifestations shortly after surfacing from a dive, it is frequently impossible to differentiate between DCS and AGE. They often occur together in the same patient. The differentiation is rarely of clinical importance as the treatment for both conditions is essentially the same. The term decompression illness (DCI) is often used to indicate either.[1]

Manifestations of DCS can vary in severity, from mild (typically paresthesias, joint pains, fatigue) to manifestations involving the inner ear (vertigo, hearing loss) and spinal cord (paraplegia, triplegia, quadriplegia).

The most common neurological manifestation is paresthesia, often without objective hypesthesia. When hypesthesia is present, it is usually non-dermatomal as it is central in origin. However, occasionally anesthesia is present in a peripheral nerve distribution which must be diagnosed accurately to avoid a therapeutic mishap.[2] We will discuss further below.

More serious manifestations include paresis or paralysis, disturbance of vision, bowel and bladder dysfunction and vertigo. Alert clinical perception must avoid mistaking fatigue for weakness for example.

Most often, the target organ is the thoracic spinal cord, perhaps due to the venous vascular anatomy of the spinal cord as veins seem to allow for nitrogen bubbles to collect producing venous infarction.

Cerebral involvement occurs in 30% of cases of type II decompression sickness.[3] Divers with cerebral involvement may complain of confusion, lethargy, encephalopathy delirium, difficulty with concentration, poor judgement, and visual disturbances.

Serious neurological manifestations usually occur shortly after surfacing, while milder symptoms may be delayed for several hours.[3] In Neurological DCS, 90% of cases reported cerebral manifestations occurred within 30 minutes after surfacing, while 90% of spinal cord manifestations occurred within 4 hours.[4]

Divers experiencing AGE or pulmonary over-inflation can experience pain and respiratory distress, coughing, hemoptysis, but also cortical symptoms of headache, altered consciousness, seizures, hemiparesis, quadriparesis, and cortical blindness. When AGE occurs following ascent from a dive in which there has been a significant depth-time exposure, where inert gases in some tissues may be close to supersaturation, the clinical manifestations may resemble those seen with DCS, such as spinal cord involvement.[5]

Neurologic history and examination

The diagnosis of both DCS and AGE is based on the clinical examination, including the neurological examination, and the dive history. Laboratory and imaging studies rarely add information.[9]

Important diagnostic factors for DCS: 1) a neurological symptom as the primary presenting symptom; 2) onset time to symptoms; 3) joint pain as an initial symptom; 4) symptom resolution after recompression treatment and 5) maximum depth of the last dive. Age, gender, or physical characteristics were not statistically important.[6]

Diagnostic factors for AGE include: 1) the onset time of symptoms; 2) altered consciousness; 3) any neurological symptoms as an initial symptom; 4) motor weakness, and 5) seizure as the early event.

Classification: The original and most widely used classification of DCS divides manifestations into Type I (originally defined as symptoms without signs) and Type II (physical signs present, usually neurological).[7] The definitions have changed slightly since then. Type I DCS is now defined in the US Navy Diving Manual as including joint pain and symptoms involving the skin (cutaneous symptoms), or swelling and pain in lymph nodes. Type II DCS includes neurological, inner ear, and cardiopulmonary.

Review of neuroanatomy facts

In cases with neurological manifestations, localization of the lesion(s) is the first and primary step. The historical temporal profile of the event is essential and helpful. However, a thorough neurological examination is necessary because any CNS symptoms and signs may help to determine the location of the injury, and to establish the diagnosis and subsequent treatment plan.

Seizures: Seizures occurring in DCI are almost always generalized. If there is a history of a focal onset, a focal structural lesion is suspected, and it may be an antecedent lesion to this presentation. An observer is usually the most common descriptor. Therapeutic requirements expand if focal onset. Prevention of recurrent seizures and observation for evidence of aspiration are initial major treatment issues. Complicating factors include the prevalence of non convulsive events is unknown.[7]

STRENGTH EXAMINATION

Upper motor neuron (UMN): Upper motor neuron connects supraspinal control centers with the spinal cord and provides modulation. In case of injury, after a period of "spinal shock," reflexes recover and become exaggerated, and muscle tone becomes spastic. The muscle tone and paresis, the pattern are important in the evaluation. (see Table 1)

Cortical lesions produce contralateral patterns of spasticity which are in upper extremities more expressed in flexor muscles than in the extensors, and in the lower extremity, the extensors have more tone and strength than the flexor muscles. On examination, the biceps, forearm flexors, wrist flexors, and finger flexors show more power or tone than the deltoid, triceps or wrist extensor muscles (contracted in posture). In the leg, the quadriceps and posterior compartment muscles predominate over the iliopsoas, hamstrings, and anterior compartment muscles (leg is extended in posture). This is a very reliable sign of UMN lesion. Anatomic lesion location can produce bilateral spastic examination in lower brainstem below mid pons or along the spinal cord. If location is cortical, the contralateral face is weak with spared forehead (usually), but in the mid pontine location, one sees the lower motor

Table 1. Upper Motor Neuron vs. Lower Motor Neuron Syndrome

	Upper Motor Neuron	Lower Motor Neuron
Muscular weakness	Spastic paresis	Flaccid paresis
Atrophy	No atrophy	Muscle wasting
Deep tendon reflex	Increased (clonus)	Decreased (Absent)
Pathological reflex	Babinski and Hoffman	No
Superficial reflex (abdominal)	Absent	Absent
Fasciculation or fibrillation	Absent	May be present

weakness of the face (entire side weak) and contralateral body affected. Description of the amount of strength loss is usually made based on the standards published by the Medical Research Council (MRC), but the extent of strength demonstrated still relies on the examiner's judgment. (See photos on pages 14 and 15)

The MRC muscle power scale[10]

Reflexes are increased (hyperreflexia) when affected by an upper motor neuron involvement. This is contralateral if the lesion is focal on one side above the Anterior Horn cell of the spinal cord of the root level for any reflex.

If the spinal cord is involved bilaterally, the reflexes are increased bilaterally below the level of injury. Any lower motor neuron involvement, perhaps preexisting neuropathy, will not allow the hyperreflexia evidence to be demonstrated. Pathologic reflexes, in the face, hands, or response in the feet will give additional localization information. A unilateral extensor plantar response, Babinski response, suggests a contralateral upper motor neuron location.

Table 2. MRC muscle power scale

Grade 5: Muscle contracts normally against full resistance.
Grade 4: Muscle strength is reduced but muscle contraction can still move joint against resistance.
Grade 3: Muscle strength is further reduced such that the joint can be moved only against gravity with the examiner's resistance completely removed. As an example, the elbow can be moved from full extension to full flexion starting with the arm hanging down at the side.
Grade 2: Muscle can move only if the resistance of gravity is removed. As an example, the elbow can be fully flexed only if the arm is maintained in a horizontal plane.
Grade 1: Only a trace or flicker of movement is seen or felt in the muscle or fasciculation is observed in the muscle.
Grade 0: No movement is observed.

Examples of conditions in which there is UMN or LMN must as always be considered in the context of localization of the lesion. Upper Motor Neuron intracranial lesions include hemorrhage, thrombosis or embolism to the cerebrum or a lacune in the internal capsule or brainstem. Spinal cord transection (quadriplegia, triplegia) or cord hemisection (Brown-Sequard) also gives upper motor neuron signs. Lower Motor Neuron signs occur from lesions of the spinal root or the peripheral nerve (compressive, traumatic, GBS) but also occur with anterior horn cell involvement (poliomyelitis, motor neuron disease).

Coordination and cerebellar testing. The midline of the cerebellum, vermis, is involved in maintaining truncal and gait stability. A Romberg test, standing with eyes closed and including tandem assessment, will evaluate the midline cerebellar function. Upper extremity finger to nose to finger testing and rapid alternating evaluation of upper and lower extremities are ipsilateral to the location of the cerebellar hemisphere involvement. Remember, ataxia can come from many causes.

Many Romberg tests have been described (17!) including the sharpen test in divers is helpful.

Sensory testing: Sensory testing includes modalities of touch, pinprick, temperature, vibration, and joint position sense.

The sensory pathways to the cerebral cortex or thalamus are separated in the spinal cord and lower brainstem until rostral brainstem/thalamus. This can produce several patterns.. Testing for a sensory level over the chest and abdomen (T4 to T10 most often) is essential and quite helpful in the localization of problems to the thoracic spinal cord. Sensory testing can be objective and often requires detail questions of response and repetition of the testing. Graphesthesia (recognizing objects or written numbers in hands) and neglect evaluation can be added features to help with localization. Localizing any level can be very helpful in determining the therapy plan.

Sensory terminology is important. Paresthesias are abnormal sensations described as tingling, prickling, or pins and needles. Dysesthesias refers to discomfort or even pain over an area when triggered by touch or pressure. Hyperesthesia indicates increased sensitivity to touch, pinprick or temperature stimulus on examination. Anesthesia, complete loss of sensation, or hypesthesia, decreased sensation, is established by examination and not by a subjective report of the patient. If the stimulus is produced, then pallesthesia is vibration, and thermohypesthesia is from cold or warm stimuli.

Additionally, general vascular events in the central nervous system produce loss of sensation. Even thalamic lacunae give loss contralaterally before producing paresthesia or pain syndromes. This is not true in demyelinating lesions of the central nervous system which often produces paresthesias at the onset of symptoms. Also, peripheral neuropathy produces these sensations with or without sensory loss in an area.[7]

Bladder and Bowel. A history of the bladder and bowel function is essential to determine if it is preexisting or new in onset. Spinal lesions usually cause spastic bladders producing retention. Lower motor, sacral, lesions or parasagittal location of the cortex produce incontinence usually. Patient history is important, but clinical findings of perianal sensory loss or reduced rectal tone must not be overlooked.

How to assess the strength

Sensory testing is always subjective since a verbal response is needed and hopefully will be consistent and reliable. Muscle testing can also be subjective, but the examiner must try to avoid this situation. Sometimes you just cannot!

Encouraging the patient to give full effort should be emphasized. "Cheerleading" often helps. Less than full effort may be due to pain, stress, wanting something to be found, and so on. Be sure that the appropriate position for muscle testing allows for the maximum effort by the individual. This assessment can be accurate only if it is fully performed consistently.

Occasionally the issue of facial asymmetry is raised: nasolabial fold reduced on one side; eye blink less on one side; eyelid ptosis; facial synkinesis. Evaluation of an old picture may help (driver's license or "selfie").

Nonphysiologic findings

Unfortunately, non-anatomic findings occur and must be recognized and factored into the therapeutic decision making efforts. In any neurologic examination, these situations need to be assessed. Some examples are the following:

Mental status: affect (cheeriness); inconsistent exam; history of other unusual presentations.

Cranial nerves: persistent squint/wink; alternating fixation of vision; convergence spasm in horizontal visual testing OU; tunnel vision results of testing; non-anatomic cranial nerve changes.

Motor: tremulous giveaway weakness or other lack of effort; inconsistent motor testing; Hoover's sign when testing leg elevation while supine; a person unable to squat but "hovers" over a chair before rising or sitting. Do not record a specific numeric label to the motor examination if unsure.

Reflexes: Non-anatomic results; inconsistent, poor relaxation of tone, guarding, augmentation.

Sensory: inconsistent, non-anatomic; splits midline on testing; vibration differs over the skull. Functional sensory loss is frequently nonanatomic, and repeated examination may demonstrate differences in the demarcation of the sensory deficits. Sometimes patient drawing their deficits on a dermagraft is revealing.

Cerebellar/coordination inconsistent, non-anatomic gait such as astasia-abasia; gait improves with speed of walking; non-anatomic findings.

Some red flags on evaluation

The temporal profile of symptoms need not be reemphasized to caregivers who evaluate for DCI. Any symptom associated with the diving event must be taken seriously. Dive physicians have the responsibility to react appropriately with the best judgment for therapy. Giving the appropriate help and avoiding inappropriate therapy is the challenge. Also, of course, deciding on the correct diagnosis may alter the steps taken next.

For example, a stroke occurring at the unfortunate time of post diving must not be confused with DCI of course. Immediate therapy may be required for each but is very different for each.

Stroke events are usually very localizable. If multiple emboli occur, or with mycotic emboli, then symptoms can be difficult to localize of course. However, clinical findings help to localize any stroke lesion. Understanding the vascular anatomy to the central nervous system is therefore essential in localization.

Stroke, is most of the time, localizable (except if multiple emboli or mycotic emboli occur). However, AGE sometimes may produce localizable symptoms.

When specific cortical findings accompany hemi-sensory or hemiparetic symptoms and signs, then localization is easier. Homonymous hemianopia is occipital or parietal if incongruent and cortical. Aphasia, if global in a right-handed person, suggests left middle cerebral artery (MCA) occlusion. If receptive aphasia, it is a left MCA posterior inferior branch occlusion or if expressive aphasia with face and arm weakness on the right in a right-handed person it is an occlusion of the MCA anterior superior branch. If only an arm or the leg or the face is symptomatic on the right, in a right-hander, then a lacunae in the posterior internal capsule is likely. Similarly, infarction can localize in many places giving specific anatomic symptoms and signs.

If the face is weak on one side and the body on the other, then localization is limited to a specific anatomic localization (pons).

Sometimes AGE produces localizable symptoms and signs which makes the next step to therapy very challenging.

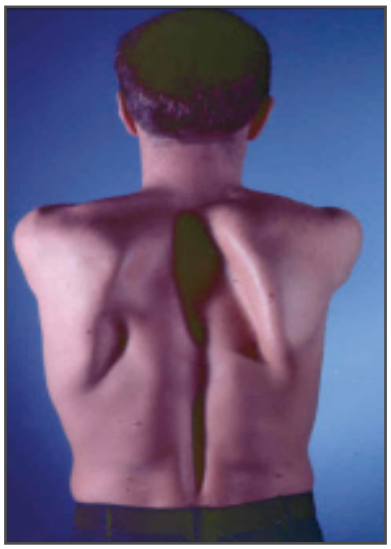
Nystagmus is often nonspecific. If nystagmus is in all directions, it is often due to medications. If nystagmus is lateralized and vertigo is present, then end organ ear disease is likely. (see ENT lecture) Vertigo due to vascular events is rarely isolated as posterior fossa strokes have accompanying symptoms.

Why is this important?

In ischemic stroke events, the use of tissue plasminogen activator and other methods to improve the vascular flow and reduce ischemic injury is time sensitive. The sooner, the better. However, the risk of bleeding is a concern and must be considered. Likewise, treatment of AGE or DCS is time sensitive, and hyperbaric therapy goals exist as well. Correct diagnosis reduces risks of delay in both.

Acute myelopathy/ cord compression is also challenging at times. Acute onset of cord localizing findings in a previously asymptomatic individual suggests DCS following a dive. However, in a person with known cord compression risk (cervical stenosis; scoliosis; neurofibromatosis) or with known myelopathic risks (MS; Chiari malformation; syrinx; venous malformation) the acute new symptoms could be caused by other reasons.

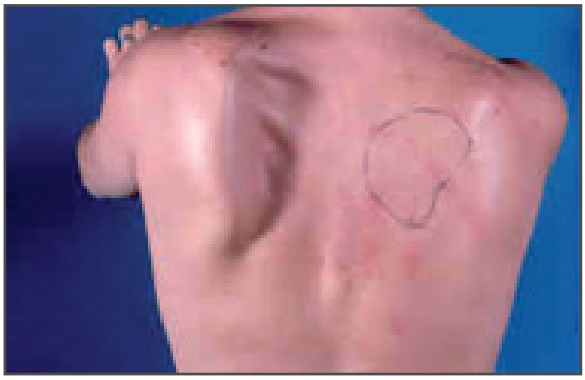
Diffential Diagnoses

Acute Immune Demyelinating polyneuropathy(AIDP) or Guillain-Barre' Syndrome (GBS)	Usually a subacute motor neuropathy with minimal sensory symptoms with little or no sensory findings. Weakness distal usually; cytoalbuminologic dissociation on CSF.
Porphyria	a motor ascending neuropathy; familial history is often present. Other symptoms persist.
Multiple sclerosis (MS)	has a waxing and waning history; vertigo, optic neuritis, focal sensory or motor symptoms; frequent urinary symptoms. Onset is highest in young females. Heat and stress can exacerbate symptoms. Diagnosis by MRI of the brain and spinal cord. CSF; EPs;
Spinal cord compression/myelopathy	can be caused by disc protrusion, epidural abscess, hemorrhage or cord trauma. MRI essential; CSF sometimes indicated. Risks (MS; Chiari malformation;) then acute new symptoms could be caused by other reasons.
Inner ear barotrauma	caused by inadequate equalization of middle ear pressure may cause rupture of round or oval windows; sudden tinnitus sudden vertigo, and may have unilateral deafness.
Facial baroparesis	is due to facial nerve compression as a result of decompression of the middle ear cavity during a dive or in aircraft flight. Usually transient; Facial weakness is lower motor neuron on the ipsilateral side.[11]
Focal peripheral neuropathy	requires knowledge of the anatomy of the peripheral nerves to rule out etiology. Carpal tunnel syndrome, median nerve neuropathy, is common and is not due to DCS but may be more pronounced post dive. Meralgia paresthetica, lateral femoral nerve neuropathy, is found in divers but is not DCS.
Brachial plexopathy  Brachial plexopathy (Parsonage-Turner) post dive	Brachial plexopathy post dive can be related but not DCS. Peroneal neuropathy, mental nerve neuropathy, notalgia paresthetica, gonyalgia paresthetica, ulnar neuropathy at the elbow, radial nerve palsy all have been found post dive. Horner's syndrome can occur from dissection post dive but is not DCS.

Glossary

Gonyalgia paresthetica neuropathy of the prepatellar branch of the saphenous nerve occurring secondary to medial meniscectomy.

Notalgia paresthetica Notalgia paresthetica (NP) is a sensory neuropathic syndrome of the midback skin, classically described as the unilateral infrascapular area. It is primarily a localized pruritus and dysesthesia syndrome, and it may present with episodic itching or pain on a small patch of the mid back, usually an area of skin just past easy reach.



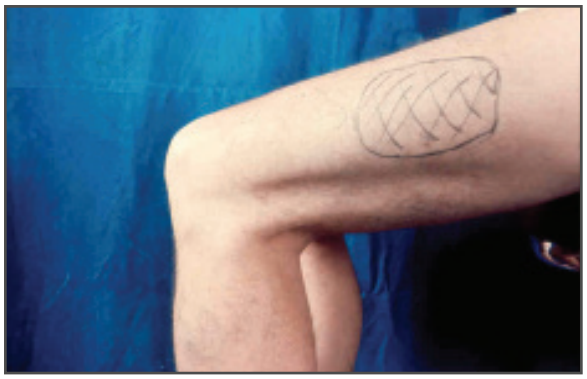
Notalgia parestetica and brachial plexus post dive

Upper motor neuron lesion or pyramidal lesion, occurs in the neural pathway above the anterior horn of the spinal cord. Such lesions can arise as a result of stroke, multiple sclerosis, spinal cord injury, DCI or other acquired brain injury.

Graphesthesia the ability to recognize the writing on the skin purely by the sensation of touch. Graphesthesia tests combined cortical sensation; therefore, it is necessary that primary sensation be intact bilaterally.

Visuospatial neglect common consequence of unilateral brain injury. It is most often associated with stroke and is more severe and persistent following right hemisphere damage, with reported frequencies in the acute stage of up to 80%. Neglect is primarily a disorder of attention whereby patients characteristically fail to orientate, to report or to respond to stimuli located on the contralateral side. Neglect is usually caused by large strokes in the middle cerebral artery territory and is heterogeneous, such that most patients do not manifest every feature of the syndrome.

Meralgia paresthetica numbness, tingling, and sometimes pain in the outer (lateral) thigh. It happens when the lateral femoral cutaneous nerve becomes compressed by tight suit, pregnancy, surgery, obesity, and is often idiopathic.



Meralgia paresthetica noted post dive

Photos

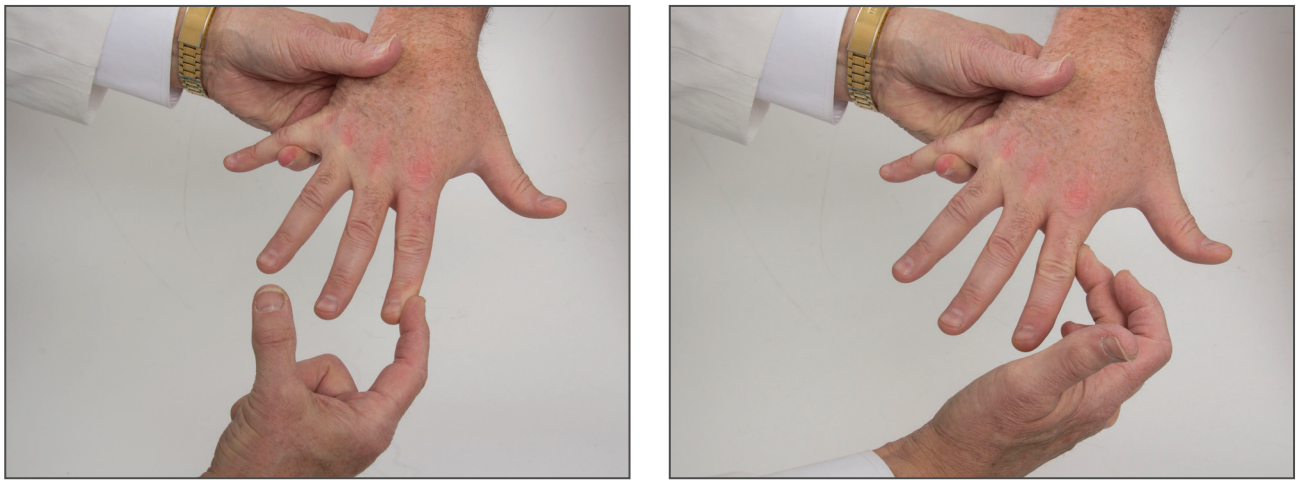


Figure 1. The right photo is the preferred exam

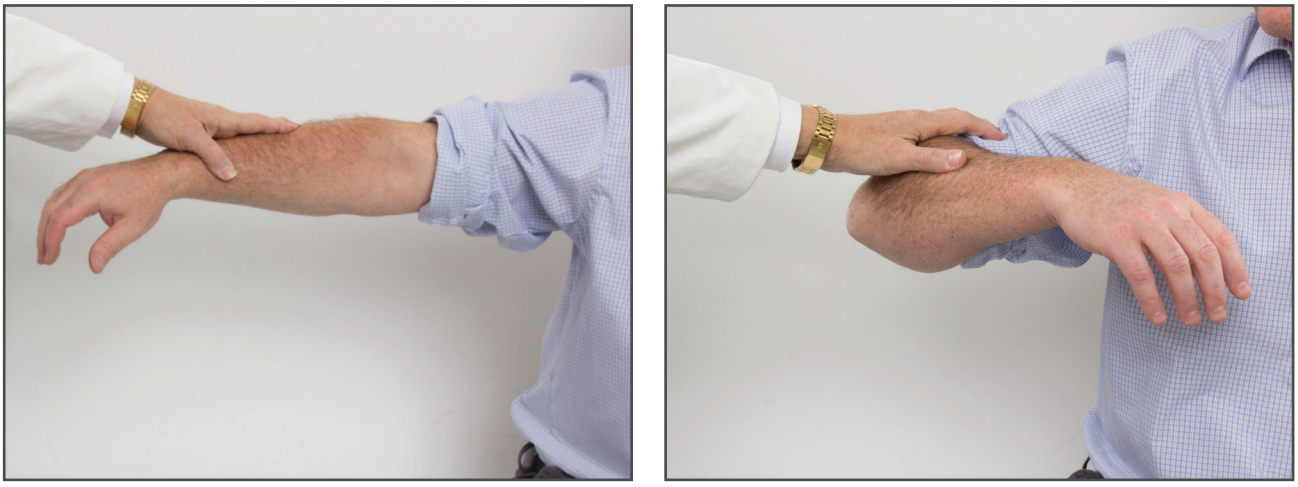


Figure 2. The right photo is the preferred exam

Figure 3. The right photo is the preferred exam

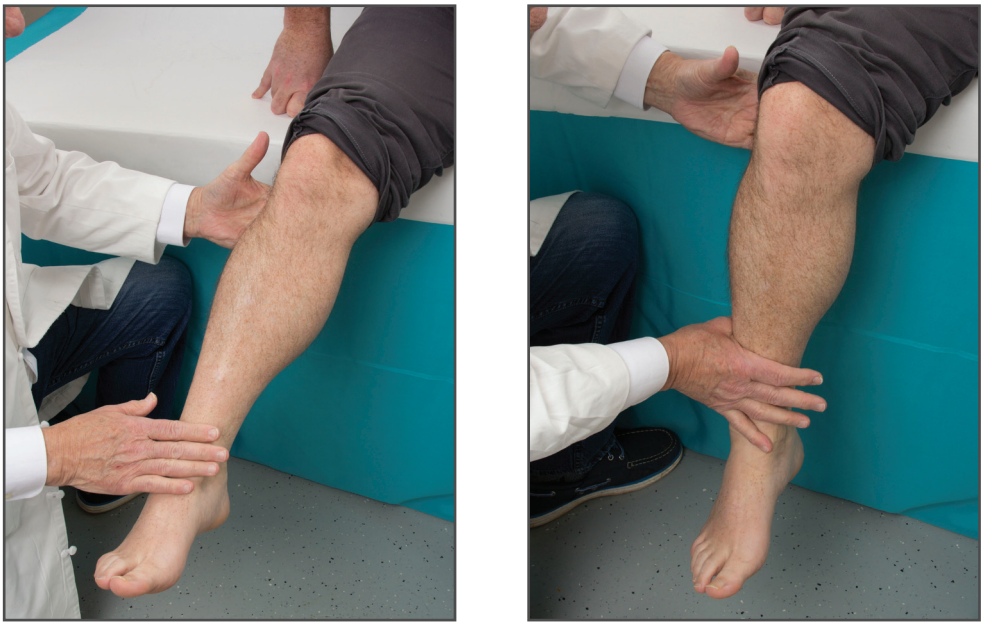


Figure 4. The right photo is the preferred exam

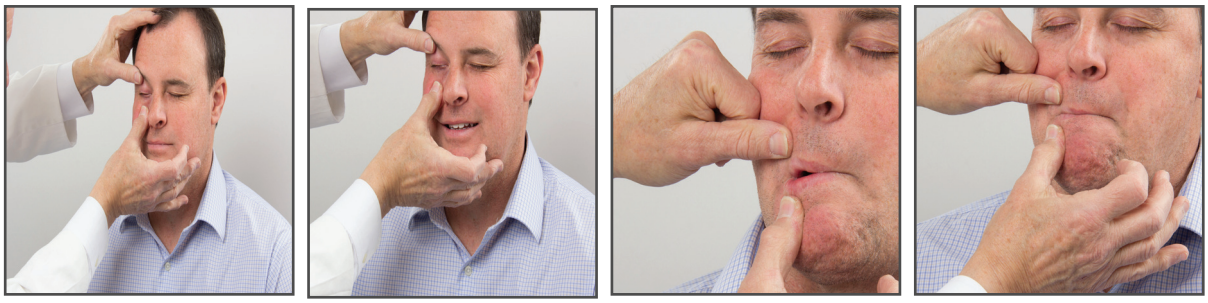


Figure 5. Facial testing

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Differential diagnosis of DCI - Challenges interpreting acute post-dive symptoms

Matias Nochetto, MD

Divers Alert Network started as the National Diving Accident Network (NDAN) in 1980 to establish a 24-hour emergency hotline with access to physicians trained in diving medicine, and to assist with evacuation to hyperbaric medical centers when indicated. Since then, DAN has answered more than 100,000 calls on the Emergency Line alone as a humanitarian service to all divers anywhere in the world and completely independent from any DAN membership benefits.

