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ASSESSMENT OF PATIENTS WITH DECOMPRESSION ILLNESS

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Key Words
Accidents, decompression illness, investigations, treatment.

History

The diagnosis of decompression illness (DCI) is made clinically and should be based entirely upon an accurate history and physical examination. Required for the diagnosis are the appropriate circumstances for the condition to occur and signs and symptoms which are consistent with the disease. The patient must have been exposed to a reduction in ambient pressure. In order to experience pulmonary barotrauma and gas embolism a breathhold ascent from 1 metre may be sufficient.[1] For bubbles to form in the body from supersaturated tissues (decompression sickness, DCS or bends) a diver must have been at depth long enough for supersaturation to occur. It was formerly believed, incorrectly, that the disease would never occur in divers whose maximum depth was less than 10 m (33 ft). Symptoms of DCS have been described after a series of breath hold dives.

The history should include the dive profile, rate of ascent, symptom onset time and changes in symptom type or intensity. As an approximate gauge of a diver's inert gas load a dive table can be consulted. Factors which increase the likelihood of bends include missed decompression stops, heavy exertion during the dive, rapid ascent and a previous history of DCI. A series of dives may incrementally augment the inert gas load. A diver's self-reported profile is often inaccurate. If a dive computer was used, some models will permit downloading an objective record of the profile. Unfortunately it is a common belief among physicians not trained in diving medicine is that divers who have stayed within the limits of a computer or table profile will not suffer DCI. This is not true and such physicians must be educated out of their dangerous ways.

For recreational divers reporting DCI of all types to DAN in 1995 the median time of onset was 1 hour, if there had been no altitude exposure. Within 24 hours 95% of all symptoms had become evident.[2] Table 1 has been compiled from the Diver Alert Network (DAN) statistics for 1995 and shows the frequency of presenting symptoms (the first symptom) and of all symptoms, in 590 recreational diving accidents reported to DAN, tabulated by symptoms. There were over three times as many total symptoms as first symptoms, emphasising that many cases have multiple symptoms. The most severe cases presented shortly after surfacing, as reported by Francis and colleagues.[3] They observed that within 10 minutes of surfacing 50% of their divers had developed symptoms and 85% had done so within an hour. Ninety six per cent of those developing cerebral symptoms had them within 60 minutes. Therefore the longer the delay between surfacing and the onset of a symptom, the less likely it is due to DCI. Symptoms beginning after 24 hours following a scuba dive are unlikely to be caused by DCI.

Exposure to even moderate altitudes can precipitate DCI. The most commonly reported altitude exposure is in commercial aircraft (typical cabin altitude 1,500-2,400 m). After a scuba dive the majority of altitude-precipitated bends occur when the flight is within 24 hours of surfacing from a dive.[4] After a saturation dive symptoms may occur during an altitude exposure days after surfacing.[5]

Physical examination

General physical examination should include measurement of pulse and blood pressure and a search for evidence of pulmonary barotrauma (pneumothorax, pneumomediastinum, subcutaneous emphysema) and otic barotrauma (erythema or rupture of the tympanic membrane, blood or fluid in the middle ear). Rarely, in cases of arterial gas embolism, bubbles can be observed in the retinal vessels. In DCI a non-specific skin rash is occasionally seen. Lymphoedema may indicate obstruction of lymphatics by gas. A specific sign of DCI is a marbling of the skin occurring shortly after surfacing, indicating heterogeneous obstruction by bubbles of subdermal blood vessels.

The examination of a patient with pain only bends usually reveals no evidence of joint inflammation and there is rarely pain on movement. A "classic" physical sign is alleviation of pain when a sphygmomanometer is inflated around the affected joint.[6] Similarly, pain in the hips or legs may diminish when the patient stands up, presumably because the resulting increase in local tissue pressure compresses bubbles. These signs are not sufficiently sensitive or specific to exclude the diagnosis of DCI.