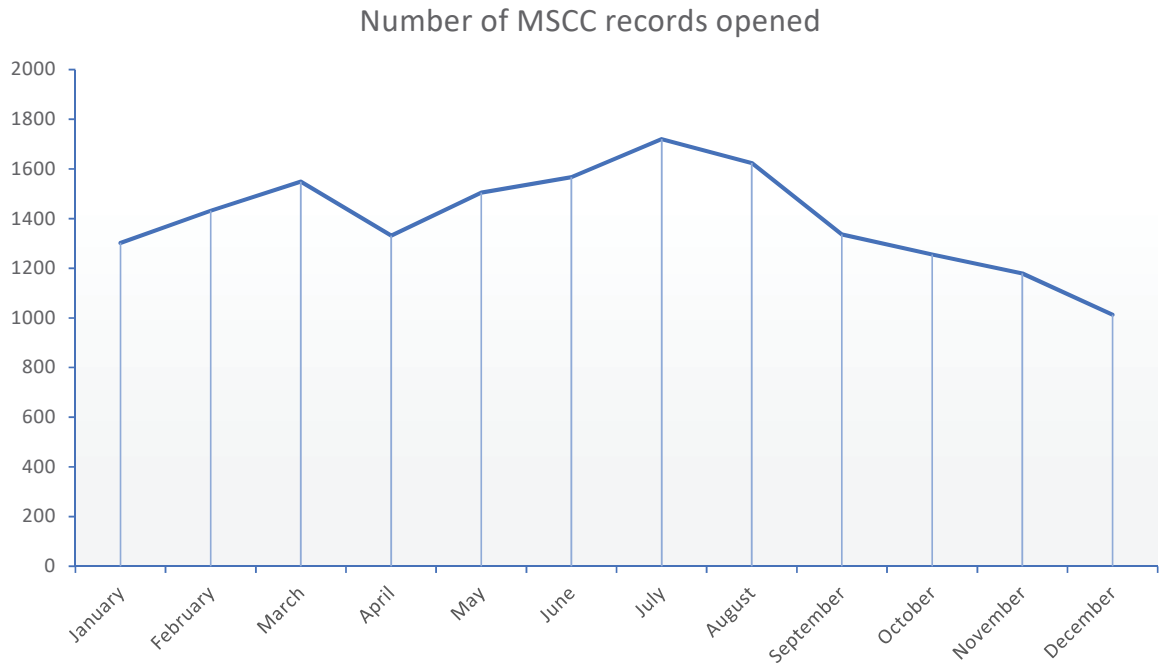

Figure 1. Average number of MSCC records opened by month (2016-2018) by DAN medical staff on MSCC (DAN's proprietary call center software). This pattern possibly represents the diving activity of the divers that call DAN medical services.

These days, DAN's Emergency Hotline staff receives around 3,500 calls per year, where about 1,500 of those calls pertain a diver experiencing symptoms following a dive. These calls are typically originated from lay people (the injured diver himself, a fellow diver or dive leader, or a family member), or from healthcare personnel seeking for expert dive medicine consultation when examining an injured diver who is already in a medical facility (ER physicians, nurses, paramedics, or chamber staff).

Limitations and challenges of telecommunication

Divers Alert Network is available for consultation on diving emergencies, on fitness to dive and diving physiology inquiries, assisting physicians all over the world make the best decisions for candidates looking for medical clearance to dive, or when examining or treating an injured diver with a suspected case of decompression illness (DCI). Additionally, DAN medical staff helps coordinate Emergency Medical Evacuations for DAN Members.

Compressed gas diving poses inherent risks. A reputable and well-planned dive operation (chartered, or private) should have clear and well defined standard operating procedures (SOPs) to help mitigate these risks. When incidents or injuries happen, a thoughtful and properly established Emergency Action Plan (EAP) should help the dive operation (chartered, or private) manage the situation effectively and efficiently. DAN's 35+ years of experience have shown us that EAPs are often suboptimal, so through its different Mission initiatives, DAN exists to assist injured divers, and their team members perform the best possible case management while in the field to ensure the best possible outcomes.

To provide the best possible recommendations in case of diving injuries, understanding the nature, possible extent and consequences of the injury become crucial. The best agents to triage these calls are medical professionals with training and experience in diving medicine. DAN's medical staff then consists of medics, DMTs, nurses, and doctors, whose only clinical tool is what the diver verbally conveys to them.

It is impossible -and it would be utterly imprudent- to try to establish a physician-patient relationship between DAN's medical staff and a diver calling the hotline. Managing the diver's expectations as to what DAN can do over the phone can sometimes be a challenge, and this limitation often needs to be made explicit.
An injured person becomes a patient when he/she is under the care of medical personnel. In the field, professional medical assistance starts with the local Emergency Medical Services (EMS). DAN does not admit patients to medical facilities, provide direct or indirect patient care, medical evaluation, diagnostic processes and treatment decisions. There are no "DAN patients" because DAN has no patients.

When the caller is a layperson in the field, DAN's medical staff will ask the caller basic questions to try to determine what might be going on (nature of symptoms, symptom onset, dive history, etc.) for then recommending what could be the best course of action for the perceived situation considering the particular geographical location where the diver might be. DAN's ultimate goal is to persuade the caller to make sure the injured diver seeks professional medical evaluation at the closest medical facility. Once the injured diver became a patient at the local healthcare system (either when EMS showed up at the scene, or once the injured diver has been admitted at the closest medical facility), DAN's medical staff makes themselves available to the assist the non-expert medical professional make the best possible decisions for their patient.

While DAN strives to provide injured divers and those caring for them with the best possible assistance through its Emergency Line, a telephonic communication imposes a rigorous limitation: the only contact DAN has with the caller (injured diver, team member, or examining healthcare professional) is verbal telecommunication. DAN accepts all incoming calls from members and non-members from anywhere in the world; languages can sometimes be a challenge. Some of these calls originate on remote locations, and the technical quality of these telecommunications is often unpredictable; which contributes to the overall challenge of remotely assisting someone in need.

Table 1. Stages of DAN's assessment, final categorization after case completion and diagnostic challenge

Diver's Chief complaint	DAN's Initial Assessment	TMD Diagnosis	DAN's Final Dx Category	Dx challenge?	Notes
"Shoulder pain. DCS?"	Mild DCS	Mild DCS	Mild DCS	No	Dx was correctly identified since chief complaint. There were no discrepancies or challenges.
"Shoulder pain. DCS?"	Cardiac Mild DCS Musculoskeletal	Cardiac	Cardiac	No	Although there were confounders during the initial assessment, the final diagnosis was identified early on, and there were no discrepancies.
"Tingling, DCS?"	Mild DCS Neuropraxia Marine life sting	Allergic reaction	Marine life sting	Yes	Although there were confounders during the initial assessment, there were discrepancies with the TMD Diagnosis.
"Tingling, DCS?"	Neuropraxia Marine life sting	Mild DCS	Anxiety	Yes	There were confounders and discrepancies with the TMD Diagnosis. Final Dx Category was not considered as a possible explanation for the case.

Post-hoc case analysis

Divers Alert Network keeps records of all calls on the Emergency Line through proprietary systems (DAN's MSCC) designed to collect information that helps the department provide good customer service and proper case management. Additionally, these records feed a database of incidents and injuries that can contribute to general knowledge through epidemiological research.

A typical call usually evolves following fairly predictable steps. The diagnostic procedure, on the other hand, may not necessarily be straight forward.

An injured/ill person has a "chief complaint"; a concise statement describing the symptom, problem, condition, perceived diagnosis, or other reason that justifies the call to DAN's Emergency Line. The job of the hotline agent (medically trained staff) is to perform basic questions to determine what might be going on. By the time the hotline agent closes the first interaction with a caller, the agent has the minimum information to formulate a reasonable -yet cautious- "initial assessment"; and be fairly confident in providing sound recommendations based on the answers to those questions.

A given case can take different turns and evolve in ways that one could not have foreseen upon the initial contact. The hotline agent can only interpret what the layperson conveys to him/her, but a thorough medical examination might reveal a different scenario than what the initial assessment provided. This only reinforces why DAN's hotline agents will always try to persuade the caller with an acute health issue to seek for professional medical evaluation.

Once at the medical facility, the injured diver -now a patient- will sooner or later have a diagnosis and a treatment plan. At this stage, DAN's role is no longer Case Management but Case Monitoring; seeking out frequent updates from the team of treating physicians, and offering them expert dive-medicine consultation if deemed necessary. The "Treating MD Diagnosis" is made, and this information is recorded in DAN's MSCC.

Table 2. Breakdown of cases analyzed by Initial Assessment and number of differential diagnoses also considered.

Initial Assessment	n	Different final DX
Ear & sinus BT	562	12
DCS Type II *	78	30
Cutaneous DCS	56	3
Pulmonary BT *	5	9
DCS Type I *	49	19
Non-diving related	27	4
Pulmonary Edema - IPE *	15	4
Mask squeeze	12	0
Barotrauma (other)	7	0
AGE *	3	2

*Identified as a large number of Different final DX

Depending on the nature of the case, the Case Monitoring phase could extend from a few hours to weeks; and the working diagnosis and treatment plan prescribed by the treating physicians may vary accordingly. Once the patient has been discharged, DAN may continue with a scheduled long-term follow-up with the diver. At this point, a case is deemed Closed.

A Closed case is subjected to Quality Assurance and Quality Control. This requires a close analysis of the case, looking at case documentation and audio recordings. Based on this post-hoc analysis made by DAN's senior medical and research teams, a case is assigned a "Final Dx Category". This one may or may not be consistent with the Chief Complaint, the Initial Assessment, or even the Treating MD diagnosis (TMD Diagnosis).

For the purpose of this presentation, we reviewed 814 cases with complete follow-ups. And asked on the Initial Assessment, we counted how many differential diagnoses were considered.

Findings

Ear & Sinus Barotraumas: This is by far the most common diving injury. We identified 562 cases where we a post hoc analysis concluded there was a high degree of confidence the final diagnosis was indeed an ear or sinus barotrauma.

Capital signs and symptoms of uncomplicated otolaryngological barotraumas included ear pain, sharp pain in the forehead/behind the eyes/on the occipital region, pain in upper molars, all usually associated with vertical travel. Muffled hearing, dizziness, and headaches as common post-dive symptoms. Vertigo and/or hearing loss are deemed serious complications of ear barotraumas.

Despite a large number of cases, there were only 12 other possible differential diagnoses identified. Under most circumstances, this was the result of a careful and defensive initial assessment on behalf of the hotline agent, where he/she documented other more serious categories to reflect what was conveyed to the caller during the telecommunication as possible differential diagnoses or complications.

Table 3. Key cues considered by DAN's hotline staff when assessing the likelihood of Cutaneous DCS.

Characteristic	Likely DCS	Less likely DCS	Unlikely DCS
Manifestations	Mottling, cyanotic	Rash	Raised or bumpy rash, vesicles, folliculitis
Localization	Areas of bodily fat	Under swimsuit, possible friction	Face, neck, forearms, lower legs, ankles and feet
Timing	Early post-dive (hours)	> 6 hours post-dive	> 12 hours post-dive
Exposure	Significant to moderate	Moderate to mild	Mild to negligible
Associated symptoms	Soreness on deep palpation, headaches, neuro sx, joint pains, SOB, dry cough	Allergy symptoms	Discoloration lasting days, and/or changing colors (bruise)
Past Medical History	Previous cases of skin bends, significant weight loss*	Similar symptoms due to allergy	

*Author's observation; unclear whether or not there is an association, but seems to be a question worth asking.

Cutaneous DCS: We identified 56 cases with confirmed cutaneous DCS, and only three additional diagnoses were considered: bruising (trauma), allergic reaction, marine life stings. Most of these cases had multiple categories checked due to the presence of other symptoms. Overall, this is usually a clear and straightforward presentation. It is not uncommon for these symptoms to be unnoticed or underestimated; such is the case of divers that call with a chief complaint of joint pains or paresthesias, and skin manifestations are only disclosed when the agent asked for any visible skin discoloration or rash.

Arterial Gas Embolism: Three cases of AGE were identified, and two additional diagnoses were considered. Many recreational divers are older and may have plethora of co-morbidities. As a result, cerebrovascular accidents (aka CVA, stroke) are a frequent differential diagnosis. Second most common differential dx considered was Inner Ear DCS (IEDCS).

The presentation usually consists of a reported acute focal neurological deficit. It is important to notice that although the mechanism of injury involves lung-overexpansion, a history of a rapid ascent to surface while holding their breath is not always present. Other possible mechanisms might include air trapping phenomena, including bronchospasms. Respiratory symptoms are not always present, and a fair number of cases of AGE seem to show no evidence of PBT. With regards to decompression stress, we consider it to have a prognostic value; cases with low decompression stress (short and/or shallow exposures) seems to have better outcomes and require fewer treatments. Paradoxical embolisms (PFO, or intrapulmonary shunts) might also debut with an acute focal neurological deficit, suggestive of an AGE.

Pulmonary Barotraumas (PBT): Cases of PBT often have multiple differential diagnoses. Five cases with final diagnosis of PBT, had initially nine differentials. This is usually the result of multiple categories being checked, and a defensive documentation approach due to the high morbimortality of these cases. Differentials included pulmonary edema, immersion pulmonary edema (IPE), drowning, cardiac, anxiety, and cardiopulmonary DCS being the most relevant.

Most cases seemed to present with fairly classic respiratory symptoms. It is not uncommon to have a history of a forceful underwater activity like retrieving an anchor, search and rescue. A dry cough seems to be a

fairly often associated with PBT, either as a mechanism of injury due to breath-holding while coughing or as a consequence of extra-alveolar air causing phrenic nerve irritation.

Negative X-rays might not rule out subtle forms of extra-alveolar air. Caution is advised before prescribing recompression therapy to a diver, included but not limited to:

- Difficulty breathing/discomfort
- Focal neurological deficit
- History of rapid ascent as a potential culprit for chief complaint
- History of a forceful underwater activity (i.e., retrieving or losing an anchor)
- History of underwater coughing
- History of COPD

Immersion Pulmonary Edema (IPE): IPE seems to have a fairly classic presentation, evolution, and findings. The chief complaint is almost always shortness of breath while still underwater or in the water. This particularity of symptom onset before a decompression insult is worth emphasizing, for it could be a key element to differentiate it from cardiopulmonary decompression stress. The shortness of breath is usually not fatal but is often incapacitating and frightening enough to make the diver abort the dive. Divers usually report "gurgling sounds," "rattling chest," and "pink sputum." Gradual spontaneous recovery once out of water is relatively classic (minutes to a few hours). With regards to cardiac enzymes, professional medical evaluation reports are inconsistent. Assessment of decompression stress seems to be of little value unless the exposure was significant enough to consider cardiopulmonary DCS.

Among the differential diagnoses considered, cardiogenic pulmonary edema (PE), pulmonary barotrauma (PBT), cardiopulmonary DCS (Type 3 DCS) are by far the most common. The IPE is sometimes misdiagnosed as the water aspiration; alleging a "malfunctioning regulator", a "teared diaphragm" or a "lose mouthpiece" as a rationalization of the fluid found in the lungs but without any supporting evidence. While water aspiration and drowning is one of the main causes of death in divers, it is hard to defend the idea that a conscious person with normal airway reflexes would not realize he/she is aspirating water.

Pain Only DCS (DCS Type 1): Among the initial assessments, musculoskeletal injuries, anxiety and cardiac (with shoulder pain) are by far the most differential diagnoses commonly considered. Highlights for DAN Medical are:

- Commonly affected joints:
  - Shoulders (+++), elbows (+++), hips (+)
  - Knees, ankles, wrists and small joints are almost always caused by something else.
  - Lower back pain is highly suggestive of spinal DCS, particularly when there is significant decompression stress or insult.
- Pain is dull, persistent, deep.
- Pain worsens remains static or remitting.
- Intermittency is highly suggestive of other causes.
- Pain is usually unaltered by movement of the affected joint.

Marginal DCS ("the niggles"): Cutaneous sensory changes are a common manifestation of DCS. While the Goldings classification described any neurological manifestations as a "Type 2 hit", there is currently a more holistic approach to classifying DCS and patchy cutaneous sensory changes not following a dermatomal distribution are considered a mild form of DCS, and the classic "Type 2" nomenclature is often reserved as a wide classification for more severe neurological manifestations.

Among the initial assessments often considered for cases which chief complaint evolves around "numbness," "tingling," or "pins and needles," Mild DCS is usually followed by anxiety, and musculoskeletal with neuropraxia ("pinched nerve"). Some dive gear configurations can restrict blood flow on upper extremities (BCDs, harnesses, dry suits), and some diving environments can also introduce other confounders that could provoke paresthesias, like partial exposure to extreme cold temperatures (gloves with poor thermal insulation on cold-water environments) or marine life

stings on tropical marine environments where the diver admitted grabbing from rocks, corals, wrecks or permanent mooring lines with bare hands.

Unspecialized professional medical evaluation often misinterprets these findings as possibly resulting from a neurodegenerative process, a stroke, or totally dismissed them unless there are dramatic and objective findings on the neurological examination.

Summary

For DAN Medical Services DCI is a clinical diagnosis based on three key elements: subjective complaints of diver, reported objective manifestations, and a thorough medical history, all carefully interpreted taking into consideration the limitations initially described;.

There are six tenets of "diagnosis" for DAN:

1. Chief complaint
   - Often subjective and imprecise. Must be clarified through thorough thoughtful interrogation and cautiously interpreted.
2. Time of symptom onset
   - The sooner the symptom onset, the worse the prognosis for DCI
   - The longer the delay, the less likelihood of DCS; and/or the less serious the case
3. Dive exposure
   - Often unreliable (unless dive computer data is available)
   - More valuable on the extremes to either seriously consider DCI or to rule it out altogether
4. Evolution of symptoms
   - Symptoms of DCS are usually static, remitting, or progressing; but hardly ever intermittent.
5. Neurological status
   - Not always available, complete, or reliable. An objective evaluation from a physician is always encouraged, regardless of any diving medicine training.
6. Source of information
   - Layperson vs healthcare professional

Considerations:

- Diagnosing DCI without a thorough medical history is a guess.
- A gross field neurological assessment may provide relevant clues to establish diagnosis and severity, although it may miss mild cases.
- A thorough physical examination helps support the working diagnosis and serial physicals help to gauge the progress during and between treatments.
- It is easy to connect the dots backward.
- The prescription of recompression therapy does not confirm a diagnosis.
- Many ailments will improve with hyperbaric oxygen therapy, as well as with a sympathetic white coat.

Diagnostic Algorithms for Decompression Sickness and Arterial Gas Embolism

Ian Grover, MD, FACEP

The symptoms of decompression sickness were first described in humans in 1841 by a French mining engineer named Triger. With the advent of SCUBA diving in the 20th century, and its dramatic increase in popularity over the last quarter of a century, diving injuries have become well known but infrequent medical disorders. It seems strange that after over 150 years of experience with decompression sickness (DCS) and over 70 years of similar experience with arterial gas embolism (AGE) we still have yet to establish an accepted case definition used by scientists and medical professionals. There are multiple reasons that the diving community needs case definitions and diagnostic algorithms for the diagnosis of decompression sickness and arterial gas embolism. These include participation in research studies, entry into databases, treatment decisions, and for providing feedback to the patient, their family, their employer, and the insurance companies. There are numerous algorithms or scoring systems that have been developed for DCS and AGE. The problem is that these systems divide patients into prognostic groups or disease severity, and they don't address the diagnosis itself or recovery from the illness.

The following are a few of the diagnostic algorithms and scoring systems that are present in the literature. One of the first scoring systems derived was published by Dick and Massey in 1985.[1] This system used a severity score that was the sum of a sensory symptom grade and a motor symptom grade. This score could be easily calculated and it indicated the severity of DCS. The researchers also looked at outcomes based on the score. In a paper published in 1993, Ball used the system Dick and Massey developed to look at spinal cord DCS. He classified these cases according to a numerical severity index and time to recompression with oxygen.[2] Ball felt that this system could help with prognosis. In 1996, Dutka proposed a modification to the popular classification of DCS and AGE that was published in Francis and Smith's article in 1991. The modified system classified DCS and AGE according to the time of onset of symptoms, how the disease progresses, and the organ system affected. It helps divide patients into narrower groups which may help with prognosis.[3]

Kelleher in 1996 developed a model to predict outcome after the first treatment.[4] This was not a scoring system but an algorithm that looked at the types and combinations of deficits as well as the specific sites involved. Boussuges published his work in 1996 and this scoring system took into account repetitive diving, clinical course before treatment, and neurological findings.[5] This score has prognostic validity (It was validated by its authors and an independent group in 1999; Pitkin et al). The problem with Boussuges score is that it does not follow outcomes. Another problem was

that it could not be applied to patients that do not have objective neurologic findings.

Mitchell et al. developed the RNZN score (1998) and it is a very comprehensive severity score that takes in to account the subjective and objective signs and symptoms associated with DCS.[6] These are used to develop a severity score. This system can be quite bulky to use, but it can be applied to all DCS and AGE patients. It has also been shown (Holley 2000) to have good predictive value for sequelae after treatment. Another benefit is that is helps to assess recovery of patients.

In the 1940's medicine was faced with the problem of diagnosing acute rheumatic fever (ARF). No sensitive or specific test existed for this illness. A series of "major" and "minor" criteria were established (Jones Criteria) to make this diagnosis.[7] For research purposes, these criteria were highly specific allowing the generation of case series that, for practical purposes, included only true cases of ARF. Conversely, interpreting these criteria more loosely helped practitioners make clinical diagnoses for treatment purposes or as an aid to advising patients.

Diving medicine is not unique in the situation of being forced to make clinical diagnoses in the absence of a defining "test" to establish the diagnosis. There are two basic reasons for attempting to define criteria to establish the diagnosis of the decompression disorders and these are to: (1) reach a treat-no-treat decision, and (2) include cases in a database for either clinical or research purposes. In both cases some sort of criteria or "case definition" for the diagnosis of decompression sickness and arterial gas embolism is necessary. Using the Jones Criteria as a model, the criteria for the diagnosis of decompression sickness and arterial gas embolism will be presented.

Before developing the criteria, a few assumptions must be made in regards to decompression sickness. Assumption #1 - There exists a minimum exposure, less than which a diagnosis of acute DCS should not be made. Otherwise, you couldn't ride in an elevator without being at risk of DCS. Assumption #2 - For any given sign or symptom(s), the greater the gas loading, the greater the likelihood that the symptom(s) are manifestations of decompression sickness. Assumption #3 - For any given sign or symptom(s), the sooner to the exposure that the symptom(s) develop, the greater the likelihood that the symptom(s) are manifestations of decompression sickness. Finally, the criteria must have a catchy name. Thus, the SANDHOG criteria were developed. The term SANDHOG was derived from the acronym for SAN Diego Diving and Hyperbaric OrGanizations for the group led by Dr. Tom Neuman, that helped to develop the criteria, and it is a slang term used for caisson workers.

Rather than using "major" and "minor" criteria like the Jones criteria, a point scale for making these diagnoses will be presented. A point scale is advantageous because it can be made more specific by raising the score needed to establish the diagnosis (for research purposes), but it can be made more sensitive by lowering the necessary score for a diagnosis (for clinical purposes). With the SANDHOG criteria, three points are needed for the diagnosis of DCS or AGE. This will keep the criteria specific enough for an uncorrupted database without unduly sacrificing sensitivity.

The scoring system has been developed for the SANDHOG criteria using data from numerous sources of diving injuries. First, there must be an exposure capable of producing DCS. Furthermore, there must be a reliable way to estimate the exposure. Not all exposures can cause DCS. In order to enter this algorithm, there must be an exposure at least equal to the compartment loading achieved by a dive to 50% of the United States Navy (USN) no decompression limits. The value of 50% of the no decompression limits was selected for two reasons. Although it is a minimal exposure, occasionally severe cases of spinal cord DCS have been seen with such exposures (especially when associated with AGE's). Secondly, analysis of North Sea Diving experiences suggests this will capture the vast majority of cases. Any symptoms associated with exposures less than this are much more likely to be due to something other than DCS. See Figure 1.[8]

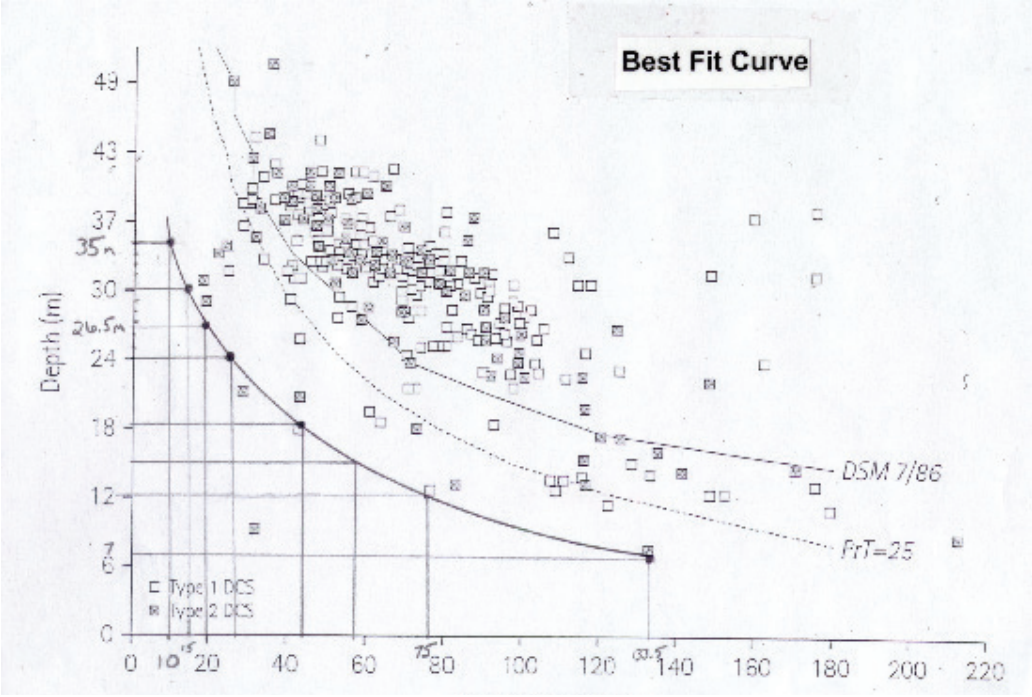

Figure 1. Depths and times at which DCS occurs in commercial offshore air-diving operation in the UK sector. Shields TG and Lee WB. "The incidence of Decompression Sickness Arising From Commercial Offshore Air-Diving Operations in the UK sector of the North Sea During 1982-1990."

Three Points are awarded for each of the following:

1. Signs and symptoms of a transverse myelitis with both sensory and motor changes (weakness rated as 3/5 or worse; not just isolated sensory changes) within 2 hours of a dive. The motor exam is scored on the 5 point scale where 0 is no movement at all, 1 is a muscle twitch but no actual movement, 2 is movement, but not strong enough to overcome gravity, 3 is movement strong enough to overcome gravity but not any other resistance, 4 is movement strong enough to overcome gravity and some resistance, but not normal, and 5 is full strength. The reasoning for this is that a transverse myelitis with motor changes is an unusual diagnosis for things other than DCS. Such findings are likely to be highly specific for DCS. Based on review of type II DCS cases, two hours was selected because the overwhelming majority of neurological DCS occurs within 2 hours of a dive. When such symptoms begin further out from a dive the specificity for DCS decreases. See Figure 2.[9]
2. A monoparesis worse than or equal to 3/5 with pathological reflexes and associated sensory changes (not isolated sensory changes) within 2 hours of a dive. The reasoning behind this is the same as # 1 above.
3. Cutis Marmorata, not an erythematous rash, but true marbling of the skin. Linear streaking is not considered cutis marmorata. Although cutis marmorata can rarely be seen in other conditions it is generally only associated with shock like states, except in DCS. Therefore, if it occurs after a dive, the finding is specific enough to be diagnostic of DCS.

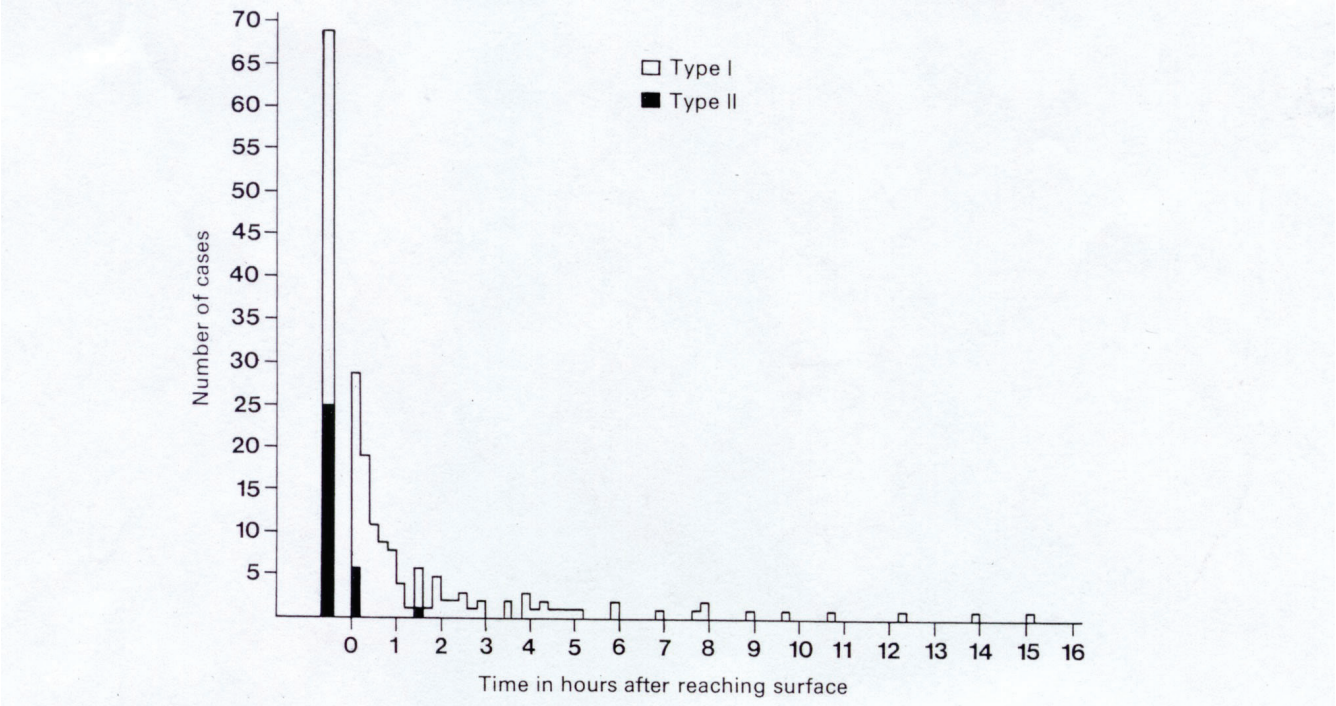

Figure 2. Decompression sickness-time of onset of cases in relation to the time of reaching surface. 196 cases from the Canadian Forces Institute of Aviation Medicine and Royal Canadian Navy Diving Establishments, July 1963 to October 1968. The cases, which occurred during decompression, are shown together before the time of surfacing.

Two Points are awarded for each of the following:

1. An exposure (without decompression) that is greater than the loading seen with exceeding the Navy no stop limits by 10% (i.e. a 60 foot dive for 66 minutes, a 70 foot dive for 55 minutes, an 80 foot dive for 44 minutes etc.) or missed decompression greater than 5 minutes. These would be highly provocative profiles and "soft" signs or symptoms after such a dive must be considered much more seriously than after a trivial exposure.
2. Any sign or symptom in the three point category occurring 2-6 hours after a diving exposure.
3. Chokes, which is defined as the syndrome of cough, substernal chest pain and shortness of breath, is worth two points. This syndrome was only assigned two points because of possible confusion with immersion pulmonary edema, aspiration etc. Chokes are most frequently associated with a heavy load of venous gas embolism (VGE) and therefore will be associated with a heavy gas load as in #1 above or #6 under the 1 point category.
4. The syndrome of inner ear (vestibular) DCS characterized by vertigo, tinnitus and hearing loss that lasts more than 5 minutes accompanied by an abnormality of tandem gait or an abnormal Romberg (not sharpened Romberg), occurring within 2 hours after a dive. This was not given three points because people who are simply "dizzy" should not enter the database, as this is too subjective a symptom. Otic barotrauma can be easily confused with this syndrome so it was assigned only two points. Once again most true vestibular DCS is associated with significant and/or provocative exposures.
5. Deep boring pain in a major joint within 2 hours of surfacing from a dive. Too many alternative diagnoses can cause pain in the joints. Therefore, this syndrome was only assigned 2 points. Again, based on extensive review of DCS cases, two hours was selected because two-thirds of all DCS occurs within this time period. The further from the dive, the less likely the symptoms are DCS related. See Table 1.10, [11]

Table 1. Kelley, Berghage, and Summit. NEDU Research Report 10-68. 1968.

CUMULATIVE PERCENTAGE OF CASES HAVING ONSET OF SYMPTOMS BEFORE GIVEN TIME AFTER SURFACING FROM A DIVE				
	Year	1997	1961-66	Rivera (1963)
During Dive		11%	12%	9.1%
	20	41%	40%	
	40	56%	50%	
1 Hr.	60	59%	56%	54.7%
	80	59%	60%	
	100	67%	61%	
2 Hr.	120	69%	65%	66.8%
	140	74%	66%	
	160	79%	68%	
3 Hr.	180	82%	71%	
4 Hr.	240	85%	78%	
5 Hr.	300	87%	84%	
6 Hr.	360	90%	90%	86.2%
	400	95%	93%	
	400	(5%) 100%	(7%) 100%	

6. Isolated sensory changes in a single limb or at a spinal cord level plus hyperreflexia within two hours of surfacing from a dive. There is always concern about isolated subjective (symptoms) complaints. The presence of hyperreflexia makes the likelihood that a true pathophysiologic abnormality exists greater and therefore the sensory changes are more likely to represent DCS. That it occurs within two hours of surfacing does the same thing. Isolated paresthesias without any other findings are not highly specific enough to be routinely considered DCS.
7. Lymphedema occurring within 24 hours of a dive. This is quite specific for DCS. However, one must make sure to differentiate this from hives and swelling due to trauma, stings etc.

One Point is awarded for each of the following:

1. Deep boring pain in a major joint from 2-6 hours after surfacing from a dive. The reason being, as more time elapses after a dive a non-specific symptom becomes increasingly less likely to represent DCS. Therefore, as one gets further from the dive the same symptoms must be worth fewer points.
2. Isolated sensory changes in a single limb or at a spinal cord level plus hyperreflexia 2-6 hours after surfacing from a dive. The reasoning behind this is the same as #1 above.
3. Complete relief from joint pain within 10 minutes of the initiation of recompression therapy. Many things improve in a chamber over a 6-hour period. The placebo effect is both real and considerable. True "pain only" bends usually responds quickly to recompression. Although cases may respond slowly, too many of those will be corrupted by false positives.
4. Complete relief of motor and sensory changes within 40 minutes of therapeutic recompression, or a full number improvement in motor signs during

the first 2 hours of recompression, i.e. a change from 3/5 to 4/5, or from 2/5 to 3/5. Again, the reasoning behind this is the same as #3 above.

5. Scintillating scotomata occurring after a dive in a patient without a prior history of migraine headaches. This symptom is too subjective and too non-specific to be weighted heavily. When it occurs without a significant exposure or without any other signs or symptoms, there should be concern with the diagnosis of DCS.
6. A dive profile (without decompression stops) between the "no stop" limits of USN '55 and VVAL 18 or a properly conducted single dive requiring staged decompression. These are still rather benign exposures with a very low incidence of DCS. Symptoms following such a dive might represent DCS, however these profiles cannot be considered very provocative.[12]

Half Point is awarded for each of the following:

1. Isolated paresthesias or "tinglies" occurring after a dive. Many people have minor non-progressive, highly subjective symptoms that are very non-specific. If isolated "tinglies" are considered DCS the database will be significantly corrupted.
2. Fatigue, dizziness, headache, nausea, or vomiting. Only half a point can be awarded for any combination of these symptoms. The reasoning for this is the same as #1 above. However, if these symptoms are associated with "harder" signs or symptoms they will be considered in the diagnosis.

Minus One Point for each of the following:

1. Presence of fever
2. History of hypochondriasis or anxiety disorder

AGE Criteria

A 3-point scale was devised for AGE also. The entrance criteria to this algorithm are any exposure using a compressed air source along with a neurologic deficit that occurs within 5 minutes of surfacing from the dive.

Three Points are awarded for the following:

Sudden loss of consciousness, disorientation (i.e. time or place), aphasia, or a hemiplegia within 5 minutes of surfacing from a dive using a source of compressed air.

Two Points are awarded for the following:

1. Hemiparesis or monoparesis, within 5 minutes of surfacing from a dive. The reason these were only awarded two points is that these may be more subjective.
2. Cortical blindness or seizures within 5 minutes of surfacing from a dive, as long as there is no history of epilepsy.

One Point is awarded for each of the following:

1. A rapid uncontrolled or any ascent associated with panic and the onset of symptoms.
2. Hemoptysis
3. Presence of barotrauma on CXR
4. CPK greater than 2 times normal in the absence of musculoskeletal trauma

These criteria were then validated against the Duke hyperbaric database of diving related injuries. The Duke hyperbaric database was started in 1997, and it is a record of all patients evaluated at the Duke Hyperbaric Center. This database consists of 1,919 records. It contains the physician evaluation, treatment records, follow-up evaluations, and insurance and billing information for all the patients.

This database was queried for all patients evaluated for a decompression illness in the period since its inception (1997) to the termination of data collection (March, 2003). A total of 124 records had sufficient data and were available for review. Patients were included only if critical data (initial evaluation and at least one post treatment evaluation) were present. For this analysis, all patients with a hypobaric exposure (excluding medical evacuation) were excluded. Dive time was not recorded in the database; therefore all cases with a maximum depth of 40 fsw were excluded. After exclusions, there were only 4 cases of AGE that survived to discharge. Given this, all the cases of AGE were excluded from the

analysis due to insufficient statistical power. A total of 86 cases were then available for application of the SANDHOG criteria.

Data on demographics, symptoms, treatment, and response to treatment were collected. The last data point collected was the "doubt" field. A case was determined to be doubtful if the treating physician at the time of initial evaluation felt that the diagnosis of DCS was unclear. The patient's response to recompression was not included in the determination of doubt.

The SANDHOG criteria were applied on a post hoc basis, and as such some of the fields did not map directly. The data on exposure were not of sufficient quality to be useful, so exposure points were initially not included. All other criteria were able to be mapped using abstracted fields or combinations of abstracted fields.

Using the SANDHOG score as presented and the "doubt" field as a standard, the SANDHOG criteria were 52.7% sensitive, 90.3% specific, and had a positive predictive value of 90.63%. ROC analysis of the original SANDHOG criteria gave an area under the curve of 0.72.

The SANDHOG criteria were developed to help clinicians identify cases of decompression sickness and arterial gas embolism accurately. The three point system would allow clinicians to diagnose these disorders with enough sensitivity and specificity to suggest a treat-no-treat decision, and it would help develop an uncorrupted database of DCS and AGE cases for research purposes. A major benefit of an uncorrupted database would be the ability to perform accurate research on these conditions. This may lead to adjunctive treatments for both of these conditions or alternative treatment regimens. The importance of this increases as underwater development and exploration continues and as more people take to the water to enjoy SCUBA diving.

Based on the application of the SANDHOG criteria against a database of diving related injuries, we found the criteria to have a high specificity and a very high positive predictive value. The area under the ROC curve (AUC) shows that the criteria are a useful test. The AUC tells us about the accuracy of the criteria. An area under the curve of .90-1.0 is an excellent test, 0.80-0.89 is a good test, 0.70-0.79 is a fair test, and 0.60-0.69 is a poor test.

The sensitivity of the criteria clearly should be improved, but we were comparing the criteria to a "doubt" field that was not a gold standard. Even outlying cases of decompression sickness were not doubtful unless the treating physician expressed doubt. Another complicating factor is that these criteria were applied retrospectively and no points were given for exposure because dive times were not recorded in the database. Further comparisons are required because the criteria have not been validated against cases of AGE. In this database, there were no clear cases of vestibular, pulmonary, or lymphatic decompression sickness. Also, the SANDHOG criteria are not applicable to hypobaric exposures, diving at higher elevations, and cases where flying after diving is involved.

The positive predictive value of the SANDHOG criteria is very high, and it suggests the SANDHOG criteria may be a useful tool for the diagnosis of DCS. A score based system represents a practical method to establish inclusion/exclusion criteria for trials and epidemiological studies. Further research is required to determine the validity of the SANDHOG criteria against cases of AGE and to apply the criteria in a prospective fashion.

The author would like to thank Dr. Tom Neuman for his mentoring and all of the work he did to develop the SANDHOG criteria.

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Diagnostic Approach for the Management of Diving Injuries - Experience of the Hyperbaric Center of Sainte-Anne Hospital, Toulon France

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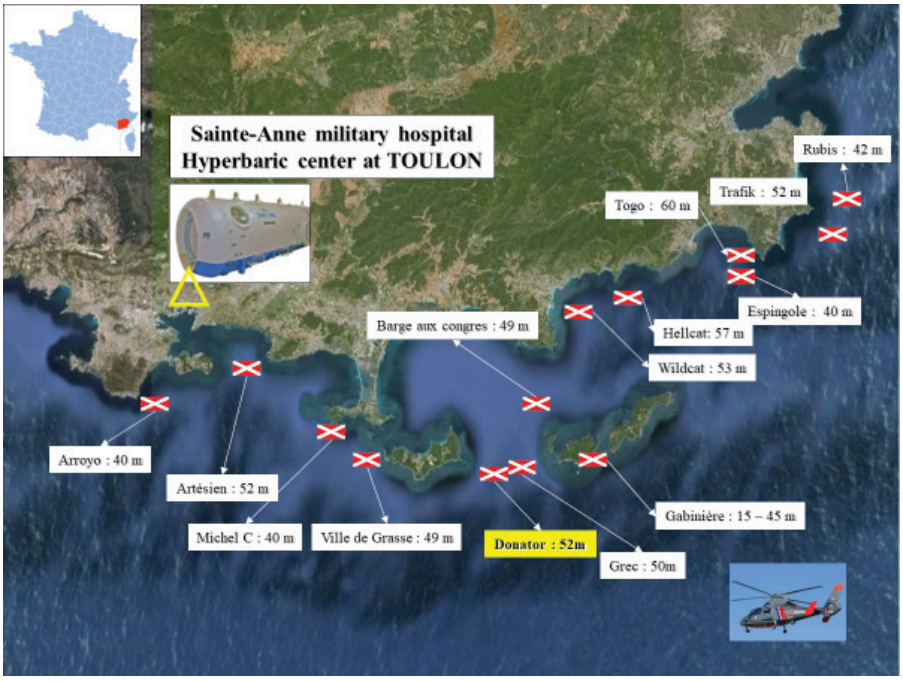


Figure 1. Location of Sainte-Anne military hospital, the hyperbaric center at Toulon, France.

The diagnostic approach for the management of diving injuries is primarily based on the identification of symptoms and their evolution after surfacing. Proper diagnosis is essential to determine what treatment is necessary and refer the patient to a medical institution that can provide it. We present in this document the main diagnostic principles and procedures in relation to the clinical presentation of diving injuries that occur on the Mediterranean coast in France.

The Mediterranean coast is very attractive for diving because of the presence of many wrecks, but unfortunately, many of these wrecks require diving up to 40- 50 msw, which is a source of a large number of diving accidents. Thus, the hyperbaric center of the Sainte-Anne hospital in Toulon is one of the centers that receive the highest number of diving accidents in Europe with 120 to 150 admissions per year.

Epidemiological data

Among admitted recreational diving injuries to Sainte-Anne military hospital, the most frequent diagnosis is decompression sickness (DCS, 56%), followed by immersion pulmonary edema (IPE, 15%) and barotrauma (9%). Serious pulmonary barotrauma is exceptional and accounts for 1 to 2% of all accidents. The barotrauma of ear and sinuses represents, in fact, the majority of injuries in recreational diving (80%) but they are rarely admitted to the hospital.

Spinal cord DCS is to be feared because it is the most frequent form of the DCS (41%), most serious, and results in sequelae in 20 to 30% cases at discharge after HBO treatment. Cochlear-vestibular DCS is also common (28% of DCS), while osteoarticular, skin and brain forms are rare.

The vast majority of decompression sickness occurs despite compliance with the standard procedure. Severe forms occur early after surfacing, sometimes with clinical signs appearing at the deco stop, or within minutes upon surfacing and leaving the water. Severe forms delayed more than six hours are rare. DCS classically occurs in an experienced diver, after a dive rather saturating (depth greater than 40 msw, and total dive time of about 40 minutes).

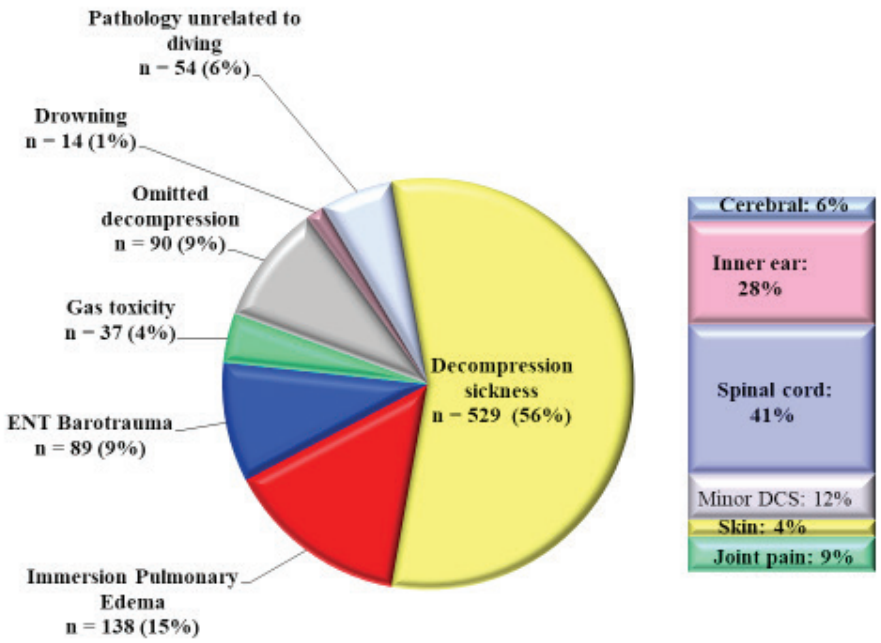


Figure 2. Diagnosis for 951 diving accident cases admitted to Sainte-Anne military hospital in the period 2010-2017

Table 1. Post-dive manifestations and the probability of DCS diagnosis

Clinical manifestations	Incidence in France	The probability of DCS diagnosis
Cardiac arrest Respiratory signs	Very Rare	Low
Joint pain Cutaneous signs	Uncommon	High
Vestibulocochlear signs Neurologic signs	Most common	High

Respiratory signs after surfacing

- Cardiopulmonary DCS is exceptionally rare
- More likely diagnoses: IPE, pulmonary barotrauma, drowning
- Treatment for these conditions is based on normobaric oxygen
- Recompression is only performed in case of neurological signs i.e. CAGE from pulmonary barotrauma
- Confirmation of the diagnosis is based on the chest CT scan

Respiratory signs after surfacing

The occurrence of respiratory symptoms following a scuba dive is a common situation that can progress to life-threatening cardio-respiratory distress or neurological signs requiring hyperbaric recompression. It requires administration of normobaric oxygen and evacuation to a hospital ideally equipped with a cardiac intensive care unit and a hyperbaric center.

The circumstances, the time of the onset and the nature of the symptoms most often make it possible to guide the diagnosis. Confirmation of the diagnosis is based on chest CT scan.

The cardio-respiratory distress is mainly caused by IPE which can occur at all levels of diving. The IPE is related to in-water physical exercise and negative pressure breathing. It can be observed during swimming on the surface, during apnea, during compressed air diving or diving with dorsal rebreathers.

Divers older than 50 years, with cardiovascular risk factors, especially with hypertension, are more at risk. Often mildly symptomatic, IPE can cause severe hypoxia and sometimes fatal myocardial dysfunction. The IPE is an underestimated cause of death in diving.

Pulmonary barotrauma, which is often observed in the context of emergency free ascent or breath-holding during ascent, is also responsible for deaths during diving, by compressive pneumothorax or cerebral

Table 2. Differential diagnosis of respiratory signs after

Post-Dive Respiratory Signs			
Exertion during diving		Loss of consciousness while diving	Emergency free ascent; breath-hold during ascent
Hypercapnia	**IPE**	**Drowning**	**Pulmonary barotrauma**
Signs disappear at rest; Residual headache	Multifactorial: negative pressure breathing, cold, stress, breath-hold diving	Gas toxicity (O_2, CO_2, N_2); Rebreathers; deep diving	Subcutaneous emphysema; Pneumothorax; AGE
Benign, except in case of loss of consciousness	Normobaric oxygen; Hospitalization	Normobaric oxygen; Hospitalization	Normobaric oxygen; Evacuate to hyperbaric center

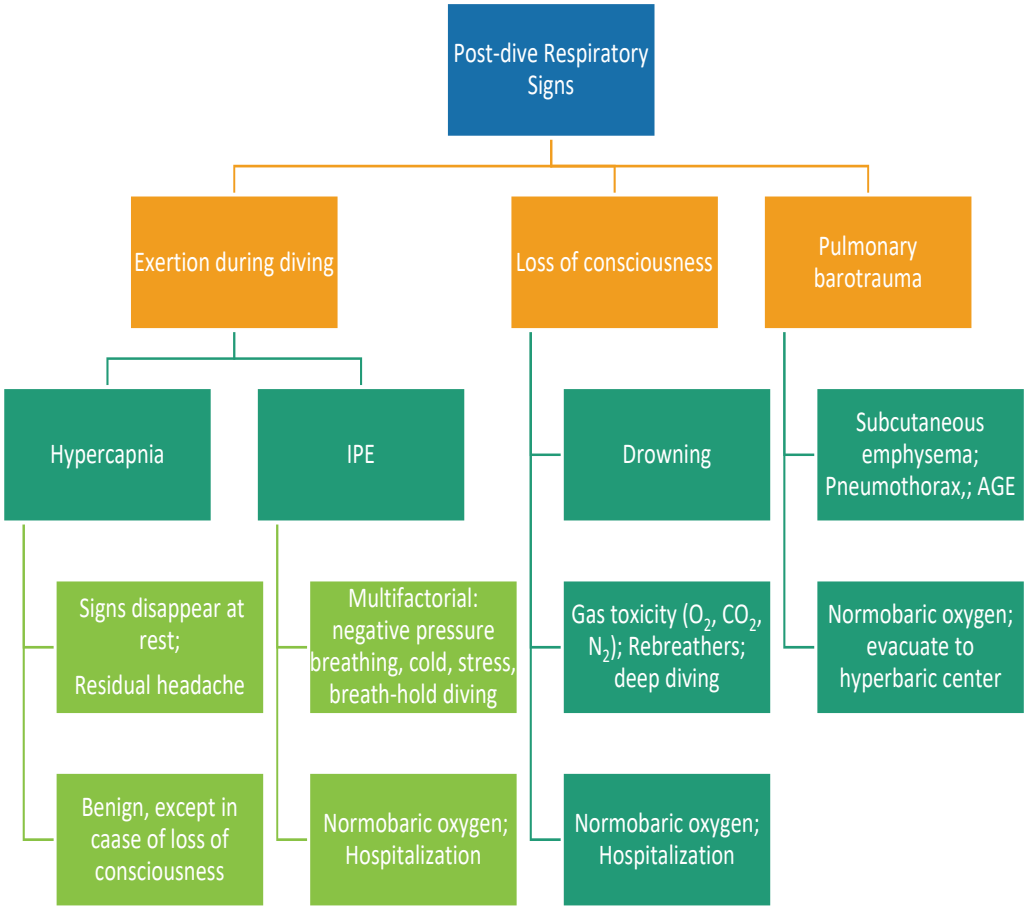


Figure 3. Post-dive respiratory signs

aeroembolism. Only pulmonary barotrauma complicated with cerebral gaseous embolism with neurological signs requires recompression in hyperbaric chambers.