Physical examination in suspected DCI should always include a neurological examination. The patterns of abnormality observed in DCI are usually different from those typical of occlusion of major intracranial blood vessels due to intracranial haemorrhage or thromboembolic stroke. In DCI patchy areas of hypaesthesia, isolated urinary sphincter abnormality (usually urinary retention) and ataxia may be the only abnormalities, but these may be missed if only an abbreviated neurological examination is performed. Walking and performing tandem gait (heel-

TABLE 1

FREQUENCY OF SYMPTOMS OF DCI IN 590 RECREATIONAL DIVE ACCIDENTS IN 1995 (MANY WITH MULTIPLE SYMPTOMS SO TOTAL OCCURRRENCES EXCEED 590)

Symptom	Occurrence as a first symptom		Total Occurrences	
	Number	(Percentage)	Number	(Percentage)
Severe neurological				
Unconsciousness	4	0.7	15	2.5
Paralysis	3	0.5	22	3.7
Visual disturbance	5	0.8	39	6.6
Difficulty walking	2	0.3	55	9.3
Semiconsciousness	1	0.2	13	2.2
Bowel control problem	1	0.2	17	2.9
Speech disturbance	-	0.0	16	2.7
Bladder control problem	-	0.0	11	1.9
Convulsion	-	0.0	-	0.0
Total	**16**	**2.7**	**146**	**24.7**
Mild or ambiguous neurological				
Numbness	129	21.9	364	61.7
Dizziness	44	7.5	134	22.7
Decreased skin sensation	1	0.2	39	6.6
Personality change	1	0.2	14	2.4
Reflex change	1	0.2	15	2.5
Weakness	38	6.4	150	25.4
Total	**214**	**36.3**	**340**	**57.6**
Total neurological	**230**	**39.0**	**486**	**82.4**
Pain/skin/nonspecific				
Pain	203	34.4	341	57.8
Extreme fatigue	25	4.2	124	21.0
Headache	35	5.9	146	24.7
Nausea	25	4.2	87	14.7
Itching	21	3.6	53	9.0
Rash	5	0.8	25	4.2
Restlessness	6	1.0	38	6.4
Muscle twitch	5	0.8	33	5.6
Hemoptysis	1	0.2	6	1.0
Total pain/skin/non-specific	**326**	**55.2**	**104**	**17.6**
Ambiguous†				
Hearing loss	3	0.5	6	1.1
Ringing in ears	1	0.2	14	2.4
Cardiorespiratory				
Difficulty breathing	8	1.4	47	8.0
Other††	22	3.6	48	8.1
Total other presentations	**34**	**5.8**	**133**	**22.5**
Total of all recorded occurrences	**590**	**100.0**	**590**	**100.0**

† Could also be due to middle or inner ear barotrauma

†† Includes stiffness (2), hot/cold flushes (2), cramps (1), swelling, (5) pressure sensation (2), lightheaded/confusion (1), fullness (1), muscle ache/soreness (4), bleeding (1), coughing (1), ear blockage (1), erratic heartbeat (1).

to-toe walking) can often reveal abnormalities which are not otherwise apparent. Inability to perform tandem gait forwards and backwards with eyes open or closed on a hard floor while barefoot suggests a neurological abnormality. Inability to stand with eyes closed, arms folded on the chest and one foot in front of the other (sharpened Romberg sign) has been correlated with DCI.[7] A thorough sensory examination may reveal small areas of hypaesthesia. In the absence of a pre-existing problem with micturition, elevated residual bladder volume can be assessed by inserting a urethral catheter after the patient has attempted to void.

Many patients with paraesthesias as the only symptom have no other manifestation of the disease. Therefore absence of neurological signs cannot be used to exclude the diagnosis of DCI.