Joint pain after surfacing

The presence of joint pain after surfacing especially at the shoulder pain is very suggestive of decompression sickness. In the absence of recompression, the intensity increases with time with irradiation of pain. There are two distinct forms depending on the location of the bubbles: the periarticular form, the most common (2/3 of cases), affecting muscle and tendon insertions, immediately improving with recompression and intraosseous form (1/3 of cases), often aggravated by recompression and which is likely to progress to dysbaric osteonecrosis. (PJD: In case of pain does not resolve with recompression) Intraosseous forms should be identified by performing an initial joint MRI and repeating this examination to follow the evolution. In these bone forms, HBOT sessions should be prolonged for several weeks to limit the risk of osteonecrosis.[1,2]

Cutaneous signs after surfacing

Clinical studies show that there is a strong link between cutaneous DCS and the presence of a right-to-left shunt (patent foramen ovale – PFO most often) which is observed in 80% of these cases (Gempp et coll 2017, Wilmshurst 2015). The pathophysiology of cutaneous DCS, therefore, seems to be related to embolization with circulating bubbles at the level of cutaneous

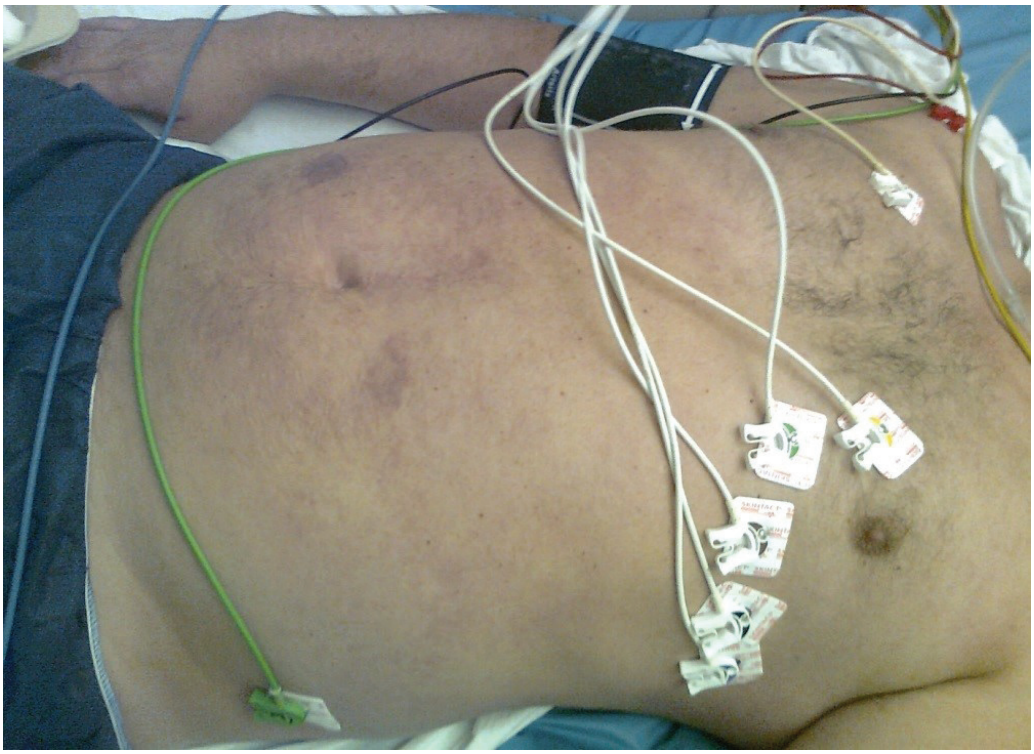


Figure 4. Diver suffering from cutaneous DCS i.e. cutis marmorata

arterioles causing livedo or cutis marmorata. Another discussed possibility is a brainstem aeroembolism that would disrupt the functioning of the autonomic nervous system at the origin of livedo ("syndrome dysautonomique"). [3,4]

Differential diagnoses to consider:

1. Vasomotor livedo in young women exposed to cold when the involvement is limited to the extremities or in the presence of acrocyanosis.[5]
2. Allergy
   - Cold, pressure or aquagenic urticaria is responsible for a pruriginous micropapular eruption that can last one to two hours after contact with water.
   - The neoprene suit is also a source of many sensitizations with thiourea derivatives or the resins used for the neoprene seams.

Vestibulocochlear signs after surfacing

Inner ear DCS are related to the presence of a right-to-left shunt (present in 80% of cases) with a mechanism of arterial embolization of the inner ear (preferentially on the right side). Vestibular signs are the most common (3/4 of cases). They occur shortly after surfacing and are dominated by intense rotary vertigo accompanied by nausea and vomiting.

The examination reveals spontaneous horizontal nystagmus, with a harmonious vestibular syndrome, signs of peripheral affliction. Hypoacusis and tinnitus may be associated with cochlear involvement.

The assessment includes the search for a right-to-left shunt, audiometry, the realization of a video-nystagmogram (VNG) and posturography to look for sequelae which are frequent despite the vestibular compensation.

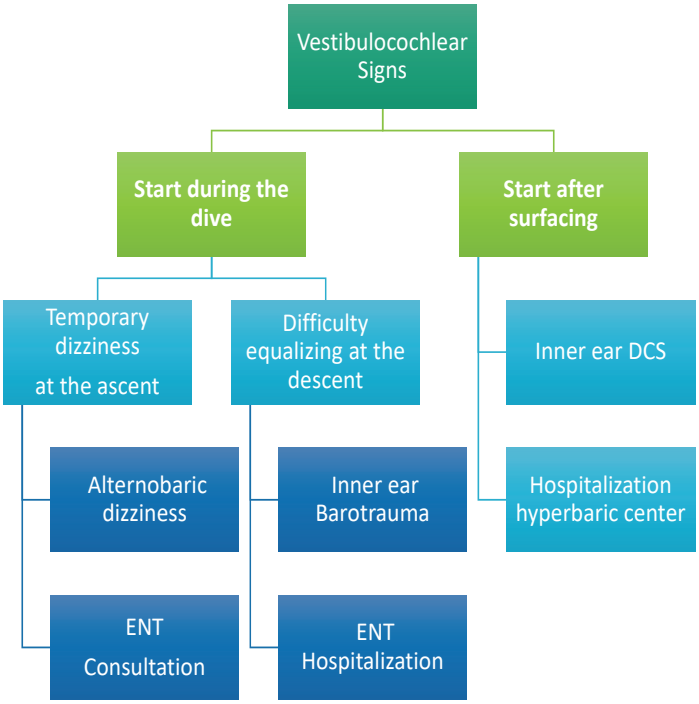


Figure 5. Vestibulocochlear signs

Distinguishing between Inner ear DCS and barotrauma is important:

- Inner ear DCS should be recompressed
- Inner ear barotrauma = no recompression because of the risk of perilymphatic fistula

FISTULA= fluctuation of vestibulo cochlear signs according to the position

- In doubt recompress - inner ear DCS is more frequent than fistula

The differential diagnosis often arises with inner ear barotrauma, but in this case, otalgia on the descent and cochlear signs are often described, with difficulties for ear equalizing. Severe inner ear barotrauma can lead to a perilymphatic fistula, which is a contraindication to hyperbaric recompression. The treatment of the fistula is surgical. It is the fluctuation of symptoms depending on the position of the head that suggests a fistula.

Positional audiometry should be performed at the slightest doubt.

Neurological signs about spinal cord DCS

Typical signs of spinal cord DCS are neurological manifestations in the limbs without the involvement of the cranial nerves. These may be objective signs with sensory or motor deficit, or purely subjective symptoms with paresthesia.

Possible causes of spinal cord DCS include: a) in situ bubbles that can damage the white medullary substance, b) venous blood stasis of epidural venous plexus due to an accumulation of gas bubbles, c) arterial embolization by venous gas bubbles passing through the right-left shunt a which is present in 50% of spinal cord DCS cases. The differential diagnosis in case of spinal cord DCS is not a problem. The problem is rather the diagnosis of its severity. A severity score should be used to guide the choice of medication and hyperbaric treatment (score Medsubhyp).

Spinal cord DCS:
- Most frequent form of DCS
- Progressive onset of the symptoms
- Presence of PFO: 50%
- Venous stasis
- Local compressive factors on spinal cord
- High sequelae risk: 20-30%

Cerebral DCS:
- Less frequent
- Early onset
- Presence of PFO: 80%
- Often favorable outcome with HBO
- Difficulties for differential diagnosis

The most serious accidents are usually accompanied by other symptoms such as vertebral pain of varying intensity, which can sometimes feel as stabbing. Motor deficits can settle insidiously, low noise, in a few hours, initially with feelings of heaviness or weakness of the limbs. Sphincter involvement with retention of urine is quite common and indicates prognosis. Serious spinal cord DCS usually worsen in 12 to 24 hours regardless of the hyperbaric treatment.

The neurological examination should seek, with the aid of the ASIA score, a motor deficit, hypoesthesia (testing all modes), pyramidal irritative syndrome and proprioceptive ataxia, which indicates lesions of the posterior tracts of the spinal cord. The spinal cord MRI, performed at least 48 hours later, shows, in severe forms, ischemic damage and sometimes the existence of anatomical compressive factors in relation to the spinal cord injury. Elevations in hematocrit and D-dimer and albumin decrease are also associated with severity.

Cerebral neurological signs after surfacing

Severe forms can lead to disturbances of consciousness, convulsions, hemiplegia, but most often there are focal lesions of the superior functions and cranial pairs, with for example visual disturbances (amaurosis, impaired visual field), speech disorders (aphasia, speech difficulties), psychic disorders (confusion, prostration, agitation).

In the first instance, the diagnosis of a cerebral decompression accident should be mentioned. Cerebral DCS is most often linked to the existence of a right-to-left shunt (80% of cases) with a mechanism of cerebral arterial aeroembolism. Depending on the context, and especially in the presence of an emergency ascent with expiratory blocking, one must mention the pulmonary barotrauma diagnosis which can also lead to cerebral aeroembolism following alveolo-capillary lesions. Performing an emergency chest CT scan makes it possible to diagnose and identify complications such as the presence of a pneumothorax that must be drained before hyperbaric management.

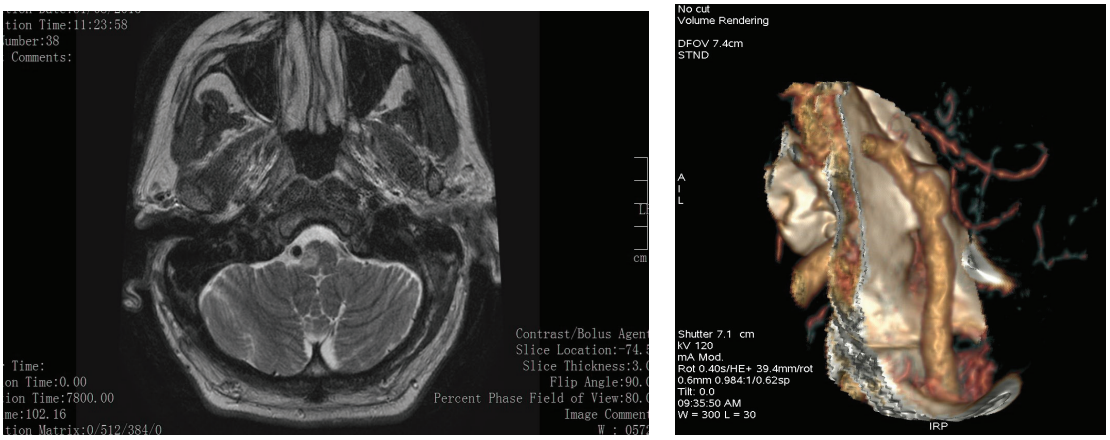
Neurological cerebral symptomatology may also correspond to ischemic stroke or hemorrhagic stroke. According to the anamnesis, it will be necessary to perform an emergency brain MRI to determine the diagnosis of a diving accident or stroke because the treatment is not the same. The stroke should NOT be recompressed but require referral to a stroke center as soon as possible for thrombectomy or thrombolysis.

In conclusion, whatever the nature of the symptoms, it is necessary to determine an etiological diagnosis and/or a differential diagnosis of a diving accident. The diagnosis is based primarily on the anamnesis and diving circumstances. But in certain situations, it may be necessary to carry out first-line complementary tests, especially in the presence of respiratory (chest CT) or cerebral (brain MRI) symptoms after surfacing.

Case report: neurological symptoms after diving: stroke or DCS?

An experienced, 53 years old male diver, carrying out a dive to 47 msw with 26 min of bottom time. No violations of deco procedure. He felt a neck pain during deco stop and paresthesia of the left side of

the body and the right face after surfacing. There was no improvement with normobaric oxygen. Neurologic examination at the hyperbaric center found a bilateral pyramidal syndrome with hypoesthesia of the left lower limb and permanent vertical nystagmus. The treatment with table Cx30 (4 ATA with Héliox 50%) and lidocaine IV 2mg/min did not bring any improvement. After the HBO, the condition of the patient was progressively worsening, and a diagnosis of Wallenberg syndrome was established. The manifestations included vertical nystagmus, an ataxic walking with polygon widening, a left thermoalgic anesthesia respecting the face and a right thermoalgic anesthesia of the face. No RLS/PFO with TCD and TEE was found. However, MRI (A) revealed an ischemic zone in the territory of the circumflex artery from the right vertebral artery and complementary 3D imaging (B) found an aneurysm of right vertebral artery and dissection of the right internal carotid responsible for the bulbar ischemic stroke.



A B

Figure 6. MRI with ischemic zone in the territory of the circumflex artery from the right vertebral artery (A); complementary 3D imaging with an aneurysm of right vertebral artery (B)

Glossary

Wallenberg's syndrome is a neurological condition caused by a stroke in the vertebral or posterior inferior cerebellar artery of the brain stem. Symptoms include difficulties with swallowing, hoarseness, dizziness, nausea and vomiting, rapid involuntary movements of the eyes (nystagmus), and problems with balance and gait coordination. Some individuals will experience a lack of pain and temperature sensation on only one side of the face, or a pattern of symptoms on opposite sides of the body – such as paralysis or numbness in the right side of the face, with weak or numb limbs on the left side. Uncontrollable hiccups may also occur, and some individuals will lose their sense of taste on one side of the tongue while preserving taste sensations on the other side. Some people with Wallenberg's syndrome report that the world seems to be tilted in an unsettling way, which makes it difficult to keep their balance when they walk. (https://www.ninds.nih.gov/Disorders/All-Disorders/Wallenbergs-Syndrome-Information-Page)

Thermoalgic anesthesia – a lack of pain and temperature sensation

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Texte original en français:

Démarche diagnostique pour la prise en charge des accidents de plongée. Expérience du centre hyperbare de l'hôpital Sainte-Anne à Toulon France.

La démarche diagnostique pour la prise en charge des accidents de plongée repose avant tout sur l'identification des symptômes et de leur évolutivité après la sortie de l'eau.

La démarche diagnostique est essentielle pour déterminer le traitement et l'orientation du patient vers une structure de soins appropriée.

Nous présentons dans ce document les principaux diagnostics retenus en fonction des symptômes observés pour les accidents de plongée qui surviennent sur la côte méditerranéenne en France.

La côte méditerranéenne est très attractive pour la plongée en raison de la présence de nombreuses épaves, mais malheureusement beaucoup d'entre elles nécessitent de réaliser des plongées entre 40 et 50 mètres de profondeur, ce qui est source d'un grand nombre d'accidents de plongée. Ainsi le centre hyperbare de l'HIA Ste Anne à Toulon est un des centres qui reçoit le plus d'accidents de plongée en Europe avec 120 à 150 admissions par an.

Données épidémiologiques

La répartition des accidents de plongée de loisirs montre que l'accident de décompression est le plus fréquent (56 %), suivi des oedèmes pulmonaires d'immersion (15 %) et des barotraumatismes (9 %). Les barotraumatismes pulmonaires graves sont exceptionnels et représentent 1 à 2 % des accidents. Les barotraumatismes de la sphère ORL (oreille et sinus) représentent, en réalité, la majorité des accidents (80 %) mais ils sont rarement admis en milieu hospitalier.

Les accidents de décompression neurologiques médullaires sont à redouter, car ils sont les plus fréquents (41% des ADD) et les plus graves avec 20 à 30% de séquelles, à l'issue de la prise en charge en centre hyperbare. Les ADD cochléo-vestibulaires sont également fréquents (28% des ADD), tandis que les formes articulaires, cutanées ou cérébrale sont plus rares.

La grande majorité des accidents de décompression survient malgré le respect de la procédure. Les formes sévères surviennent précocement après l'émersion, avec parfois des signes cliniques apparaissant dès les paliers, ou en surface dans les minutes qui suivent la sortie de l'eau. Les formes retardées après six heures sont plus rares. L'ADD se produit classiquement chez un plongeur expérimenté, au décours d'une plongée plutôt saturante (profondeur supérieure à 40 mètres, durée totale de plongée de l'ordre de 40 minutes).

Signes respiratoires à la sortie de l'eau

L'apparition d'une symptomatologie respiratoire au décours d'une plongée sous-marine est une situation fréquente qui peut évoluer vers une détresse cardiorespiratoire mettant en jeu le pronostic vital ou s'accompagner de signes neurologiques nécessitant une recompression hyperbare. Elle impose une mise sous oxygène normobare systématique et une évacuation vers une structure hospitalière idéalement équipée d'une unité de soins intensifs cardiologiques et d'un centre hyperbare.

Les circonstances, le moment de survenue et la nature des symptômes permettent le plus souvent d'orienter le diagnostic. La confirmation du diagnostic repose sur la réalisation d'un scanner thoracique en urgence.
Les pathologies respiratoires sont dominées par les OPI qui s'observent pour tous les niveaux de plongée. Ces OPI sont liés à la réalisation d'un effort physique en immersion et d'une ventilation à pression négative. Ils peuvent s'observer lors de natations en surface, lors d'apnée, lors de plongée à l'air comprimé ou de plongées en recycleurs à port dorsal.

Le plongeur de plus de 50 ans, avec des facteurs de risque cardio-vasculaire, en particulier avec une hypertension, est davantage concerné. Souvent pauci symptomatiques, ces OPI peuvent provoquer une hypoxie sévère et une dysfonction myocardique parfois mortelles. Ils représentent une cause importante et sous-estimée de décès en plongée.

Le barotraumatisme pulmonaire qui s'observe souvent dans le contexte de la formation à la remontée d'urgence, est également responsable de décès en plongée, par pneumothorax compressif ou aéroembolisme cérébral. Seuls les barotraumatismes pulmonaires compliqués d'embolie gazeuse cérébrale avec présence neurologiques nécessitent une recompression en caisson hyperbare.

Douleurs articulaires à la sortie de l'eau

La présence d'une douleur articulaire apparaissant après la plongée en particulier au niveau d'une épaule est très évocatrice d'un accident de décompression. En l'absence de recompression, l'intensité augmente avec le temps avec une irradiation de la douleur. Il existe deux formes distinctes selon la localisation des bulles : l'atteinte péri-articulaire, la plus fréquente (2/3 des cas), touchant les insertions musculaires et tendineuses, d'évolution immédiatement favorable à la recompression et l'atteinte osseuse (1/3 des cas), non calmée voire aggravée par la recompression et qui est susceptible d'évoluer vers une ostéonécrose dysbarique. Les formes osseuses doivent être identifiées en réalisant une IRM articulaire initiale en répétant cet examen pour évaluer. Dans ces formes osseuses les séances d'OHB doivent être prolongées sur plusieurs semaines pour limiter le risque d'ostéonécrose.

Signes cutanés à la sortie de l'eau

Les études cliniques montrent qu'il existe un lien fort entre les accidents cutanés et la présence d'un shunt droite-gauche (foramen ovale perméable - FOP le plus souvent) qui est observé dans 80% de ces cas d'ADD cutané (Gempp et coll 2017) (Wilmshurst 2015). La physiopathologie des ADD cutanés semble donc être liée à un mécanisme d'embolisation artérielle de bulles circulantes au niveau cutané responsable de livedo ou cutis marmorata par occlusion artériolaire. Il est également évoqué la possibilité d'un aéroembolisme du tronc cérébral qui perturberait le fonctionnement du système nerveux autonome à l'origine d'un livedo par un « syndrome dysautonomique » (Kemper et coll 2015) (Germonpre et coll 2015).

Il faut notamment évoquer des diagnostics différentiels : livedo vasomoteur chez la femme jeune exposée au froid lorsque l'atteinte se limite aux extrémités ou en présence d'acrocyanose (Gibbs et coll 2005). Les causes allergiques doivent être aussi recherchées. Il faut aussi évoquer l'urticaire au froid, à la pression ou encore l'urticaire aquagénique responsable d'une éruption micropapuleuse pruriginineuse qui peut durer une à deux heures après le contact de l'eau. Le port de la combinaison néoprène est également source de nombreuses sensibilisations avec les dérivés thiourées ou encore les résines utilisées pour les jointures du néoprène.

Signes cochléo-vestibulaires à la sortie de l'eau

Les accidents de désaturation cochléo-vestibulaires sont liés à la présence d'un shunt droite-gauche (présent dans 80% des cas) avec un mécanisme d'embolisation artérielle de l'oreille interne (préférentiellement du côté droit), L'atteinte purement vestibulaire est la plus fréquente (3/4 des cas) : peu de temps après l'émersion, survient un vertige rotatoire intense, accompagné de nausées et vomissements.

L'examen retrouve un nystagmus horizontal spontané, avec un syndrome vestibulaire harmonieux témoignant d'une atteinte de type périphérique. Hypoacousie et acouphènes peuvent être associés en cas d'atteinte cochléaire.

Le bilan comporte la recherche d'un shunt droite-gauche, une audiométrie, la réalisation d'un vidéo-nystagmo-gramme (VNG) et d'une posturographie pour rechercher des séquelles souvent fréquentes malgré la compensation vestibulaire.

Le diagnostic différentiel se pose souvent avec un barotraumatisme de l'oreille interne, mais dans ce cas, une otalgie à la descente et des signes cochléaires sont souvent décrits, avec des difficultés d'équilibration. Un barotraumatisme de l'oreille interne sévère peut entrainer une fistule périlymphatique dont le diagnostic et contrindique la recompression hyperbare. Le traitement de la fistule est chirurgical. C'est la fluctuation des symptômes en fonction de la position de la tête qui suggère une fistule. Une audiométrie positionnelle doit être réalisée au moindre doute.

Signes neurologiques d'origine médullaire à la sortie de l'eau

On observe des symptômes neurologiques au niveau des membres sans atteinte des paires crâniennes. Il peut s'agir de signes objectifs avec déficit sensitif ou moteur, ou de symptômes purement subjectifs avec des paresthésies isolées.

Le mécanisme des accidents de décompression médullaires est celui d'une ischémie localisée de la moelle épinière dont l'origine est multiple. Des bulles in situ peuvent léser la substance blanche médullaire. Il a également été mis en évidence un phénomène de stase sanguine liée aux bulles au niveau des plexus veineux drainant la moelle épinière. Le passage de bulles artérielles via un shunt droite-gauche semble également être contributeur (50% de shunts retrouvés).

Les accidents de décompression médullaires ne posent pas un problème de diagnostic différentiel mais plutôt un problème de diagnostic de gravité. Il existe pour cela un score de gravité qui permet d'orienter la prise en charge (score de Medsubhyp).

Les accidents les plus graves sont généralement accompagnés par d'autres symptômes comme une douleur vertébrale d'intensité variable, pouvant aller jusqu'à une sensation de « coup de poignard ». Les déficits moteurs peuvent s'installer de manière insidieuse, à bas bruit, en quelques heures, avec initialement des sensations de lourdeur ou de faiblesse des membres. On observe assez souvent une atteinte sphinctérienne avec rétention d'urine, de mauvais pronostic. Les ADD médullaires graves s'aggravent habituellement dans 12 à 24 heures quel que soit la prise en charge thérapeutique. L'examen neurologique recherchera, en s'aidant du score ASIA, un déficit moteur focalisé, une hypoesthésie (avec des sensibilités parfois dissociées), un syndrome irritatif pyramidal et une ataxie proprioceptive, signant une atteinte fréquente des cordons postérieurs de la moelle. L'IRM médullaire, réalisée au minimum 48h après, permet de mettre en évidence, dans les formes graves, une atteinte ischémique et parfois l'existence de facteurs compressifs anatomiques en regard de la lésion médullaire. Les élévations de l'hématocrite et des D-Dimères et la diminution de l'albumine sont également associées à la sévérité.

Signes neurologiques cérébraux à la sortie de l'eau

Les formes sévères peuvent entraîner des troubles de conscience, des convulsions, une hémiplégie, mais on observe le plus souvent des atteintes focalisées des fonctions supérieures et des paires crâniennes, avec par exemple des troubles visuels (amaurose, altération du champ visuel), des troubles de la parole (aphasie, difficultés d'élocution), des troubles psychiques (confusion, prostration, agitation).

On évoque en première intention un accident de décompression cérébral qui est le plus souvent lié à l'existence d'un shunt droite-gauche (80% des cas) avec un mécanisme d'aéroembolisme cérébral. En fonction du contexte, et notamment en présence d'une remontée d'urgence avec blocage expiratoire, on doit évoquer le diagnostic barotraumatisme pulmonaire qui peut aussi entraîner un aéroembolisme cérébral à la suite de lésions alvéolo-capillaires. La réalisation d'un scanner thoracique en urgence permet de faire le diagnostic et d'identifier des complications comme la présence d'un pneumothorax qu'il faudra drainer avant la prise en charge hyperbare.
La symptomatologie neurologique cérébrale peut aussi correspondre à un AVC ischémique ou hémorragiques. En fonction de l'anamnèse, il faudra réaliser une IRM cérébrale en urgence, afin de déterminer le diagnostic d'accident de plongée ou d'AVC car le traitement n'est pas le même. La prise en charge d'un AVC ne relève pas d'une recompression hyperbare mais nécessite l'orientation vers un centre spécialisé le plus rapidement possible pour une thrombectomie ou une thrombolyse.

En conclusion, quel que soit la nature des symptômes, il est nécessaire d'établir un diagnostic étiologique et/ou un diagnostic différentiel d'accident de plongée. Le diagnostic repose avant tout sur l'interrogatoire et les circonstances de la plongée. Mais dans certaines situations, il peut s'avérer indispensable de réaliser en première intention des examens complémentaires notamment en cas de présence de signes respiratoires (scanner thoracique) ou cérébraux (IRM cérébrale) à la sortie de l'eau.

Cardiorespiratory Post-dive Conditions

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The objectives of this discussion are to be able to better formulate a differential diagnosis for cardiorespiratory complaints following a diving or hyperbaric exposure, review diagnostic and some management considerations in order to evaluate and rule out diving related versus other medical conditions.

A web-based polling software was utilized throughout the presentation to facilitate discussion obtain participant opinion regarding particular questions and differential diagnosis considerations.

Many of the case-based discussions was surrounded variation on a fictional dive profile conducted by a 51-year-old female with no history of DCS. The dive profile was as follows: 130 feet for 49 meters, followed by 1:15 hr/min surface interval, and another 63 feet dive for 60 meters. The dive times were reported as "total dive time" which was discussed as it may not represent "actual bottom time" as many divers do not spend the majority of their time at the maximum dive depth. It is assumed (unless specifically discussed) that there were no ascent rate violations, and the patient did not exceed the no decompression limits for either of these dives.

The first case was elaborated by the diver telling the boat captain she is feeling tired and short of breath with mild chest tightness. This case was used to start discussion over differential diagnosis but did not give a definitive answer to the etiology of this diver's symptoms. A broad differential diagnosis was formulated including but not limited to: Acute coronary syndrome/MI, Pulmonary embolism, Cardiac arrhythmia, Aortic dissection, Aspiration, COPD, Asthma, CHF, Pneumonia, Pneumothorax, Pulmonary barotrauma, Decompression Sickness, Immersion Pulmonary Edema, Viral infection, Obesity hypoventilation, Exercise intolerance, Hypo/hyperglycemia, Dehydration, Electrolyte disorder, Anemia, Urinary tract infection, Foodborne illness, Toxin, Parasitic infection, Anxiety and Malignancy. Most poll respondents felt ACS/MI was most likely, with a few choosing decompression sickness (DCS) or arterial gas embolism (AGE).

It was determined that additional information would be helpful in establishing a diagnosis. This includes:
Medical History: Past medical problems, medications, recent travel, sick contacts, previous diagnostic testing, surgical history, smoking history, drug use, and pertinent past family history.

Recent non-diving related activities such as exercise, environmental exposure, climbing, flying.
Diving history: diving experience, previous diving injury such as DCI, AGE, previous diving physical exams.
Additional details about the dive: actual bottom time, actual profile, gas mixture, gas consumption, type of diving, exertion level, water temperature, computer

alarms/violations, computer settings, concerning or unusual events during the dive.
Physical examination

The second case discussion was the same diver and profile as above, but this time complains of chest tightness and shortness of breath (SOB) following the series of dives. Additional information gathered included this was an experienced diver, symptoms started shortly after being caught in a current and relays working harder than normal to follow the dive group. She was breathing air, had no computer violations. The chest discomfort was substernal, pressure-like, non-radiating, was associated with SOB and lightheadedness and the patient was mildly diaphoretic and pale on exam.

The group collectively recommended further medical assessment. We discussed transport considerations that varied depending on the situation. Serial electrocardiograms (ECG) were presented which the audience correctly identified as an ST Elevation Myocardial Infarction and appropriately chose to activate the cardiac catheterization lab. Acute coronary syndrome was discussed in addition to other differential diagnosis of cardiac related chest pain including valvopathy, myocarditis and pericarditis.

The third case followed the same 51-year-old diver with the same series of dives. This time, the patient had a history of atrial fibrillation (A-Fib) and was recently taken off rivaroxaban after ablation surgery and transition to aspirin alone. This patient is on metoprolol as well, but mentions she neglected to take her morning dose. She presented after diving with sudden onset palpitations and racing heartbeat which initially went away, then returned and is now persistent. An ECG was presented to the audience which was determined to be atrial fibrillation. Additional history was obtained and the patient relays this sensation is common when she goes into atrial fibrillation. We discussed this in the case of acute management, risk stratification and the ability to dive in the future. All of this is very situational depending on multiple factors, recommendations and interventions from cardiology. The concerns with future diving is exercise tolerance and the need for anticoagulant medication. I do not generally recommend diving while on an anticoagulant due to trauma risk (ie: falling and striking head on a dive boat), but given the current lack of consensus, this can be discussed on an individual basis with recreational divers.

The fourth, but similar case discussed was a 51-year-old female with chest tightness and shortness of breath after the dive. Other past medical history includes hypertension, intermittent palpitations/racing heartbeat over the past 20 years which she has never had evaluated. She developed palpitations and tachycardia for 2 minutes, about 1 hour prior to a single dive to 70 feet for 43 minutes. She had no symptoms during the dive but had recurrent palpitations after the dive. Her ECG was felt to be a narrow complex tachycardia, likely supraventricular tachycardia (SVT) vs. atrial flutter. A second ECG was shown for comparison which the audience identified as ventricular tachycardia.

The fifth case was about a 45-year-old female with chest tightness and shortness of breath after a single dive to 60 feet. This caused her to abort the dive after 15 minutes when she began coughing into her regulator. She had not been diving in a few years and does have a history of well controlled exercise induced asthma. She made a normal ascent to the surface and had a residual dry cough with shortness of breath on the dive boat. Most of the audience identified this as a likely acute asthma exacerbation. Cold water, increased gas density, saline misting from the regulator and exercise are all commonly found with SCUBA diving and may exacerbate or precipitate an asthma attack. Exacerbations can be managed with a combination of bronchodilators, possibly steroids, magnesium, BiPap, epinephrine, observation, and intubation if needed. Chest xray is sometimes used to rule out other diagnosis such as pneumothorax which may be warranted given her diving history, however, with her normal ascent to the surface, and symptoms starting underwater, this diagnosis would be less likely.

The sixth case discussed was a similar middle aged female who became short of breath during a dive. This patient had a past history of borderline diabetes and a heart murmur. She walks regularly on a treadmill without issues but stopped swimming recently due

coughing shortness of breath. Her dive was conducted in 55° F water with a 7 mm wetsuit and moderate current. She hadn't been diving in a few years and had about 30 previous dives. Her symptoms started about 15 minutes into the dive while at depth and included coughing into her regulator along with shortness of breath. This caused her to abort the dive. She continued to have cough with pink sputum when back on the dive boat. Differential diagnosis from the group included aspiration, swimming induced pulmonary edema (SIPE), CHF, acute coronary syndrome and pulmonary embolus. The most likely diagnosis was SIPE. The most common presentations are dyspnea/shortness of breath while exercising, immersed in cold water. This is partially mediated by peripheral vasoconstriction but is likely multifactorial. Adjunctive testing usually shows some degree of hypoxemia and chest x-ray with pulmonary edema. Lung ultrasound may show pathologic comet tails. Management is usually supportive with most cases resolving spontaneously. Rare, extreme cases could require more aggressive management and could include diuretics, BiPAP and nitrates.

The next case featured a 51-year-old male with a history of treated hypertension, on/off palpitations and previous episodes of syncope who developed chest tightness, palpitations and syncope. He had sudden onset palpitations lasting 15 minutes and a brief episode of syncope while on the dive boat after a series of dives to 130 feet for 49 minutes, a 75 minute surface interval and a second dive to 63 feet for 60 minutes. He had no issues during the dives. An ECG was shown and the majority of respondents identified a delta wave and short PR interval indicative of Wolf-Parkinson-White syndrome. This is a pre-excitation syndrome which is caused by aberrant electrical conduction through an accessory bundle, bypassing the AV node in an orthodromic or antidromic fashion. Other causes of syncope and sudden cardiac death in healthy, young individuals include hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, long QT syndrome and Brugada Syndrome. Coronary artery disease however, tends to be a more common cause of sudden cardiac death in individuals over 35 years old.

ECG's consistent with the above diagnosis of hypertrophic cardiomyopathy (HCM), arrhythmogenic right ventricular cardiomyopathy (ARVC), long QT syndrome and Brugada Syndrome were reviewed. HCM can be identified by the presence of left ventricular hypertrophy (LVH) notable in the septal leads with deep, narrow Q waves which may be found in the lateral leads. Long QT syndrome can be identified by a "Long QT segment." A normal QT interval is usually 0.37 to 0.44 seconds. The interval should be corrected for heart rate (QTc). In a cursory review of an patient's ECG, the T wave should terminate before the midpoint of the R-R interval. Long QT may be present and place the patient at increased risk for cardiac arrhythmia (R on T phenomenon) as the T wave approaches the P wave. There are multiple ECG presentations of Brugada syndrome. ECG may show a tall, broad appearing R wave with gradual downsloping ST segment in the anterior precordial leads. These leads may also have a "saddle back" appearance. "Epsilon waves" on the other hand, are seen in approximately 30% of cases of ARVC with ST depressions and T-wave inversions in the precordial leads being more common (approximately 85%).

The following case discussion surrounded a 51-year-old female who experienced a "heavy heartbeat." She is a diabetic on insulin who never missed doses, hypertension and hyperlipidemia. She was cleared by a diving physician after being diagnosed with diabetes. She has had increasing fatigue and lightheadedness over the past few days but still conducted two dives at 58 feet for 39 minutes with a 2-hour surface interval and a second dive at 40 feet for 52 minutes. She experienced progressive lightheadedness during the 2nd dive. Her heavy heartbeat started during the dive, and she was noted to be diaphoretic and mildly short of breath when back on the dive boat. Most of the poll respondents selected "administration of orange juice" as the next best step. Most felt this patient was likely experiencing hypoglycemia. A short discussion ensued about the necessity of calling EMS, being seen in the emergency department and return to diving. Most agreed this patient should not return to diving until her blood sugar was stabilized.

The next case discussed was again a 51-year-old with no significant history other than obesity and taking escitalopram for her anxiety disorder. She had difficulty clearing her mask during her second dive of the day. She made an unfortunate rapid ascent from 20 feet and immediately developed chest tightness and shortness of breath. She had no weakness, numbness, tingling or loss of consciousness. Images consistent with gas in the subcutaneous tissues, pneumothorax, and pneumomediastinum were shown and correctly identified by the participants. Physical exam may reveal findings of subcutaneous emphysema (crepitus), decreased lung sounds, tachycardia, tachypnea, JVD, hypoxemia, and potentially an abnormal neurological exam. This most likely represents pulmonary over-pressurization syndrome due to rapid ascent and air trapping, although this individual probably does not have a clinically significant cerebral arterial gas embolism given the lack of neurologic findings. The history of anxiety could also be significant if this rapid ascent was due to a panic situation. This individual should have a thorough fitness to dive exam prior to returning to the water.

Nearing the end of the talk, we discussed a case of a 31-year-old female who developed chest tightness and shortness of breath after a series of dives while on vacation. She has no major medical problems but had a left knee arthroscopy 6 weeks ago. She dove to 130 feet for 49 minutes total dive time with a 75-minute surface interval, followed by a dive to 63 feet for 60 minutes total dive time. She did not violate her no decompression limits according to her dive computer. She felt winded after diving, worsened with walking back to her room. The next morning she felt fatigued, her chest pain worsened with deep breathing and was now short of breath at rest. Her ECG showed an S1Q3T3 pattern with right heart strain. The differential diagnosis was discussed as this is a potentially provocative dive to illicit cardiopulmonary DCS. Pulmonary embolus (PE) was felt to be more likely given she did not have other DCS symptoms, and her ECG and symptomatology are more consistent with pulmonary embolus. Clinical findings of PE include shortness of breath, tachycardia, tachypnea, hypoxemia. Patients may have lower extremity swelling and calf tenderness indicative of deep vein thrombosis. The "PERC" and "Wells Criteria" are clinical decision rules which can help the clinician evaluate PE risk. While ECG was abnormal in this case, it is not often the case and is neither sensitive of specific to diagnose PE. CT angiogram or ventilation/perfusion (VQ) scan remain the primary diagnostic studies to evaluate for PE.

The next case was a 51-year-old with hypertension and hyperlipidemia. This was a new diver completing her open water diver training course. She began coughing during a dive to 30 feet for 22 minutes in 82°F water. She had no rapid ascents or other violations. She felt okay during the dive but developed shortness of breath the next morning. Physical exam findings included mild tachycardia, mild hypoxemia to the low 90's on SPO_2 and fever to 102°F. DCS was unlikely in this case as the dive profile is not provocative. While PE is certainly on the differential due to the tachycardia and hypoxemia, aspiration with pneumonitis or developing pneumonia is more likely. Additional history taking about the dive itself would probably reveal some event where the patient aspirated. While there are certainly many opportunities for a diver to aspirate, training dives typically involve drills such as regulator removal and recovery and alternate air source use. There is risk of aspiration while clearing the regulator underwater if not performed correctly.

The next case discussed was a 35-year-old male who complained of chest tightness and shortness of breath following a dive. His profile was significant with a depth to 180 feet breathing trimix 18/45, a 3 minute bottom time and decompression with 50% and 100% O_2 decompression gases at a PO_2 of 1.6 ATA. Surface interval after that dive was 3 hours, followed by a second dive to 140 feet on Trimix 18/45 for a 25 minute bottom time, and the same decompression gases. His computer began alarming during the second dive after descending past 20 feet. Physical findings upon evaluation included a lacy appearing rash to his chest and back, progressive numbness to his legs, chest pain, cough and shortness of breath. The group was polled again, with the majority feeling this was probably a case of decompression sickness. His dive profiles were provocative and further investigation of his dive computer revealed that he did not reset his computer from 100% O_2 (deco gas from the

first dive), back to his "bottom mix" of trimix 18/45 for the second dive. The computer alarm was likely a "high PO_2" alarm if it thought the diver was breathing 100% O_2 at depth. His computer would not be able to accurately calculate his decompression obligation as it thought he was breathing pure oxygen.

Cardiopulmonary DCS is a rare but potentially fatal form of DCS. Symptoms generally include cough and shortness of breath commonly referred to as "the chokes." This may include frothy, pink sputum, retrosternal chest pain, rapidly progressive hypotension and may result in cardiac arrest and even death. Patients may also improve once put on oxygen or treated with recompression therapy. As discussed above, the differential diagnosis of cardiopulmonary DCS is broad. Immersion pulmonary edema which has similar symptoms, would typically start during the dive while immersed, or at the surface swimming with exertion, and usually in cold water but does not require a "provocative" dive profile.

The final case of a 51-year-old male with chest tightness and shortness of breath following a dive broadens our differential to include cardiovascular disease and heart attack, another potential mimic of cardiopulmonary DCS. This time, the diver has a history of hypertension and hyperlipidemia, previous n-STEMI with a stent to the LAD 4 years ago. He is also a former smoker. He is an experienced technical diver who made a single dive to 180 feet for 20 minutes on trimix 18/45 with 50% and 100% O_2 decompression gases. He had no violations on his computer and completed the dive according to a pre-determined decompression schedule. This was conducted in the ocean with no current, 72°F water, using a wetsuit. He developed chest tightness, left arm numbness, cough and shortness of breath after climbing out of the water and removing his gear. His ECG on initial evaluation demonstrated ST elevations in lead V1-V3, with hyperacute T waves. It was agreed upon by the group that this is like a case of ST-elevation myocardial infarction (STEMI). While DCS is a possibility given the deep dive this diver performed, the medical history, symptoms and ECG are more consistent with a heart attack.

In summary, the differential diagnosis of cardiopulmonary decompression sickness is broad. DCS is largely a clinical diagnosis and identified with a thorough medical and dive history, along with a good physical examination. Adjunctive testing such as chest xray, chest CT, ECG, and laboratory evaluation is sometimes necessary to confirm or rule out a particular diagnosis.

Abdominal Issues in Diving: From Belching to Lethal

Aaron Heerboth, MD

Introduction

I would like to thank Dr. Denoble for inviting me to speak and especially for considering this generally neglected topic. Abdominal complications from diving is generally neglected entirely from most text books or at best addressed with a cursory paragraph. The GI tract is a gas filled space, just like the lungs or middle ear, and is susceptible to the same physics that can cause injury while diving. Given the lack of emphasis seen in texts, the literature, and at most hyperbaric conferences, you may find that the incidence of minor abdominal issues is quite surprising, and although less common, life-threatening abdominal injuries do occur more often than you would likely suspect.

There are really only two simples goals for this talk. I want you to know a couple common causes of abdominal pain in divers. It is not necessary to memorize all things that can cause (or mimic) intraabdominal diving injury. But more importantly, I hope to demonstrate an approach so that you can diagnose the rare and life threatening stuff. And equally importantly, have a sense of who should probably not be diving in the first place. We cannot necessarily follow the guidelines as you will see

Approach to abdominal complaints in a diver

The abdomen is a black box. Abdominal pain can be secondary to decompression sickness, which frequently (and often erroneously) jumps to the top of the differential when a diver presents to the Emergency Department. There are numerous other dive-related causes of abdominal pain, as well as the usual differential diagnosis for anyone presenting to the ER with abdominal pain. If you decide to dive a patient who developed periumbilical/poorly localized pain 3 hours after surfacing only to discover their pain was now localized to the RLQ after completion of a Table 6, you would certainly not be the first to discover that appendicitis is not readily treatable with hyperbaric oxygen.

This lecture is primarily case-based to emphasize the approach to both the acutely injured diver and the fitness to dive patient. These cases are chosen because they highlight important and recurrent concepts about mechanism of injury, or risk for potential injury in a fitness to dive scenario.

Case 1

- A 35-year-old male is performing his very first dive in a PADI open water certification class
  - Maximum depth is 30 feet
- During a **controlled** ascent with his BCD, he develops a sudden onset of epigastric pain, it seems the BCD is squeezing his abdomen, and he has a sensation of tearing in his chest
- Upon reaching the surface, he has significant shortness of breath and anxiety
- The instructor removes the diver's weight belt and BCD and places him on 100% oxygen by face mask. The wet suit is also removed as it begins feeling very tight

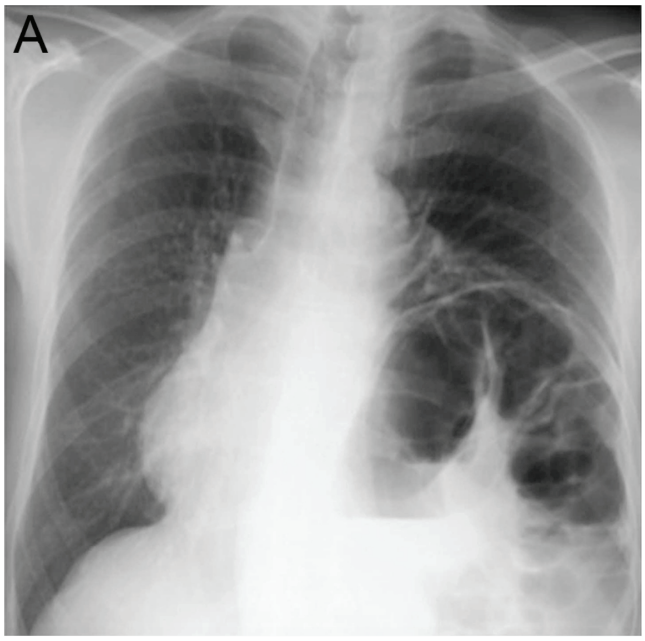

Figure 1. X-ray shows loop of bowel in the thoracic cavity. CT scan confirms diaphragmatic rupture. EKG is normal

To step away briefly from the case…don't forget basic first aid. 100% O2 may help, and is very unlikely to cause harm until you identify what is going on.. Could this be decompressions sickness? Definitely not. Pulmonary barotrauma? It's possible though ascent was slow and controlled. AGE also seems less likely. Our usual diving differential isn't very satisfying here.

- By the time an ambulance arrive 15 minutes later, his symptoms have resolved fully
- Vitals BP 130/80, HR 88, RR 18, O2 99%
- Physical exam notable for clear lungs and normal neurological exam
- He is told by medics personnel he had a panic attack
  - He declines transport to the ER
- The following day, he begins experiencing progressive nausea, anorexia, malaise, and shortness of breath. On day 4 he decides to see his primary doctor
- Vitals HR 108, BP 120/80, RR 18, T 98.6, O2 95%, exam notable for dry mucous membranes, mild epigastric tenderness, and diminished breath sounds at the left lung base
- Thoughts on differential? Any quick diagnostic tests you would like?
  - His primary doctor starts with an EKG and chest x-ray

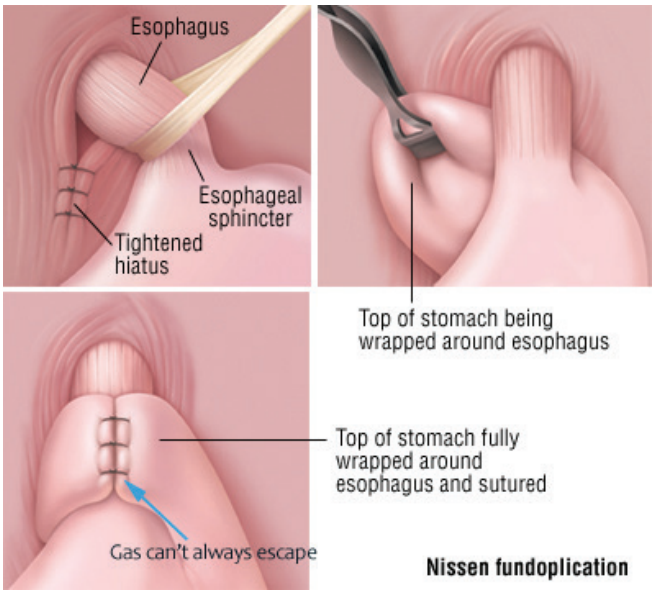

Figure 2. Nissen fundoplication

A tiny bit of additional history:

- The patient's only medical history was a long history of GERD with a hiatal hernia
- He had wanted to learn to dive for years, but he was told his hiatal hernia and GERD made him ineligible.
- So…he decided to have an elective Nissen Fundoplication 3 months prior
- His GERD symptoms had resolved fully! And he was cleared by his surgeon to begin scuba diving

What happened here? It is essential remember the GI tract is a gas filled space. It is susceptible to the same forces that may cause barotrauma to the lungs, sinuses, and ear.

Following a Nissen fundoplication (Figure 2), the loop of stomach intended to increase tone at the lower esophageal sphincter can prevent pressurized gas, ingested at depth, from escaping. This can lead to rupture of the diaphragm as in this case, as well as gastric rupture. There are numerous reported cases in the literature, but exact incidence of complications following Nissen is unknown. It is not considered a long term contraindication to diving in any of the major guidelines, but it is certainly something that should be considered carefully on a case by case basis

Case #2

- 4 days into a dive trip in Australia, a 41 year old previously healthy male diver develops burning epigastric pain shortly after his second dive of the day
- He is diving on air.
- First dive is 80 feet for 30 minutes.
- Three hour hour surface interval
- Second dive 60 feet for 30 minutes.
- No alarms at any point on his dive computer
- He has significant prior dive experience, and has had similar symptoms several times before (always after surfacing and usually after multiple dives) that resolved spontaneously.
- However, this time it is quite a bit worse and more persistent.
  - He suspects he has DCS and is driven by a friend to a local ER where there is a hyperbaric chamber
- He has normal vitals. Exam with mild diffuse abdominal tenderness. Abdominal x-ray interpreted as ileus with large gastric bubble.
- This is considered confirmation of "abdominal decompression sickness" and he is treated with a USN Table 6.
- **Symptoms resolve over the first hour of HBO treatment**

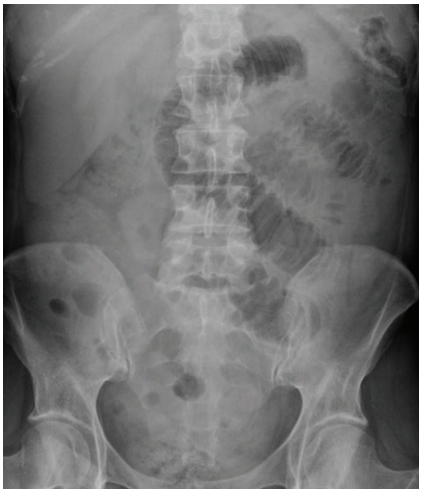

Figure 3. Abdominal x-ray shows ileus pattern without evidence of free air or small bowel obstruction

- He returns to diving 2 months later and again experiences similar symptoms of persistent epigastric abdominal pain several days into his trip
- AGAIN, he is treated with a USN table 6 with symptoms resolution
- An ECHO with bubble study is negative, and when he returns to diving he begins using Nitrox an air tables.
  - He develops symptoms yet again on a third trip after a single 50 feet for 45 minute dive
  - When he goes to the ER for his third HBO treatment, the correct diagnosis is made

So what is the correct diagnosis? What is going on here? This is actually a case of something which is extremely common, but likely under-reported with often somewhat milder symptoms.

GI barotrauma and reflux esophagitis

- Stomach over-inflation is a common problem causing abdominal pain during or shortly after ascent
- It is caused by gas which accumulates in the GI tract at depth and expands during ascent
- Several causes of GI tract gas accumulation
  - Most commonly seen in divers who forcefully valsalva to clear their ears and descend head first
  - Also associated with anxiety (air swallowing or aerophagia)

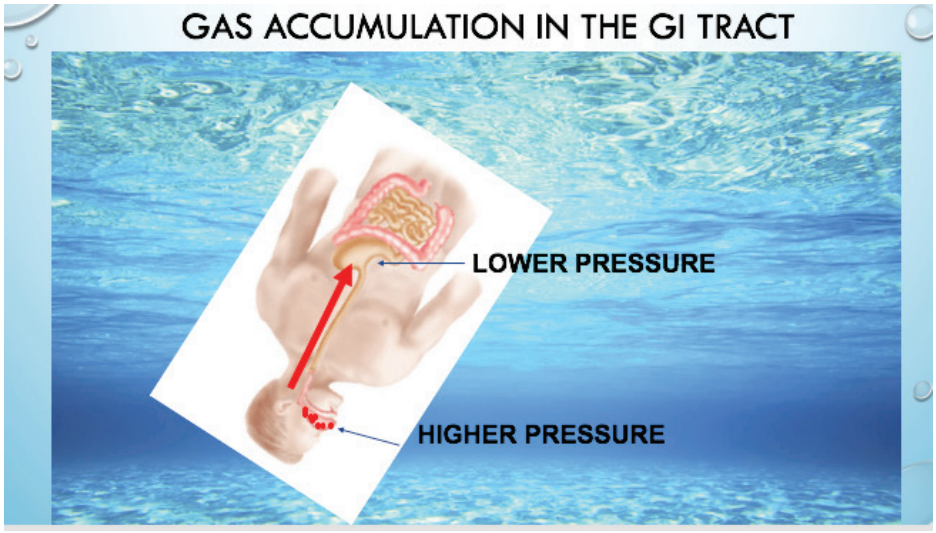

Figure 4. Gas accumulation in the GI tract

In head first descent, gas, depicted as red dots in Figure 4, travels from the higher pressure it experiences at a slightly greater depth in the oropharynx to lower pressure in the stomach which is at a shallower dept. Upon ascent, the accumulated gas expands causing GERD symptoms or potentially barotrauma to the stomach or other parts of the GI tract.

Exact incidence of GI barotrauma is unknown

- 1975 survey of 2053 divers found that 111 (5.4%) had experienced abdominal pain after a recent dive
  - A majority of those reported head first descent
  - Many reported difficulty clearing their ears as well.
    Frequent swallowing in order to clear ears could result in additional gas accumulation in the stomach

Management

- Antacids/PPI/H2 blocker may help with reflux symptoms
- No large meals or carbonated beverages before a dive
- The majority of cases resolve/can be prevented with feet first decent
- Belching during ascent if necessary also provides relief

Complications

- Multiple cases of **gastric rupture** reported in the literature
- Important consideration in a diver with severe abdominal pain or distension after surfacing
- Obtain upright chest x-ray as a screen, or straight to abdominal CT
- Surgical emergency
- Broad spectrum antibiotics, IV fluids
- **Reflux and aspiration** can also be life-threatening under water

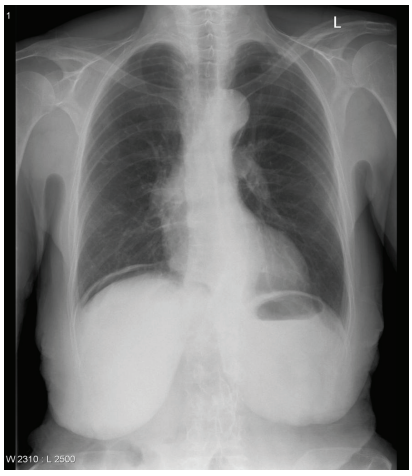

Figure 5. Abdominal XR of patient with gastric rupture

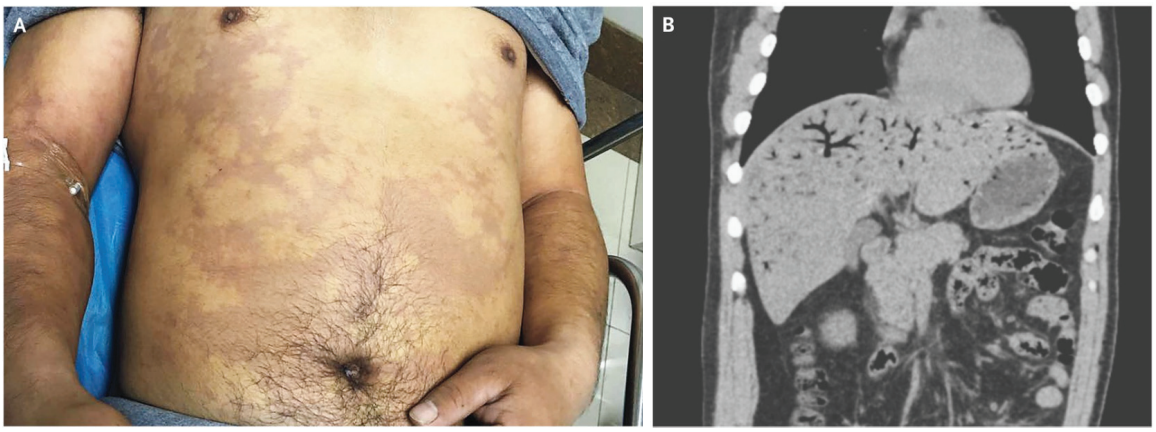

Figure 6 A and B. CT shows significant portal venous gas

CASE #3

- A 38 year old male completes a single dive to 80 feet for 55 minutes on air
- No issues during the dive, controlled ascent
- He reports he was using a dive computer and denies any alarms
- 2 hours later, he develops diffuse abdominal discomfort pain to his right shoulder, generalized body aches, and palpitations
- Vitals HR 118, BP 110/70, RR 18, O2 98% T 98.6
- Physical exam is notable for diffuse rash as well as moderate tenderness throughout the abdomen, greatest in the RUQ, rash as depicted below
- EKG sinus tachycardia. Labs notable for mildly elevated ALT/AST, Lactate 3.4 and and elevated CPK.
- The treating physicians suspect DCS.
  - However, given his abdominal pain and labs, CT abdomen obtained (see figure 6 below).