Diagnostic tests

The plain chest radiograph is the most available and least expensive of all of the various radiographic techniques which have been applied to DCI. Abnormalities on chest radiographs in DCI include pulmonary oedema in cardiorespiratory DCI ("chokes"),[8] focal opacities due to aspiration of water or vomitus, or pulmonary over distension.[9] Discovering these abnormalities rarely changes the management of the patient, however chest x-ray studies may still be useful. However, conditions which can predispose to pulmonary barotrauma, such as bullae,[10] and evidence of barotrauma such as subcutaneous or mediastinal gas may support the diagnosis of gas embolism if neurological symptoms have resolved. The existence of a significant, and increasing, pneumothorax may dictate the insertion of a chest drainage tube before recompression treatment.

Abnormalities have been reported using neuro-imaging techniques such as computed tomography (CT),[11-13] magnetic resonance imaging (MRI)[14,15] single photon emission tomography (SPECT)[16-18] and positron emission tomography (PET).[19] However, their value in the management of DCI has not been demonstrated, except to rule out unrelated conditions such as haemorrhage which require different therapy. Occasionally chest CT can detect predisposing factors to DCI which are not easily detected with plain chest radiography.[10,20]

Abnormal neurophysiological tests such as EEG,[12] brainstem auditory evoked response (BAER) and somatosensory evoked responses (SSEP)[15] have been described in DCI. While they can occasionally be useful in assessing the patient when clinical evaluation is unsatisfactory, they are not sufficiently sensitive to be used routinely. Because audiograms and electronystagmography (ENG)are more accurate than clinical examination, they are extremely useful in following the course of inner ear DCI or barotrauma.[21] Clinical testing of hearing loss by non-specialists, without access to a series of tuning forks with a range of frequencies, is likely to be inaccurate. The nystagmus produced by vestibular injury is characteristically inhibited by visual fixation, but can easily be tracked and quantified with the eyelids closed by ENG.

There are no specific blood markers of DCI, however elevation of serum creatine phosphokinase (CPK) has been described in air embolism,[22] presumably due to muscle injury. Haemoconcentration has been described in both forms of DCI,[23,24] presumably due to endothelial damage and the extravasation of plasma into tissues ("third space" loss). If contaminated breathing gas is suspected because of symptoms or signs of carbon monoxide poisoning, measurement of a blood carboxyhaemoglobin level may confirm the diagnosis. Appropriate blood or urine assays can be used to detect other metabolic causes of encephalopathy such as hypoglycaemia and drug or alcohol intoxication.

Cases of DCI exist in which the only manifestation appears to be a mild encephalopathy observed by the diver, who realises that he is unable to perform a routine, familiar task. Neuropsychological tests have been used to demonstrate abnormalities in DCI which are not apparent after history and physical examination.[25] If trained personnel are available to administer short neuropsychological tests, they could also be useful in following the course of treatment. Additional investigation is needed to determine the appropriate tests to use and their role in the management of DCI.[26]

Differential diagnosis

Onset of pain, rash, dyspnoea or a neurological abnormality after a dive is usually (correctly) assumed to be due to DCI. However, unrelated disease processes may coincidentally become evident shortly after a dive and can therefore complicate the diagnosis. Severe symptoms which begin after more than six hours following decompression without altitude exposure and any symptom occurring more than 24 hours after surfacing should raise the suspicion of an alternative diagnosis. A diagnosis of DCI should also be re-evaluated in a diver who fails to improve despite prompt recompression treatment. Table 2 shows several diagnoses which may be confused with DCI.[27]

Conclusions

The treatment of decompression illness using appropriate first aid measures and recompression treatment should be based upon clinical evaluation. To date, with the exception of inner ear damage (due to barotrauma or decompression injury), in which repeated audiograms and electronystagmograms are the most accurate means of

TABLE 2

CONDITIONS WHICH CAN MIMIC DCI
(Modified from Moon[27]).

Contaminated breathing gas (carbon monoxide)

Headache, nausea, vomiting, impaired consciousness. The diagnosis is suggested if several individuals who obtained their breathing gas from the same source are similarly affected. Diagnosis made by measurement of blood carboxyhemoglobin level or carbon monoxide in breathing gas.