Finally, we have something we are all familiar with, a case of decompression sickness. Abdominal involvement in DCS however is not well studied.

- *Exact incidence unknown*
- *US Navy Dive Manual does not include GI in its list of symptoms*
- *DAN reports "bowel and bladder" symptoms in 3-5 percent of DCS cases*
- *Likely much higher*
- *2013 case series in Japan demonstrated portal venous gas in 4 of 9 patients with DCS for which CT chest or abdomen was obtained*
- The patient has improvement in his rash on 100% O2
- The initial abdominal pain improves somewhat as well.
- The nearest HBO chamber is a two hour helicopter flight or 4.5 hour ambulance ride away
  - Given symptom improvement, the on call hyperbaric physician feels the patient is stable for ground transport
- Unfortunately, in route, the patient begins to develop a new "bandlike" pain across his flank area radiating towards the abdomen
- He reports progressive paresthesias to his lower extremities. By the time he arrives at the treating hospital, he has significant bilateral lower extremity weakness (4/5 motor LLE and 4+/5 RLE on exam)
- He is hyperreflexic with upgoing Babinski bilaterally
  - Concerning for spinal DCS
- Treated with USN Table 6 with full extensions with near resolution of symptoms
- Tailing treatment Table 9 three times over following two days with full recovery
- ***Band-like back, flank, or abdominal pain can be a harbinger of spinal badness and is frequently a presenting symptom for spinal DCS***

Figure 7. This is the actual clinic/hospital, the only facility on the island. Needless to say, it is not well equipped for treating this patient

CASE #4

- A 28 year old male who reports no significant past medical history on his PADI health questionnaire (which he filled out honestly) during his certification course last year, is on a multi-day dive trip off the coast of Thailand.
- On day 3, he develops a gnawing epigastric discomfort during his first ascent from a 45 minute dive at 60 feet. Upon reaching the surface, he belches several times and the discomfort resolves
- After a 2 hour surface interval, he makes a second dive to 45 feet for 30 minutes.
- He again notices the same discomfort on ascent
- Upon reaching the surface, his pain resolves, again with belching.
- 2 hours later, shortly before the dive boat makes its return to the island of Koh Tao, he **vomits a large amount of bright red blood** and has a melanotic bowel movement
- He is rushed to the local clinic and continues to vomit about 600 ml of blood per hour.
- His initial vitals are bp 120/70, HR 105, RR18, T 98.4, O2 100%
- Initial Hgb is 15.4 g/dl
- IV fluid resuscitation is initiated
- The patient reports that 8 years ago, after a 500 mile trek along the Appalachian Trail, he was diagnosed with cryptogenic cirrhosis when he complained of fatigue and abdominal distension
- He had a paracentesis and was started on a diuretic. He was able to wean off the diuretic several years later and had required no further treatment
- EGD at that time revealed grade I esophageal varices
- Medivac to Bangkok, a 120 minute flight
- Hypotensive to the 80s on arrival, repeat Hgb is 6 g/dl
- Given 4u additional PRBCs, emergent EGD with grade IV varices which were banded
- He was released from the hospital several days later and recovered fully

So, what caused this patient to have a nearly fatal GI bleed while diving? His cirrhosis had been stable for years and it was unlikely to be coincidence. There are several factors that could be a play. First, the immersion response (particularly in cold water) shunts blood to the central circulation and can dilate varices and lead to increase portal pressure. Second, given is development of pain while surfacing, he likely had a component of GI barotrauma during ascent. The shearing force placed on the varices from gas volume expansion in the stomach and esophagus could certainly have contributed to bleeding. Third, acid reflux, which again is often made worse by ascent, can cause erosion of the esophageal vessels. Finally, this patient had a large meal prior to his dive, which leads to hyperemia of the GI tract and likely should be discouraged for potentially vulnerable divers.

Remember, this patient honestly and accurately filled out his PADI medical questionnaire. On the final page below are the medical screening questions, with those which address GI issues circled in red.

You will notice that cirrhosis or varices does not appear. Our Nissen fundoplication case also would get a pass. This is not to say that all possible ailments should be on the questionnaire for recreational divers, but it is certainly something that we as dive physicians must be aware of and address on the patient and diver population levels. Cases like the cirrhotic GI bleed do not appear particularly common based on available data, though it is certainly possible it is under-reported; it is unlikely that most physicians (excluding those attending this lecture, of course) would make the connection between an upper GI bleed and a possible diving injury. Although variceal bleeding in divers has historically been fairly rare, it may begin to increase. The incidence of NASH cirrhosis, for example, is rapidly increasing in the US, but we do not know how many NASH patients are diving. Changing health demographics, combined with the aging of the general diving has the potential to significantly to change the pattern of dive-related illness and injury.
We must periodically reassess our fitness to dive standards. The list of "classic" abdominal contraindications is short and may not be adequate. Below is the list from a widely referenced dive medicine textbook:

Contraindications: (brief explanation)

1. **Recent abdominal surgery** (wait 6 weeks)
2. **Inguinal or ventral hernias** (gas trapping leading to incarceration)
3. **Peptic ulcer disease** (usually the consideration is reflux/aspiration, though obviously barotrauma and gastric rupture could be an issue)
4. **Colostomy** (largely related to bag spilling, not a true contraindication)
5. **Paraesophageal** hiatal hernia

There are numerous things to take away from this brief list. Perhaps most obvious is that it is far from comprehensive. A broad range of other GI disorders, such as inflammatory bowel disease, are not considered and clearly pose some degree of risk of complications. They need to be approached on a patient by patient basis.

Many GI diseases fall on a spectrum. Most divers with well-controlled "GERD" or "PUD" can dive safely. Hiatal/paraesophageal hernia also falls on this list, and yet the procedure that is often used to treat and potentially cure both (Nissen fundoplication), may actually be much more dangerous and yet is not considered.

Summary

- Keep a broad differential on the acutely ill diving patient with abdominal pain
- Consider the physiology involved (potential for abdominal barotrauma, shunting of blood, etc) as well as the dive profile
- Careful history and exam, and remember that, particularly for divers presenting primarily with abdominal complaints, imaging is potentially your friend to exclude intraabdominal surgical emergencies prior to hyperbaric treatment for DCS
- Fitness to dive should be done on a case by case basis for many patients with abdominal issues. The available guidelines are not helpful for many cases, and there is insufficient data most abdominal problems.

R·S·T·C

UNDERSEA & HYPERBARIC MEDICAL SOCIETY

MEDICAL STATEMENT

Participant Record (Confidential Information)

Please read carefully before signing.

This is a statement in which you are informed of some potential risks involved in scuba diving and of the conduct required of you during the scuba training program. Your signature on this statement is required for you to participate in the scuba training program offered

by_____and
Instructor

_____located in the
Facility

city of_____, state/province of _____.

Read this statement prior to signing it. You must complete this Medical Statement, which includes the medical questionnaire section, to enroll in the scuba training program. If you are a minor, you must have this Statement signed by a parent or guardian.

Diving is an exciting and demanding activity. When performed correctly, applying correct techniques, it is relatively safe. When established safety procedures are not followed, however, there are increased risks.

To scuba dive safely, you should not be extremely overweight or out of condition. Diving can be strenuous under certain conditions. Your respiratory and circulatory systems must be in good health. All body air spaces must be normal and healthy. A person with coronary disease, a current cold or congestion, epilepsy, a severe medical problem or who is under the influence of alcohol or drugs should not dive. If you have asthma, heart disease, other chronic medical conditions or you are taking medications on a regular basis, you should consult your doctor and the instructor before participating in this program, and on a regular basis thereafter upon completion. You will also learn from the instructor the important safety rules regarding breathing and equalization while scuba diving. Improper use of scuba equipment can result in serious injury. You must be thoroughly instructed in its use under direct supervision of a qualified instructor to use it safely.

If you have any additional questions regarding this Medical Statement or the Medical Questionnaire section, review them with your instructor before signing.

Divers Medical Questionnaire

To the Participant:

The purpose of this Medical Questionnaire is to find out if you should be examined by your doctor before participating in recreational diver training. A positive response to a question does not necessarily disqualify you from diving. A positive response means that there is a preexisting condition that may affect your safety while diving and you must seek the advice of your physician prior to engaging in dive activities.

Please answer the following questions on your past or present medical history with a YES or NO. If you are not sure, answer YES. If any of these items apply to you, we must request that you consult with a physician prior to participating in scuba diving. Your instructor will supply you with an RSTC Medical Statement and Guidelines for Recreational Scuba Diver's Physical Examination to take to your physician.

_____ Could you be pregnant, or are you attempting to become pregnant?

_____ Are you presently taking prescription medications? (with the exception of birth control or anti-malarial)

_____ Are you over 45 years of age and can answer YES to one or more of the following?
- currently smoke a pipe, cigars or cigarettes
- have a high cholesterol level
- have a family history of heart attack or stroke
- are currently receiving medical care
- high blood pressure
- diabetes mellitus, even if controlled by diet alone

Have you ever had or do you currently have...

_____ Asthma, or wheezing with breathing, or wheezing with exercise?

_____ Frequent or severe attacks of hayfever or allergy?

_____ Frequent colds, sinusitis or bronchitis?

_____ Any form of lung disease?

_____ Pneumothorax (collapsed lung)?

_____ Other chest disease or chest surgery?

_____ Behavioral health, mental or psychological problems (Panic attack, fear of closed or open spaces)?

_____ Epilepsy, seizures, convulsions or take medications to prevent them?

_____ Recurring complicated migraine headaches or take medications to prevent them?

_____ Blackouts or fainting (full/partial loss of consciousness)?

_____ Frequent or severe suffering from motion sickness (seasick, carsick, etc.)?

_____ Dysentery or dehydration requiring medical intervention?

_____ Any dive accidents or decompression sickness?

_____ Inability to perform moderate exercise (example: walk 1.6 km/one mile within 12 mins.)?

_____ Head injury with loss of consciousness in the past five years?

_____ Recurrent back problems?

_____ Back or spinal surgery?

_____ Diabetes?

_____ Back, arm or leg problems following surgery, injury or fracture?

_____ High blood pressure or take medicine to control blood pressure?

_____ Heart disease?

_____ Heart attack?

_____ Angina, heart surgery or blood vessel surgery?

_____ Sinus surgery?

_____ Ear disease or surgery, hearing loss or problems with balance?

_____ Recurrent ear problems?

_____ Bleeding or other blood disorders?

_____ Hernia?

_____ Ulcers or ulcer surgery ?

_____ A colostomy or ileostomy?

_____ Recreational drug use or treatment for, or alcoholism in the past five years?

The information I have provided about my medical history is accurate to the best of my knowledge. ***I agree to accept responsibility for omissions regarding my failure to disclose any existing or past health condition.***

_____ _____ _____ _____
Signature Date Signature of Parent or Guardian Date

Page 1 of 6

Typical ENT Presentations & Differential Diagnosis of Decompression Illness

J. Nicholas Vandemoer, MD, FACS

Abstract

The differential diagnosis between decompression sickness and other entities in the ENT area can be difficult, especially within the cochleovestibular system. As with all diving injuries, the key is first obtaining the dive history with a timeline of the appearance of symptoms. Next, a past history of diving-related problems which, when coupled with the presenting symptoms, often makes the diagnosis. A past history of DCI, eustachian tube dysfunction, vertigo, current history of a URI, sinus problems or allergy will help make the diagnosis. A must is a detailed ENT physical examination looking for the Tullio phenomenon and the Hennebert's sign, and a neurologic examination that includes the HINTS examination. A history of vertigo and/or hearing loss during or after the dive will lead to a possible diagnosis of inner ear decompression sickness (IEDCS), inner ear barotrauma (IEBt), perilymph fistula, middle ear barotrauma (MEBt), alternobaric vertigo, or of a central CNS source. Other neurologic ENT pressure-related problems are important to consider, including alternobaric facial nerve paralysis from middle ear overpressurization and trigeminal paresthesia occurring from pressure effects on the nerve within the maxillary sinus. Blindness can be caused by sphenoid sinus overpressurization, and an orbital hematoma can lead to visual disturbance. Air diverted from the inner ear, middle ear, and sphenoid sinus dehiscence can cause pneumocephalus.

Introduction

Thank you, Dr. Denoble, for inviting me to be part of this stellar and important conference. I'm very honored to be able to present a discussion of diving ENT problems that must be differentiated from DCI.

We will start with the ear, the most common problem encountered in diving. The differentiation of decompression illness of the inner ear, from inner ear barotrauma, and middle ear barotrauma can be very difficult. I'm going to give you some pointers that will help make it easier to make this diagnosis, then go on to other ENT diving problems that may mimic DCS. In particular, we will talk about sinus barotrauma, facial nerve baroparesis, and orbital barotrauma.

This is a case of a 37-year-old female marine biologist who was studying a Caribbean reef. On her third dive of the day, she descended to 110 feet (in a planned reverse profile dive) to drill out a core sample. She had no trouble with

the descent, and no problems on ascending, with a total dive time of 10 minutes. On the surface, however, she immediately felt severely vertiginous with some nausea, and after she got on the boat, one episode of vomiting. She had a feeling of fullness in the right ear. On the boat, she lay on her right side and would not let us turn her over to look in the right ear because that exacerbated her severe vertigo. When lying on the right side, the vertigo came and went intermittently. When she got to shore she was finally able to turn for us to look in the right ear. Both tympanic membranes were normal. The Weber test lateralized to the left. What is the preliminary diagnosis?

Otology

I'd like first to discuss inner ear decompression sickness (IEDCS) to help us compare and contrast to the other otologic entities.

Let's talk about the inner ear vascular anatomy. The vestibular vascular supply has multiple branches that don't collateralize well, unlike the cochlea, where the arterial supply is radial and straightforward with excellent perfusion. The vestibular vascular channels are much smaller, and the perfusion is much slower. Besides, the cochlea has a smaller tissue volume compared to the vestibular system.

The other anatomic point is that with any right to left shunt, bubbles will travel up the brachiocephalic system, into the right carotid artery and therefore directly into the right inner ear. It is a "straight shot" from the left heart to the right inner ear.

These two facts mean that the right inner ear vestibular system is affected about 80% of the time.

The nitrogen washout half time of the brain is 1.1 seconds, and by contrast, the nitrogen washout time of the inner ear circulation is 8.8 seconds. This supersaturation of the inner ear can be made worse by dehydration. The arteriolization of bubbles created by a PFO or missed decompression or marginal dives enter the inner ear circulation, exacerbating the already existing supersaturation of this organ. We, therefore, have the picture of a right-sided vestibular inner ear injury in a patient with a clinically significant PFO resulting in inner ear decompression illness. Is this the likely diagnosis in our patient, or is it inner ear barotrauma (IEBt)?

Of all severe DCI cases, 24 to 34% have cochleovestibular symptoms. Therefore, IEDCS is not necessarily diagnostic of generalized DCI.

How do you make the diagnosis of IEDCS? The dive history has to be compatible; that is a prolonged deep dive or perhaps gas-switching at depth. The symptoms start at or soon after surfacing, with 85% of symptoms appearing within an hour, the median 15 to 20 minutes, most within 6 hours. There are usually no otologic symptoms on descent or ascent, with 76% vestibular, 17% mixed and 6% cochlear symptoms on arrival at the surface. From our previous anatomic discussion, you can see why there are predominantly vestibular and not cochlear symptoms. Rotary vertigo is prominent, associated with vomiting. There is ataxia, with falling toward the affected side. There is a hearing loss on the affected side, most often described by the diver as a blockage feeling. There is tinnitus in that ear; there is no pain. It is extremely rare to have this happen bilaterally. In 17% to 48% of cases, there are other associated DCI symptoms or signs. A clinically significant PFO is present in approximately 80% of cases.

Making a firm diagnosis is difficult. What helps is obtaining a past history of DCI, inner ear DCS, or inner or middle ear barotrauma. It is surprising how many of the patients are repeat offenders, but when you think of the common thread of a PFO, you realize why that is the case. For the most part, the tympanic membrane examination is normal. Of course, if you have examined asymptomatic divers ears, you know that they are not "normal"! There is always some sign of mild barotrauma that is not clinically significant. The 512Hz tuning fork examination demonstrates a sensorineural hearing loss on the affected side. For the first few hours, nystagmus is seen and directed to the affected side. Later, nystagmus might be directed to the good ear as central compensation begins to occur. When testing for lateral gaze nystagmus, it's important to eliminate fixation by placing a sheet of white paper on each side of the head

Table 1. Inner ear vs. cerebral involvement

HINTS component	IEDCS	CNS DCI or stroke
Head Impulse Test	Catch-up nystagmus on the affected side	No catch-up nystagmus
Nystagmus	Unidirectional, spontaneous nystagmus	Lateral gaze, direction changing nystagmus
Test of Skew	No skew movement	Skew movement present

as it's tested. Even better, Frenzel glasses are ideal to use when examining for nystagmus.

As long as the patient has spontaneous nystagmus, the HINTS examination (**H**ead **I**mpulse, **N**ystagmus, and **T**est of **S**kew) is the next step. The examination has three components: determine that the patient has spontaneous nystagmus, then perform the head impulse test and then the test of skew. This test helps to differentiate IEDCS from stroke or CNS DCI.

The spontaneous nystagmus must be unidirectional. If the lateral gaze nystagmus is direction-changing, the lesion is more central, and you worry about brain DCI or stroke. The Head Impulse Test is performed by taking the patient's head between your hands and slowly rotating it on the vertical axis until the neck is loose, then suddenly bringing it to the midline in an arc of about 20 degrees. You do this by testing with the quick motion starting both to the right and left. There is no catch-up nystagmus in the normal individual; that is, there is no nystagmus directed toward the side which is toward the previous head position. In the injured diver, there will be catch-up nystagmus on the affected side, indicating that the inner ear is affected on that side. If the eyes are rock solid and there is no catch-up nystagmus, then the problem is a central, possibly a stroke. The test of skew is the last part of the HINTS exam. You do this using the cover-uncover test by putting your hand over each eye separately, and then uncovering the eye, looking a the uncovered eye for vertical movement, often associated with a lateral component. If there is this skew movement, it indicates a CNS problem.

Thus we are reassured when we have unidirectional nystagmus, abnormal head impulse test, and no vertical skew deviation. Otherwise, a CNS diagnosis has to be entertained, including stroke.

The post-shake nystagmus is similar to the head impulse test. A sharpened Romberg and Fukuda stepping test help to firm up the diagnosis of an inner ear problem. Of course, if in your initial general neurologic exam you come upon a positive finding, then you have made the diagnosis of DCI, presenting with vertigo. By the way, if there is no improvement with the administration of normobaric oxygen on the way to the facility, you have to worry that the symptoms may represent a CNS vascular origin.

So we have made the diagnosis of IEDCS quickly without the need of any expensive additional instrumentation except for Frenzel glasses. We've ruled out a CNS source for the problem, such as a Wallenberg Syndrome.

Back to our case of the graduate student. Her history was negative for previous DCI, Eustachian tube inflation difficulties or nasal symptoms. The general neurologic examination ruled out any CNS DCI component; it appeared to be an inner ear problem, but is it IEDCS or Inner ear barotrauma?

How do we differentiate IEDCS from inner ear barotrauma (IEBt)? As Joseph Farmer MD wrote in 1977, "any diver who experiences persistent vertigo, neurosensory hearing loss or tinnitus following dives in which decompression sickness is unlikely, should be considered as a possible cause inner ear barotrauma and perilymph fistula"[9]

Well, IEBt is almost always secondary to eustachian tube clearing difficulties, with forced clearing attempts setting up pressure differentials between the CNS and inner ear fluids. Three results can occur 1) inner ear hemorrhage, 2) Reissners membrane tears, or 3) perilymph fistula formation. For example, at 3.9 fsw (1.2 m), with a forceful Valsalva maneuver trying to equalize the middle ear pressures, a pressure differential

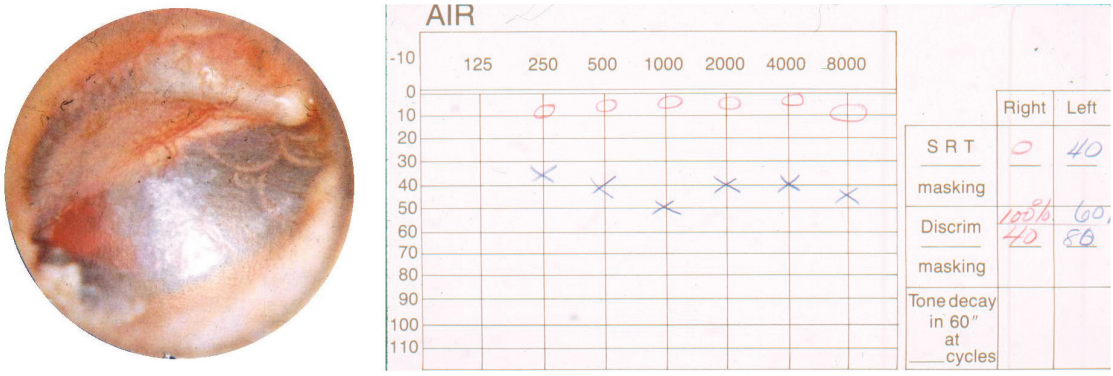

Figure 1. Evidence of middle ear barotrauma (left) and a corresponding flat sensorineural hearing loss due to inner ear hemorrhage (right).

of about 300 mmHg is created between the CSF and still un-cleared negative middle ear pressure, enough to cause any of the three problems mentioned above. This pressure differential in the inner ear can cause cochlear hemorrhage and Reissner's membrane tears. Transmission of the pressure from the CSF to the inner ear is through the cochlear aqueduct or endolymphatic sac. Having better perfusion and having the pressure transmitted by the cochlear aqueduct predisposes the cochlea to suffer more damage than the vestibule.

With IEBt, as compared to IEDCS, the dive history is usually not compatible with decompression illness. The dive is shorter and not as deep. There is a prominent history of failed Eustachian tube clearing. There is usually a history of Yo-Yo diving. Almost 90% of the divers have symptoms that begin while still in the water with symptom development in either the descent or ascent. Symptoms may not be fully developed until after reaching the surface, even up to several hours afterward. The hearing loss is prominent and bothersome and may fluctuate, and may be either mild or profound. Vertigo, if there is any, may be mild and may fluctuate, depending on the location of the injury. Severe vertigo may indicate a fistula. There may be ear pain, and there is tinnitus.

Examination usually shows a barotraumatized tympanic membrane, but may be normal. There is a sensorineural hearing loss in the affected ear (Fig 1). There is usually nystagmus. The HINTS exam is positive for an inner ear problem.

A round or oval window fistula occurs when the generated pressures overcome the window seals, the round window being the most common. There are two theories to explain the etiology of the fistula. One is the explosive theory where the CNS and increased inner ear fluid pressures rupture the membrane into the negatively pressured middle ear. The other is the implosive theory, where the membrane ruptures inward after the middle ear is suddenly cleared, with pressure directed medially, displacing the membrane inward. Either way, these mechanisms let middle ear air enter the vestibular system, causing bubbles to form in the inner ear fluids, which then enlarge on the ascent, causing rupture of the inner ear membranes.

One-third of the IEBt patients have a fistula. The vertigo is more prominent, and there are nausea and vomiting. The hearing loss may fluctuate but may worsen with time. The Hennebert's sign can be positive for a fistula, although rarely it can be negative. This sign can be elicited by sealing the ear canal with the pneumatic otoscope, applying positive and negative pressure to the tympanic membrane and observing for ocular deviation to the involved side. The Tullio phenomenon can be positive as well. It is performed by exposing the affected

ear to the sound of around 80 dB, at 250 and 500 Hz., causing vertigo, nausea and sometimes eye deviation to the affected side. The audiogram will demonstrate a sensorineural hearing loss with an associated discrimination score loss, similar to the IEDCS findings. The high frequency sloping loss indicates a relatively poor prognosis for recovery, whereas a flat hearing loss (Fig 1) indicates a good prognosis.

It is rare that IEBt co-exits with IEDCS. When there are cochleovestibular symptoms during or after the dive, 81% are usually due to IEDCS and 19 % due to IEBt.

So how do you differentiate IEBt and IEDCS? First of all, by the depth and length of the dive. The symptoms often start underwater and can be present on descent or ascent. There is a history of aggressive ear clearing maneuvers. There are no symptoms or signs of decompression illness. The audiologic symptoms are prominent compared to IEDCS, where the symptoms are mostly vestibular. The audiologic symptoms progress over time, whereas those of IEDCS remain stable or improve. Symptoms and signs worsen with compression, especially with a fistula, whereas IEDCS symptoms improve.

With this differential being so difficult, often it is prudent to perform bilateral myringotomies and compress, since IEDCS requires emergent treatment, with recovery based on the time to hyperbaric treatment.

IEBt is not frequent, despite the frequent occurrence of middle ear barotrauma, because of significant labyrinthine tissue compliance and differences in the pressure transmission through the cochlear and vestibular aqueducts.

There is an antecedent inner ear problem that can cause a problem in the differential with both entities just described. Superior Canal Dehiscent Syndrome is a developmental entity consisting of a unilateral or bilateral bony dehiscence of the superior semicircular canals. It is characterized by pressure or sound induced vertigo or oscillopsia associated with torsional eye movements. A nose-pinch Valsalva maneuver might bring on these symptoms, which may mimic a fistula. The examination elicits a positive Tullio phenomenon or Hennebert's sign. Some of these patients can remember a precipitating factor when these symptoms began; unfortunately, often it is diving. I mention this entity to emphasize how important it is to take a good history, which might elucidate the existence of this problem before the diving incident, thus avoiding compression. An ENT consult is best to try to differentiate this problem. An emergency high-resolution CT of the temporal bones might be in order.

Middle ear barotrauma (MEBt)

Let's move on to middle ear barotrauma (MEBt). The pathophysiology is characterized by poor eustachian tube clearing. On the descent, the eustachian tube becomes locked at 3.9 fsw (1.2 msw) and at that point requires significant nasopharyngeal pressure to open, sometimes up to 250 mmHg. MEB is the most common diving injury, found in 10% of experienced divers and 30% of novice divers. It happens on descent, often in the head-down position. There is sometimes a history of previous eustachian tube problems, or a history of a current URI, allergy or sinus problems. There is often a history of pseudoephedrine use. Medical problems account for 50 % of the etiology, with poor or inadequate training accounting of the rest. It only takes about 400 mmHg of middle ear pressure differential from the ambient water pressure to cause significant middle ear/ tympanic membrane injury or rupture of the tympanic membrane.

The symptoms include ear fullness, pain, decreased hearing, tinnitus, and even some vertigo. Recall that with IEDCS there is usually no pain. There may be mild vertigo or unsteadiness due to differential pressures in the middle ears.

The TEED gradation of middle ear trauma gives a visual representation of the amount of barotrauma to the middle ear. The foreshortening of the manubrium gives the best idea of how much negative pressure exists in the middle ear.

Table 2. Differential diagnosis of diving vertigo

	IEDCS	IEBt	MEBt	Alternovaric vertigo
Incidence	Rare	3Rd most common	2Nd most common	Common
Dive exposure	= or > deco limits	Any, usually shallow	Any, usually shallow	Ascent
Onset	At depth, ascent, post-dive	Descent, ascent, post-dive	Descent, ascent, post-dive	Ascent
Inner ear symptoms	76% Vestibular, 17% mixed, 6% cochlear	Cochlear, +- vestibular	Cochlear	Vestibular
Eustachian malfunction	No	Yes	Yes	Yes
Tullio, Herbert	No	Yes (with PLF)	No	No
Head Impulse Test	Yes	No (yes w/fistula)	No	No
Otic barotrauma	No	Yes or no	Yes	No
DCI symptoms	Possible	No	No	No
iEDCS=inner ear decompression sickness IEBt=inner ear barotrauma MEBt=middle ear barotrauma ALT. VERT.=alternobaric vertigo				

There is also the barotrauma of ascent, or "reverse squeeze," which occurs with eustachian tube blockage on ascent. It usually occurs in the diver who has used pseudoephedrine, and the effect has worn off. The symptoms are ear fullness, slight vertigo, with minimal pain. The problem resolves quickly on proper equalization but can last for several minutes after leaving the water, depending on the amount of edema caused by the barotrauma.

How do you differentiate MEBt from IEDCS? Well, the dive is shallow because of the lack of ear clearing. Symptoms start almost immediately under water. The tympanic membranes have suffered barotrauma. There is no nystagmus, the Tullio, Hennebert and head impulse tests are negative. There is pain or significant discomfort, which does accompany IEBt but not IEDCS.

The differentiation between MEBt and IEBt is the nature of hearing loss which in MEBt is conductive while in IEBt and IEDCS it is sensorineural. Proper tuning fork testing is critical!

Alternobaric vertigo occurs due to eustachian tube dysfunction as well. Different middle ear pressures exist when there is a one-sided blockage. This unequal pressure is transmitted to the vestibular system, causing rotatory vertigo. It doesn't take any more than 2 fsw (60 cms) of pressure differential to cause these symptoms. They tend to occur closer to the surface but can occur at any depth. Symptoms occur on ascent with a feeling of asymmetrical pressure in one ear. There is no hearing loss and no tinnitus. Symptoms last a few seconds to 10 minutes but may last much longer if there is also a URI present.

How do you differentiate alternobaric vertigo from IEDCS and IEBt? The symptoms occur on ascent and are short-lived. The ear-clearing attempts occur before or after the onset of vertigo. Interestingly, there is often a history of otitic barotrauma in the past. There is no pain and no hearing loss.

Table 2 helps to differentiate some of the sources of vertigo appearing during or after a dive.

As an aside, it is important to mention the dive position that helps to minimize vertigo under water. This position keeps the horizontal semicircular canal in the horizontal plane with the body inclined forward 60° to the horizontal sea floor. The most stimulating position is the body facing down 30° from the horizontal.

I have to mention that the long-expected FDA approved Balloon Dilatation of the Eustachian tube is the first successful surgical treatment for eustachian tube dysfunction. This treatment will hopefully allow more divers to dive without suffering ear problems. There are no peer-reviewed long-term reports on divers yet, but from personal experience, it has the potential to help solve this problem. Stay tuned!

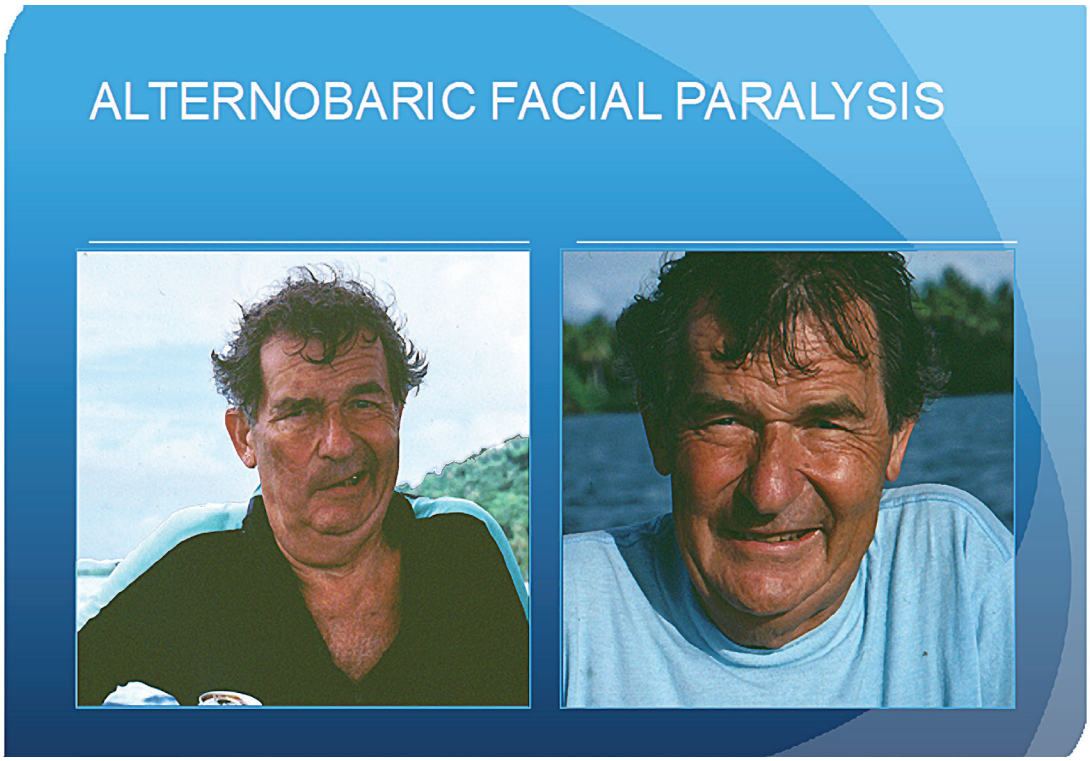

Figure 2. Right-sided alternobaric peripheral facial nerve paralysis, clearing after 2 hours (right)

There are many other sources of vertigo that can occur in divers. Those patients with **significant exostoses** can suffer unequal caloric stimulation of the outer ear canal, especially when wearing hoods. The vertigo is momentary. **Carbon monoxide contaminated breathing gas** can cause vertigo and other symptoms leading to death. **The Blue Orb syndrome** is an agoraphobic reaction that occurs in divers when there are no visible underwater points of reference, causing panic and vertigo. **Mal de Debarquement** can cause rocking or swaying vertigo on land after leaving the boat.

Alternobaric facial nerve paralysis can develop from middle ear barotrauma. The facial nerve courses through the middle ear and is sometimes dehiscent, not covered with bone. The negative pressure then causes neurapraxia by interfering with venous return (Fig 2). It is usually immediately evident on ascent, with the peripheral pattern of paralysis differentiating it from a DCI problem. There may be an associated alternobaric vertigo, but mild. Because the facial nerve carries taste fibers to the anterior 2/3 of the tongue, the taste may be affected. The paralysis lasts for a few minutes to hours and clears spontaneously. There are no sequelae. Differentiating it from DCI is that it is peripheral-type paralysis, and although there may be slight vertigo, hearing loss or tinnitus from the middle ear barotrauma, they are short-lived. The facial nerve paralysis lasts a few hours and clears spontaneously. Incidentally, because of the bony defect, pressurized air can travel along the tegmen, reaching the CNS, possibly causing pneumocephalus.

Paranasal Sinuses

We will transition to the sinuses. We are concerned with how barotrauma to the sinuses can mimic DCI. The outer bony walls are resistant to the pressure change; the inner thin bony walls are not. The epithelium is virtually the same in the nose and the sinuses. Sinus barotrauma occurs when there is blockage of the communication from the sinuses through the natural ostea to the nose. This communication usually allows small volume changes to occur within the sinus. They can be partially blocked with edema from a URI, allergy, chronic sinusitis (16% of the US population) and from abnormal sinus anatomy that partially blocks sinus drainage.

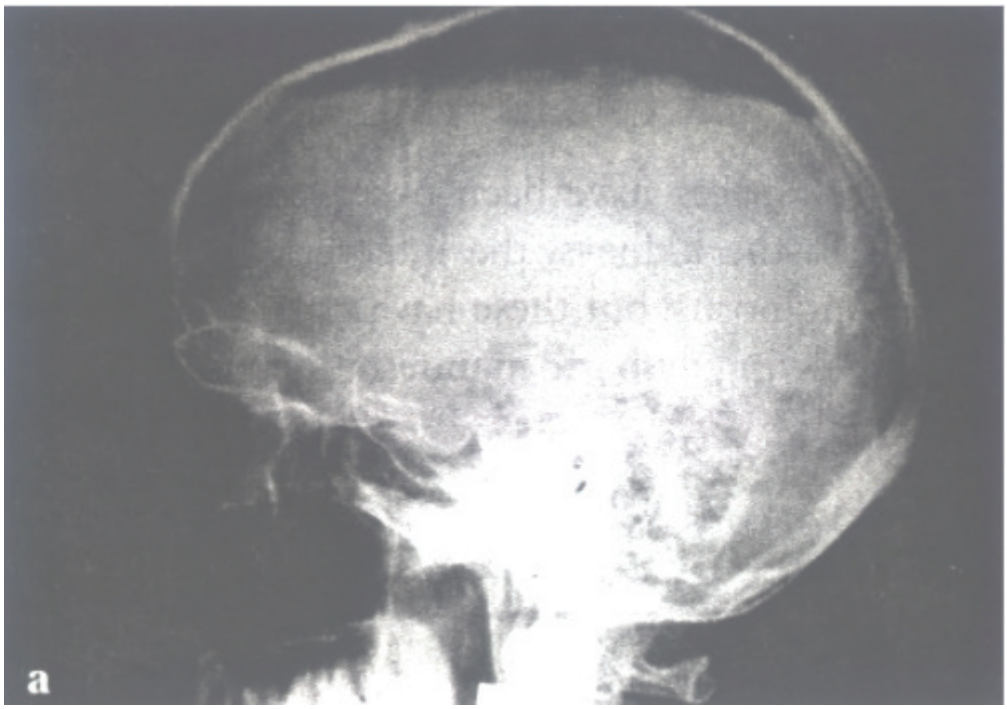

Figure 3. Pneumocephalus

The clinical picture is one of pain over the frontals (68%), ethmoids (16%), maxillaries (8%) and sphenoids (6%). There is epistaxis in 58% of the divers. Symptoms can occur on descent (usually) or ascent. Radiology demonstrates fluid in the affected sinus and a possible anatomic abnormality on CT.

When the maxillary sinus is involved, there can be the development of paresthesia of the second division of the V nerve, causing anesthesia of the cheek skin, ipsilateral lip, and gingival mucosa. There is a 2% incidence of dehiscence of the bony covering over the nerve within the roof of the sinus, so when there is severe negative pressure within the sinus, it can stop the perineural venous return, and create a barotraumatic nerve paresis. Pressurized air can travel perineurally, causing crepitus in the cheek skin. Dental barotrauma must be ruled out. The symptoms resolve quickly without treatment. If the symptoms recur, I've had success eliminating the problem with an office balloon sinuplasty treatment to the maxillary sinus ostia, which generally has a sinus drainage malformation.

The sphenoid sinus is often titled the "silent sinus" because symptoms affecting it are not very specific: generalized vertex or occipital headache and few if any nasal symptoms (drainage is directly into the nasopharynx). The thin walls of the sinus are bordered by significant structures: the internal carotid, optic nerves, and the cavernous sinus. The most common problem leading to sphenoid sinus barotrauma are nasal polyps blocking its drainage. Unfortunately, they may be asymptomatic otherwise. The symptoms mentioned above occur on ascent or descent and can be made worse by a Valsalva. A generalized headache can persist for days after the dive. As with other sinus and ear problems, the precipitating problems are the same: URI, chronic allergy sinusitis, etc. As with middle ear barotrauma, the pressure differentials needed to cause the problem can be as little as 380 mm Hg. The physical examination may be perfectly benign, with possibly only minor bleeding being seen in the oropharynx. Nasal endoscopy by an Otolaryngologist is usually diagnostic.

Unfortunately, the over-pressurization may not be benign. It can result in an acute, persistent headache, only diagnosed if a CT is obtained. If there is bony dehiscence, pneumocephalus may be the result. Disruption of the optic pathways has resulted in blindness. Because symptoms mimic a CNS problem,

imaging studies will be ordered, making the diagnosis. Treatment to relieve the pressure is surgical.

We have discussed **pneumocephalus** (Fig 3). It can develop through tegmen defects, through the superior canal dehiscent syndrome, by air coursing through the facial nerve canal or from sphenoid barotrauma.

The differential diagnosis of sinus problems with DCI revolves around pinpointing the sinus related neurologic signs versus those related to general DCI such as other cranial neuropathies and optic neuropathy. If there is facial CN V paresthesia form maxillary sinus barotrauma, the factors that help differentiate are shallow depth of the dive, history of nasal congestion with allergy, URI or sinusitis, and rapid clearing with O2 administration. The complications of sphenoid sinus barotrauma are differentiated with endoscopy if that is available, and imaging.

Other

Finally, **mask barotrauma** can lead to an orbital hematoma which, in turn, can lead to diplopia in a specific gaze direction, depending on the location of the hematoma. This pseudo-neurologic finding will be obvious because of the periocular ecchymoses and the history of difficulty with mask clearing.

Where do we stand with our initial case? Additional history reveals that she expended a significant amount of energy at depth trying to bring up the sample of coral reef. The dive itself was supervised by the university dive program and was designed to be a reverse profile on purpose. The diver adhered to planned surface intervals, decompression stops and dive times. Further general neurologic examination was negative. Recall that her Weber test lateralized to the left, and air conduction was greater than bone conduction bilaterally, signifying that her hearing loss was sensorineural in type. To make sure this was not a CNS problem, the HINTS exam was performed. She had right-beating unidirectional nystagmus; the head impulse test was positive to the right, that is there was catch-up nystagmus when the head was rotated rapidly from the right to center. There was no skew deviation on testing either eye. A pneumatic otoscope was used to pressurize each ear, watching for eye deviation. The Tullio phenomenon was negative. It was concluded that she had IEDCS, and so recompression was carried out (without performing myringotomies) with fortunately an excellent result. Incidentally, a clinically significant PFO was found on subsequent workup.

Wallenberg Syndrome: an infarction or stroke in lateral medulla. Symptoms include hoarseness, nausea, vomiting, hiccups, rapid eye movements, or nystagmus, a decrease in sweating, problems with body temperature sensation, dizziness, difficulty walking, difficulty maintaining balance

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Pain, Aches and Trauma

Jim Chimiak, MD

Pain is one of the primary reasons that a patient seeks medical attention in general, and it is one of the most common manifestations of diving injury. Pain warns a patient of a pathologic process and often promotes action to correct the problem. These actions can include ceasing further activity, first aid, seeking medical attention, etc. Acute pain occurring after a dive should be taken seriously and prompt medical evaluation. There are many possible causes of pain after a dive with decompression sickness (DCS) always part of the differential.

Figure 1. Descartes pain model

Physiology of Pain

Pain is defined as an unpleasant sensory or emotional experience associated with actual or potential tissue damage or described in terms of such damage. Much has been written recently on both chronic and acute pain. Our discussion will focus on acute pain as it relates to decompression illness. The acute pain that follows a dive can give us useful information to determine a diver's injury. Chronic pain is distinctly different from acute pain. The pain itself may no longer signal tissue injury and may be impacted by an array of factors. Chronic pain needs to be included in the differential just like any other medical condition a diver may have before entering the water.

Pain Perception

The perception of pain has gone through several iterations throughout history. Descartes in 1664 developed an interesting hard-wired model of pain where the painful inciting stimulus, heat particles, stimulated animal spirits that turned a physiologic valve in the body that induced a pain withdrawal reaction (flinch). The pain was the physiologic switch that energized a hydraulic system that drove a response.

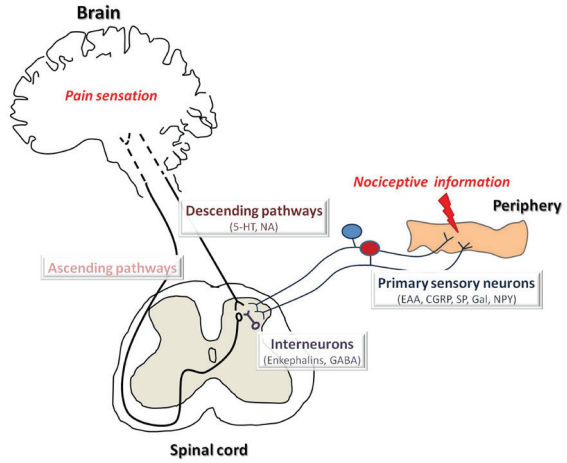

Figure 2. Nociceptive neural transmission

This simple model was replaced as the knowledge of the nervous system advanced. This understanding continues to grow as new pain transmitters, pain receptors, nervous system pathways, and cortical processing is better understood. The simple flinch outlined by Descartes has been replaced by the discovery of these new developments:

Joint pain

Pain in the major joints and joints surroundings is seen with pain only decompression sickness historically called "bends". The joints are richly innervated and have several types of nerves that transmit pain signals from various structures that make up the joint except cartilage.[1] The nerve type involved is important and affects how the patient perceives and reports the pain. The myelinated A-delta and unmyelinated C fibers transmit specific pain signals from the joint. Other fibers include A-beta fibers that are equipped with the corpuscular endings in addition to post ganglionic sympathetic C fibers that can play a role in pain transmission as well as vascular changes to the involved area. Vague non-dermatomal, somewhat confusing complaints by the patient also may result.

An injured joint can release inflammatory mediators and cytokines such as interleukin-6, prostaglandin E2, bradykinin, TNF alpha, etc. that lower the pain threshold by rendering voltage-gated ion channels more excitable. Conversely, there are inhibitory receptors that raise pain thresholds such as the mu opioid receptor.

The peripheral pain signal is sent and then impacts a synapse in the spinal cord which also may take input from the other joints, muscle and skin hence a referred pattern of pain may result in which the joint may be injured yet the patient may report confusing symptoms involving these other normal structures. It is prudent to understand these referral patterns.

After both inhibitory and excitatory modification in the spinal cord, the pain signal is transmitted to the brain where awareness occurs. The pain is modified by structures such as the amygdala which has been implicated in the role of fear as well as descending inhibitory signals. These inhibitory signals are initially effective and can fatigue with time.[2]

Central sensitization is a process where large areas of the spinal cord are recruited, and a variety of pathologic pain responses may occur. This process may create counterproductive responses and even result in chronic pain syndromes. These areas are often more sensitive to normal painful input.

The new models help show the functions experts believe involved in pain:

- Transduction: primary afferents
- Transmission: synapse periphery, spinal cord, brain
- Modulation: signal modified, dampened or enhanced
- The perception: subjective sensation of pain

This model was enhanced as mechanisms were discovered to explain:

- Stress-induced analgesia
- Central sensitization, wind-up

Also, pain is characterized as somatic, visceral, neuropathic, psychosomatic and chronic. The expression of pain may be affected by a variety of factors including damaged tissue, innervation of that tissue, cultural issues, awareness of pathology, psychologic factors, etc. To enhance reporting, constructs such as pain scores recorded as the 5th vital sign, have been proposed. Despite the scientific advancement, understanding and treating pain remains a challenge today and continues

Figure 3. Trace amounts oxycodone in the tissues of bay mussels (Mytilus trossulus) Washington state's Puget Sound Researchers at the Puget Sound Institute PSI research scientist C. Andrew James, PhD presented the findings at the 2018 Salish Sea Ecosystem Conference

to humble those in the field using current thought. One has only to look at the prescription opioid problem to see how a well-intentioned program of liberal opioid prescription to treat pain proved to be marginally effective in treating pain while contributing to a deadly epidemic. The prescription opioid oxycodone is so widely prescribed that it has been found in shellfish in Puget Sound.

Effects of diving on the perception of pain

The perception of pain can be profoundly altered while diving. The discovery of significant injuries incurred while diving and only discovered post dive are not uncommon. There are some identifiable reasons for this:

- cold, vasoconstriction: cooling has long been known to decrease acute pain transmission, cool sprays are now widely used before IV placement to lessen the pain of insertion
- distraction: methods to distract the processing of pain can be effective such as meditation
- endorphin: elevation of levels because of the diving experience
- adrenalin: significant wounding may not be noticed during periods of high stress/activity
- nitrogen narcosis: narcotic effects of nitrogen can impact pain perception

A decreased pain perception, therefore, has been postulated and then demonstrated by Kroener.[3] Divers were taken depth and pain responses indeed were changed with both the amount of pressure needed to obtain a response as well as pain perception to the same stimulus between surface and diving exposure.

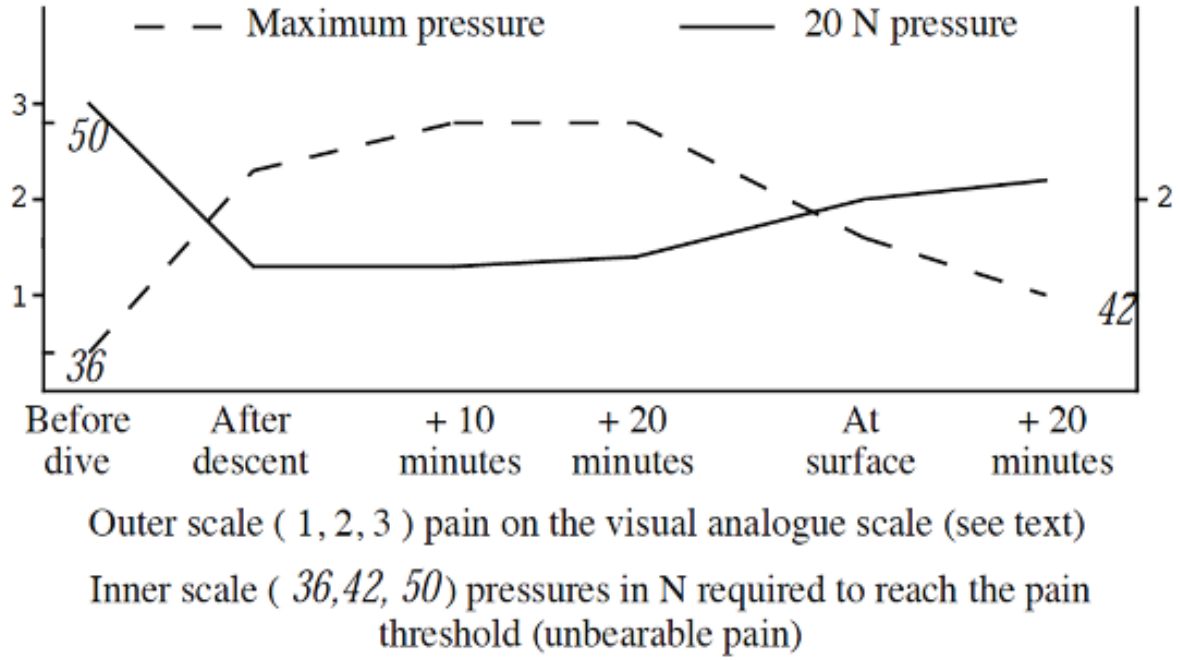

Figure 4. Pain Perception is reduced during scuba diving[3]

Pain in Diving Injury

Pain is one of the most common symptoms of dive-related injuries. It occurs with ear and sinus barotrauma, pulmonary barotrauma, decompression sickness and various decompression unrelated injuries. In decompression illness, pain may affect major joints, large muscle groups, abdomen or headache. The most common is joint pain. Pain is not specific to DCI. Differential diagnosis includes trauma, infection, overuse conditions, degenerative process, and other.

The pain only decompression sickness (DCS Type 1)
The joint pain after dive, affecting usually single major joint, with a thorough neurological examination normal, is classified as pain-only, mild DCS (Type 1 DCS). It involves bubble induced injury of the joint tissue and possibly surrounding tissues. This delineation can become difficult especially with pain in symmetric joints which may be a result of CNS involvement, or in case of hip pain that may be difficult to discern from truncal or abdominal pain. In the case of the decompression unrelated pathology at the spine affecting a nerve root, radicular pain can encompass the location of an unaffected joint enervated by that nerve.

The joint pain associated with decompression sickness can range from barely perceptible to severe. The pain has been observed in some extreme cases to be debilitating. The pain usually appears within hours of surfacing. Pain that occurs while still decompressing may announce a more concerning DCS.