Near drowning and hypoxic brain injury

Impaired consciousness, confusion. May be impossible to differentiate from arterial gas embolism.

Seafood toxin poisoning

Ingestion of large reef fish (Ciguatera),[28-30] Puffer fish (paralysis)[29] or shellfish (paralysis).[29,31] May present with focal neurological signs after ingestion of fish. Nausea, vomiting and abdominal pain frequently precede the onset of neurological symptoms in ciguatera poisoning. Diagnosis suggested if meal companions similarly afflicted.

Envenomation

Cone shell (paralysis),[29] Sea snake[29] or "Sea stroke".[32] "Sea stroke" is respiratory failure after presumed envenomation while swimming in the ocean on the east coast of the USA.

Migraine[33,34]

Focal neurological deficit preceding headache. Diagnosis suggested by a history of migraine headaches.

Guillain-Barré syndrome

Progressive neuromuscular weakness which can lead to respiratory failure. Progression is over several hours or days, unlike severe neurological DCI, which usually progresses over minutes.

Porphyria

Neuropathic pain, frequently in the abdomen, may be accompanied by neurological signs, including impaired consciousness and mental changes. Diagnosis suggested by history of similar attacks.

Sickle cell crisis

Acute onset of severe pain, usually in the limbs. Most common in black people. Diagnosis suggested by history of similar attacks. Microcytic anemia and hemoglobin S.

Multiple sclerosis

Onset of neurological symptoms/signs usually over hours or days. Acute, reversible abnormalities can sometimes be experienced by patients with a history of the disease, apparently triggered by stress such as a change in environmental temperature.

Transverse myelitis

Acute onset of back pain and signs of spinal cord dysfunction. Progression usually over hours, rather than minutes. Abnormal cord MRI.

Spinal cord or root compression

Due to disc protrusion, hematoma or tumor. May be difficult to differentiate from DCI. Poor response to recompression. Diagnosis confirmed by MRI.

Middle ear or sinus barotrauma with cranial nerve compression[35-41]

Isolated cranial nerve (V or VII) abnormality due to "reverse squeeze". Associated with sinus pain or difficulty clearing middle ear on ascent.

Inner ear barotrauma[21,35]

Symptoms of vertigo, hearing loss usually occur during compression, rather than after decompression. Usually a history of difficulty equalizing middle ear pressures and middle ear barotrauma evident.

Stroke

Ischemic[42] or hemorrhagic stroke. May be difficult to differentiate from DCI. Poor response to recompression. Diagnosis confirmed by MRI.

Subarachnoid haemorrhage

Suggested by nuchal rigidity, subhyaloid haemorrhages. Diagnosis confirmed by MRI, lumbar puncture.

Cold water immersion pulmonary edema[43]

Pink frothy sputum, dyspnea, usually occurring early in a cold water dive, before significant depth-time exposure. Chest radiograph demonstrates pulmonary edema. "Chokes" (cardiorespiratory DCI), caused by high levels of venous emboli, also causes pulmonary edema, but is not usually accompanied by expectoration of pink sputum, and requires significant depth-time exposure.

Unrelated seizure, post-ictal state (hypoglycemia, epilepsy)

Seizure in water during decompression or after exiting the water usually attributed to AGE until proven otherwise. Hypoglycemia severe enough to cause unconsciousness and seizure only likely to occur after administration of insulin or other blood glucose lowering medication, or insulinoma.

Functional abnormality[44]

Suggested by history of hysteria or conversion reactions, secondary gain (e.g. compensation for disability) with apparent deterioration days or weeks after good response to recompression.

assessment, there are no laboratory, radiographic or electrophysiological tests which are more sensitive that clinical examination for the diagnosis of decompression illness. A history and physical examination are usually sufficient to make the diagnosis and plan the treatment.

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