Interestingly, there is no agreement as to the etiology of the pain in bends. It would seem evident that one merely needs to demonstrate a bubble at the site of pain. But this has not proven successful. Bubbles have been readily seen in asymptomatic joints and no bubbles in painful ones. Several sites where the pain may be generated include:[4]

- Marrow space
- Joint space/tribonucleation

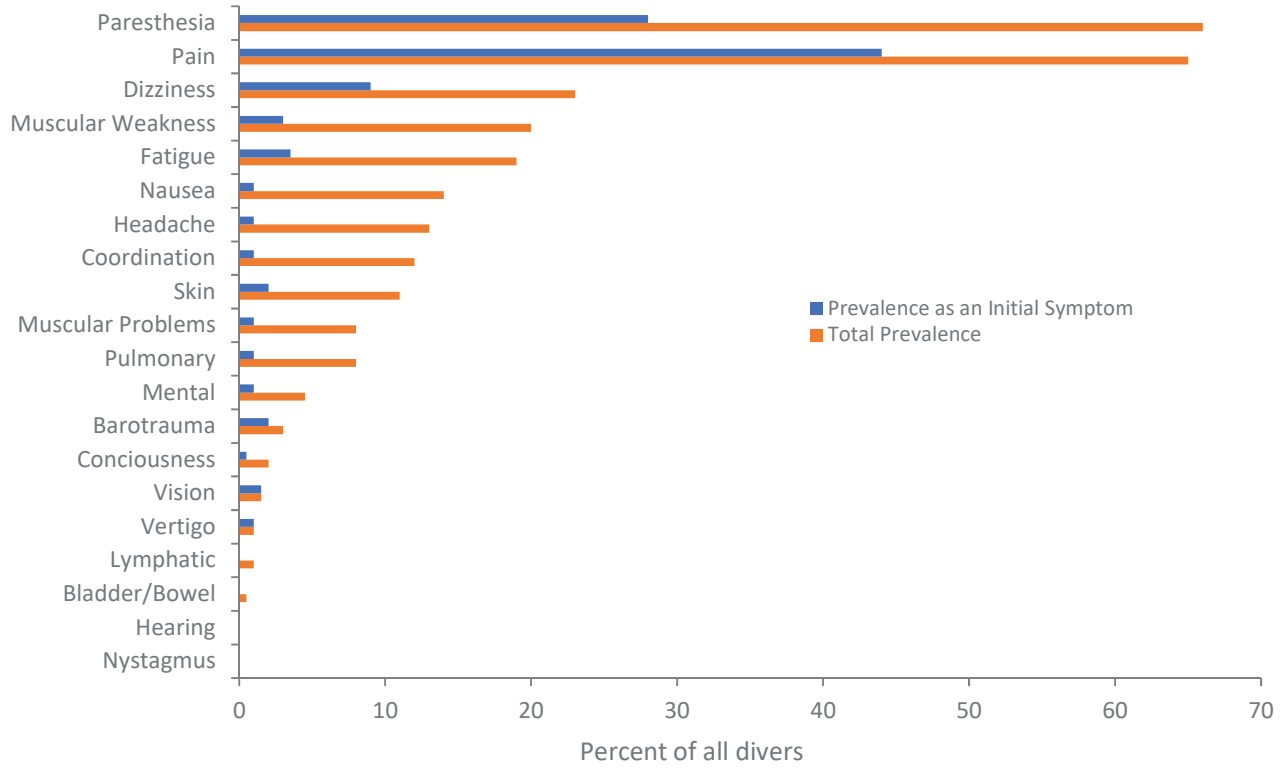

Figure 5. Symptoms of decompression sickness in recreational divers indicating prevalence as an initial symptom and the total prevalence.

- Fascial planes
- Soft tissue, muscle
- A distraction of connective tissue
- Periosteum
- Ligaments, tendon
- Probably not cartilage
- Referred- spinal cord, nerve sheath or plexus
- Bubble related inflammation

An interesting theory for a cause of joint pain in DCS was introduced by Straus[5]. The type 2 Ruffini corpuscle was proposed as a very plausible candidate for both the source and the actual transducer necessary for pain transmission. The Ruffini capsule is a unique organelle that richly enervates joints throughout the body. It is particularly sensitive to stretch and accounts for the severe pain one experiences when a joint is stretched abnormally. This pain serves a crucial purpose to effectively warn and prevent abnormal joint stretch that would lead to serious injury to the joint, sprain/strains to dislocations.

There is animal evidence utilizing experimental, severe dive profiles that produced DCS may be associated with medullary bubble formation and subsequent dysbaric osteonecrosis (DON). These findings have not been reproduced in recreational divers other than suspected anecdotal cases.

If recompressed soon after onset, Type 1 DCS joint pain can respond dramatically within minutes of recompression. The use of shorter therapeutic recompression tables can be chosen and followed based on the time it requires to achieve complete resolution. When following the prescribed algorithm, shorter hyperbaric treatment tables can be as effective as longer ones.[6]

Relevant non-diving related painful condition

There are several common issues that may contribute or even cause the development of pain for the traveling diver. They include: transportation to the dive site/ gear toting (strain from carrying heavy equipment), sleep deprivation, dehydration, GI disturbance, drugs, alcohol, jetlag, lack of fitness, multitasking (distracted), Internet searching (sensational medical info), globetrotting

Figure 6. Sedentary office worker scurrying during the one week he has scheduled from his job to move self and gear to a vessel on the other side of the world, complete all 20+ dives promised and fly back.

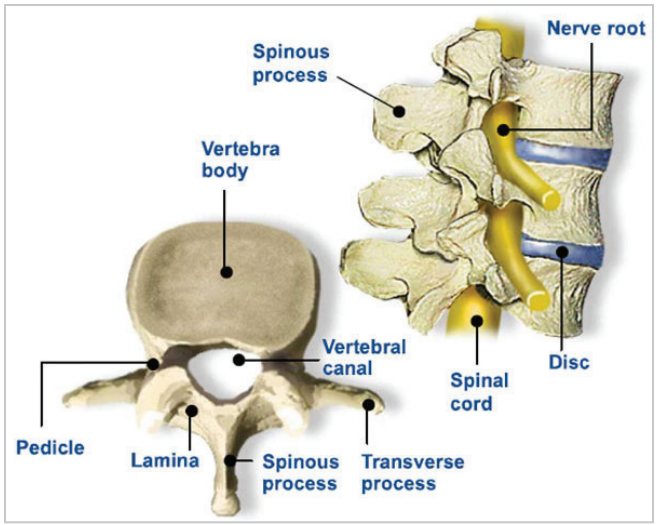

Figure 7. Vertebral column demonstrating relationship of intervertebral disc and neural foramina with the exiting nerve root.

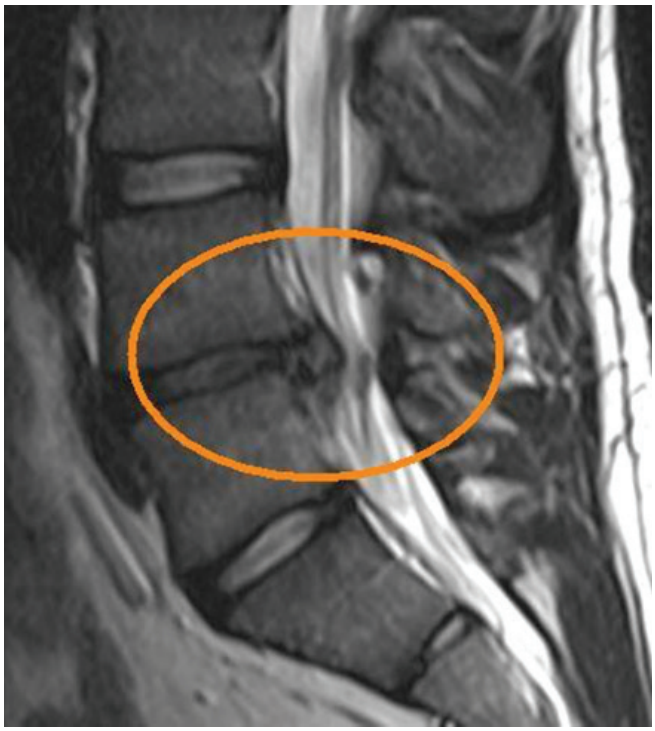

Figure 8. Imaging of disc herniation.

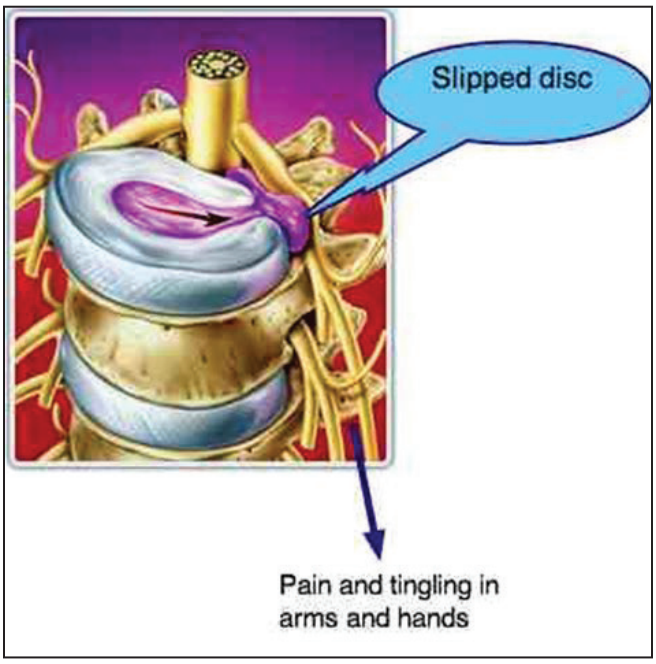

Figure 9. Herniated disc impacting spinal nerve root and resulting in radicular pain along its distribution that could include shoulder, elbow, hand, hip, knee, ankle or foot depending on location of herniation.

(illness), altitude, gripping ascent line or gear, constrictive suit, fins, cold (mononeuropathies, TMJ (cold, mouthpiece). Specific pain conditions include:

Cervical or Lumbar spine pathology

An interesting study of over 160 experienced scuba divers with a mean age of 35 reported that over 50% incidence of low back pain (LBP) mostly confined to back (axial) with almost a quarter reporting radiating symptom. Six percent required surgery or intervention with an average of 4 months before diving. Over 50% faulted the dive equipment and almost all desired formal education on preventing LBP problems.[7]

Referred pain

Referred pain appears to come from tissues in one area, but the pathological process is occurring in another. Spine injury can affect the spinal nerves that enervate specific areas of the body. If a mass impacts a spinal nerve, pain may be sensed in a distal extremity in a radicular fashion. Various organ pathologies have very distinct referral patterns such as the shoulder pain experience with the gall bladder or the periumbilical pain associated with appendicitis. Hip fractures have been missed because of pain complaints to the knee.

Pain Interference with neurologic examination

Pain can be of such severity that a give-way may be when muscle strength is tested. This weakness may not be secondary to a neurologic deficit but rather a pain. Also, pain can interfere with the conduct of a thorough neurologic exam. Guarding may prevent detection of abnormalities necessary to rule out serious decompression illness.

Other common conditions include patella-femoral syndrome, overuse, sprains, strains, toxins such as ciguatera, connective tissue disease, panic (internet), hyperventilation and infectious disease (Lyme's, dengue, chikungunya, flu etc.).

Just because an individual has a pain condition does not prevent them from also being experiencing DCS. Having a traumatic painful injury or recovery from surgical procedures can theoretically increase the chance of also experiencing DCS given the altered vascularity affecting

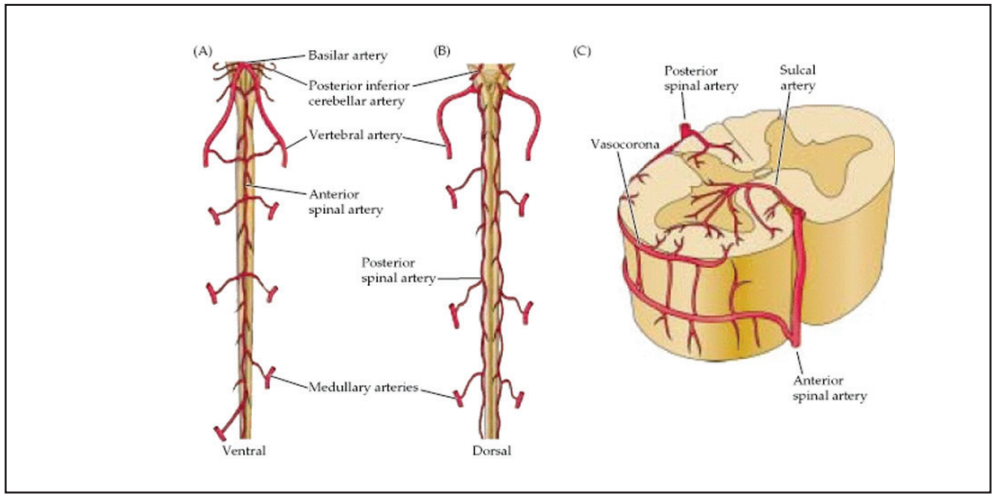

Figure 10. Blood supply to the spinal cord. Note supply to both white and grey matter in a cross-sectional view.

the uptake and elimination of inert gas to those tissues. An experienced orthoped and dive medicine physician treated a case of type 1 DCS occurring in conjunction with a post-dive injury. The onset of pain-only DCS occurred three hours after a dive just at the time he also strained his knee horseback riding. The deep boring knee joint pain quickly resolved with recompression while the pain from his traumatic injury to his knee's collateral ligament showed little improvement.[8]

This concern was evident when an experienced team of diving medical physicians faced an acute herniation of the L5-S1 disc that occurred during an experimental saturation dive while the subject was performing his exercise protocol. His pain required rest, morphine, anti-inflammatory medication and diazepam to control. His physicians were aware that the inflamed tissue could also develop DCS and prompted a 12-hour hold of decompression at that depth and an elevation of the ppO2 to 0.5%. His pain improved over the 60 hours of required decompression. He was managed conservatively, and he recovered after six weeks.[9]

Figure 11. Lightning can strike twice

Reporting the pain: The caller who is reporting the onset of pain following a dive should be asked:

- Recent dive history, time of pain onset
- Any other associated symptoms such as shortness of breath, illness mental status change, numbness, weakness, gait problems, rash
- Location of pain such as joint, bone or surround soft tissue
- Characteristic of the pain, deep dull joint; intensity using a scale
- Multiple or single location
- If multiple, is it symmetric that may be associated with a spinal lesion
- Is pain affecting limb only or spine, pelvis, hips involved
- Is it affected by movement, palpation, position

- Any discoloration, warmth, streaking
- History of joint problems, or associated medical conditions
- Recent illness or injury

Diagnostics

Joint or muscles pain and normal neurological exam are the requirements for Pain Only Type 1 DCS. The pain in Type 1 DCS is classically described as a deep boring pain. It often has no palpable point of tenderness. The diver often can find neither a position that alleviates or worsens the pain. The pain can be quite severe. There can be more subtle forms of pain only DCS. It has been called **niggles** with an attempt to define it as transient pain lasting no more than 30 minutes in one joint and one hour in multiple joints. It generally does not warrant recompression. Researchers have even given a fractional consideration as DCS in manned decompression table testing.[10]

Differentiating the cause of the pain from non-diving injuries is the task confronting the diving medical physician. The history and physical remains the standard of care in the diagnosis of DCS. The neurologic exam is the most important part of the evaluation and diagnosis of decompression sickness. In some cases, the joint pain is associated with and may precede neurological manifestations. The neurologic deficits may not be present at the time of the initial exam and serial neurologic exams should be conducted.

When confronted with a diver whose presentation is atypical, several controversial measures have been advocated to assist in making the differential:

Cuff test: This is a test where a blood pressure cuff is applied to the painful joint and joint compressed. If the pain is relieved, it was once thought that it was diagnostic for type 1 DCS. However, this maneuver has shortfalls. It may provide support to a traumatized area and give relief like wrapping tendonitis or a sprain that brings relief and even function back to that joint. The diagnostic value of the cuff test was evaluated in a study with 179 patients with type 1 DCS. The cuff test was only positive in 61% of those cases.[11] A tight cuff may also have an effect and a false positive response solicited by the diver.

Test of pressure: Some physicians use this test when a diver with a painful joint has presented with a confusing picture where DCS is questionable. The diver is recompressed in a hyperbaric chamber and administered hyperbaric oxygen. A quick resolution of pain under pressure is expected if the recompression followed soon

RICE AND HBOT

RICE	HBOT
• Rest - recline	• Rest/ recline for several hours
• Ice - vasoconstriction Reduce inflammation	• Vasoconstriction • Reduce inflammation
• Compression - reduce swelling and edema	• Reduces edema
• Elevation - reduce edema, improve oxygenation	• Improves tissue oxygenation

Consider any period of rest therapeutic "tincture of time"

enough after the pain onset. If the pain is slowly or only partially relieved, it may be due to the time and oxygen effects on traumatized tissue, and not due to squeezing bubbles. There may be some similarities between RICE therapy used for sprains/strains and hyperbaric oxygen therapy. The pain relief may not necessarily indicate the injury was DCS.

Also, consider the psychologic effect of these tests in someone who may believe they have DCS. Since there are no imaging studies or blood tests to definitively diagnose DCS, this test may yield faulty data in the anxious patient, it is unblinded, and the diagnostic purpose is usually articulated clearly to the patient. No one would look for Waddell signs in this fashion to evaluate low back pain.

The diagnosis of DCS is made by first taking a careful diving history. The exposure data should include the depth/time, number of dives, surface interval, days of diving, type of dive, gas and decompression measure, the time of onset of the first symptoms a as well as the time of subsequent symptoms or their change. Dive history should include any altitude exposure after dive including plane, car or hiking. A thorough review of the past medical history should include recent injuries, exertion, age, medical conditions that include painful syndromes, neurologic or spinal pathology. Serial neurologic examinations are required to uncover type 2 DCS that may start with pain only symptoms.

Suspicion of other etiologies should be entertained before reflexively recompressing the diver for any pain complaint.

A diver with severe abdominal pain and difficulty raising his right leg with paresthesia after four deep air dives were recompressed with slow resolution of his leg complaints, but abdominal pain persisted. A perforated appendix was discovered after his chamber treatment and treated with surgery/antibiotics with full recovery.[12]

Caution with exercise-induced left shoulder pain or recent weeklong right sided stinging sensations. Avoid diverting a diver hastily to a recompression chamber before exploring indications for evacuation to a cardiac cath lab or for thrombolysis., Review of a diver's medical history in conjunction with his recent dive physical will help reduce the differential. A diver may have been cleared with a painful condition that may have been aggravated during the dive trip and has now been brought to medical attention.

Treatment of type 1, pain only DCS

"If there is any doubt as to the cause of the pain, assume the diver is suffering from decompression sickness and treat accordingly. Any decompression sickness that occurs must be treated by recompression."[13]

This has long been the basis of treating suspected DCS cases when the population is young healthy divers. Treatment success reinforced this strategy. Simply recompressing everyone who has pain is not good practice and certainly reinforces the saying when all you have is a hammer, everything looks like a nail. Ruling out DCS and finding the correct etiology is important especially if other treatments are indicated. First aid should include surface level oxygen, hydration, and serial neurologic exams. Differentiating type 1 from type 2 may seem unimportant if treatment table 6 was the only definitive treatment. But utilizing a much shorter USN treatment table 5 or not treating at all are two acceptable options under certain conditions. A study showed for DCS Type 1 there was no difference in good outcomes utilizing treatment table 5 early and strictly following the guidelines for its use. (reference)

New Guidelines for the Management of Mild DCS in Remote Locations

The correct differentiation between mild or type 1 DCS and type 2 DCS becomes even more important when it occurs in a remote location where there is no hyperbaric chamber and evacuation may not be possible or may even be hazardous. Experts have agreed that in such cases and under certain conditions the recompression may not be required for pain only, mild DCS type 1 which has been stable or improving for 24 hours. The diver must also have normal serial neurologic examinations to meet the stringent requirements of this guideline. There is no further diving until the return home and approval by a diving medical physician.[14,15]

Case presentation: A new diver presents for evaluation following his dive that morning. He complains of headache, fatigue, knee pain, and rash. He is bent over from abdominal pain. You place him on oxygen and recall your chamber personnel to line up the chamber while you complete the exam. You discover he has made one uneventful dive to 45 fsw for 35 minutes earlier that day with no impact on his multiple joint pain. His neurological examination is completely normal. You noticed his eyes are red and a trace of nosebleed remain about his nares. He states he has no medical problems but just remembered he had a fever three days ago that has improved. He states his abdominal pain and multiple joints have been aching since yesterday. He relays that he and his friends have been camping on the beach over the last week. A repeat neurologic exam again is normal. He was found to have contracted Chikungunya by history of his multiple arthralgias that occurred after his febrile episode following his environmental exposure in an endemic region. Abdominal pain, conjunctivitis and low platelet count were also consistent with the diagnosis that prompted further positive serologic testing. Recompression was not indicated.

This case was given as an example to highlight the diagnostic difficulties the diving medical physician will confront. The luxury of knowing your divers who you know are healthy and fit is not the usual scenario. Your first encounter with the diver is often his initial presentation after diving. Medical colleagues may defer to you without complete workup of other medical conditions besides DCS. Evacuation to the nearest recompression chamber may seem reasonable and innocuous but the dangers have been well described elsewhere: the delay of necessary treatment, transportation hazards, harmful altitude exposure, etc. Conversely, prompt recognition of DCS is important. Properly trained emergency responders are key to identify and expedite those patients through the emergency system. The local diving medical physician plays a key role not only for emergent consultation but for ongoing training for everyone involved from dive site to the emergency room. Those unstable, with shortness of breath, altered mental status or paralysis are not easily missed but the following presentations should also raise the level of urgency:

- Bilateral, symmetric pain: may signify a serious, central lesion of the nervous system
- The onset of joint pain while conducting decompression/ascent may warn of worsening problems when brought aboard. Treat with urgency and conduct serial neurologic exams

A-alpha nerve fibers carry information related to proprioception (muscle sense).
A-beta nerve fibers carry information related to touch.
A-delta nerve fibers carry information related to pain and temperature.
C-nerve fibers carry information related to pain, temperature and itch.

- Rapid onset and worsening pain on surfacing: early time of onset, progressing symptoms may indicate an unstable condition that would prompt more frequent repeat neurologic examinations should more serious forms of DCS evolve.
- Abdominal or truncal pain: can be associated with tingling or even mild weakness of legs which can gradually ascend and progress in severity (often incorrectly ascribed to overexertion by the diver)

Conclusion

Pain is an important warning of a pathologic process. It is complex, and its signal is impacted by many factors before it is registered by the individual. Unfortunately, the pain is generally insufficient to define a definitive etiology without a detailed history and physical investigation. The onset time, characteristic of pain, location, evolution, possible other symptoms and signs, and detailed diving history helps make the diagnosis of DCS.

In conclusion, the diagnosis of pain only DCS can be established after thorough neurological examination with no abnormal findings and after exclusion of other possible causes. Recompression may be indicated once the diagnosis of DCS is made.

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Differential Diagnosis of Decompression Illness

Differential Diagnosis of Decompression Illness (Massey and Moon 2014b) caused by bubble formation within tissues due to inert gas supersaturation. Arterial gas embolism (AGE) (Vann et al. 2011)	
Conditions	**Description**
Thromboembolic or hemorrhagic stroke (Kohshi et al. 2017), (Bartsch et al. 2009), (Buttinelli, Beccia, and Argentino 2002), (E Gempp et al. 2014), (Hayden, Buford, and Castillo 2015)	Age and risk factors help differentiate. MRI will reveal hemorrhage or ischemia. If doubt exists, recompression therapy will not worsen outcome after stroke. Guidelines for the early management of patients with stroke were just revised.
Carotid or vertebral artery dissection (Brajkovic et al. 2013), (Kasravi et al. 2010), (Alonso Formento et al. 2016), (Nelson 1995), (Konno et al. 2001), (Gibbs 3rd, Piantadosi, and Massey 2002), (Skurnik and Sthoeger 2005), (Bartsch et al. 2009), (Kocyigit et al. 2010)	Neck pain is usual.
Multiple sclerosis (MS) (Jan and Jankosky 2003)	Vertigo, visual loss, focal sensory, motor or cerebellar symptoms due to MS may mimic decompression illness when temporally related to a dive. Heat stress in tropical conditions may exacerbate symptoms in demyelinating diseases. MRI, CSF evaluation may help.
Seizures	Spontaneous seizures immediately after a dive may be difficult to differentiate from arterial gas embolism.

Migraine (Engel et al., 1944; Ferris et al., 1951; Flinn and Womack, 1963; Lieppman, 1981; Ostachowicz, 1987; Butler, 1991)	Visual manifestations, including scintillating scotomata, have been described in altitude-related DCS. Migraine history may help differentiate from decompression
Spinal cord compression/myelopathy (Emmanuel Gempp et al. 2008), (E Gempp et al. 2013) Spinal cord lesions and the development of spinal cord decompression sickness (DCS) (Jager et al. 2002)	Hemorrhage, disc protrusion, or epidural infection. Diagnosis usually confirmed by MRI, which is commonly negative in decompression illness. The spinal fluid analysis may help.
Guillain-Barré syndrome (AIDP)	Sensory symptoms usually minor or absent, and progression usually slower than in decompression illness.
Psychosis (Hopkins and Weaver 2001), (Kenedi, Sames, and Paice 2018)	
Porphyria	History of the disease usually present.
ENT conditions	
Inner ear barotrauma (Farmer, 1977; Money et al., 1985; Shupak et al., 1991; Klingmann et al., 2007)	Inadequate equalization of middle ear pressure during descent can cause rupture of the round or oval windows, resulting in sudden onset of tinnitus, vertigo, and unilateral deafness.
Facial baroparesis (FB) (Molvaer and Eidsvik, 1987; Basnyat, 2001; Grossman et al., 2004) or other facial palsies, (Yetiser 2012)	FB is due to facial nerve compression as a result of inadequate decompression of the middle ear cavity during decompression from a dive or aircraft flight. Differentiated from gas embolism as it presents with both upper and lower facial weakness. Manifestations of FB are usually transient,
Paranasal sinus overpressurization (Idicula, 1972; Neuman et al., 1975; Shepherd et al., 1983; Garges, 1985; Murrison et al., 1991)	Compression of the trigeminal nerve within the maxillary sinus due to over-pressurization in the same manner as in facial baroparesis (above).
Cardio-respiratory conditions	
Immersion Pulmonary Edema (Shupak et al., n.d.), (Hampson and Dunford 1997), (Slade et al. 2007), (Fraser et al. 2011) which could serve as a possible preventive strategy for the development of immersion pulmonary edema (IPE) (Peacher et al. 2014), (Edge CJ 1997), (Grünig et al. 2017)	This disorder usually occurs shortly after the start of a dive, while the diver is still at depth, and might be confused with cardiorespiratory decompression sickness since both cause dyspnoea and cough. 78–80 Symptoms of immersion pulmonary edema typically begin during descent or at depth, whereas the onset of cardiorespiratory decompression sickness occurs after the dive.

Water aspiration	Water aspiration could be mistaken for cardiorespiratory decompression sickness. Both cardiorespiratory decompression sickness and water aspiration can cause pulmonary edema, although the diver is usually aware of aspiration.
Ischemic heart disease, pulmonary embolism, cardiac arrhythmias	
Abdominal issues	
Abdominal pain and discomfort (Payor and Tucci 2011), (Goumas et al. 2008), (Lucas 2011)	
Kidney injury (Gleeson et al. 2015), (Hibi et al. 2017), (Meyne KJ, Kahler W, Tillmans F, Werr H, Binder A, Koch A, 2017)	
Seafood toxin ingestion	
Ciguatera poisoning (Bagnis et al., 1979; Eastaugh and Shepherd, 1989; Swift and Swift, 1993)	Due to ingestion of heat-stable toxin in large fish, such as barracuda, grouper, red snapper, amber jack, king fish. Vomiting and diarrhea usually precede paresthesias, altered thermal sensation, occasionally weakness, vertigo, ataxia.
Puffer fish poisoning (Eastaugh and Shepherd, 1989; Mines et al., 1997)	Due to tetrodotoxin. Mild poisoning is similar to ciguatera poisoning; severe poisoning can cause paralysis and death.
Paralytic shellfish poisoning (Eastaugh and Shepherd, 1989; Mines, StahmerShepherd, 1997)	Due to saxitoxin or brevetoxin. Paresthesias, and burning around the lips, tongue and face occur within 30 minutes of ingestion. Ataxia, aphonia and death due to respiratory muscle paralysis have been reported.
Miscellaneous	
Musculoskeletal strains or trauma sustained before, during, or after diving	Time of onset and history of trauma or strain are helpful. Pain due to decompression illness is rarely accompanied by tenderness or position-related or motion-related exacerbation physical examination.
Infections (Rudge 1991)	

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