This process is outlined in more detail by both Sackett and Bennett.[3,4]

The most appropriate outcome is of course, better practice with improved outcomes for patients. The process described here is not foolproof and does not guarantee best practice. Each finding will require careful synthesis by the clinician into the overall situation of the individual patient. EBM provides systematic advice on existing evidence, only the clinician can actually treat the patient.

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A POSSIBLE CASE OF CEREBRAL ARTERIAL GAS EMBOLISM IN A BREATH-HOLD DIVER

David Williams

Key Words

Breath-hold diving, case report, decompression illness, cerebral arterial gas embolism

Introduction

Cerebral arterial gas embolism (CAGE) is second only to drowning as the most common cause of death in recreational SCUBA divers;[1] however, it is extremely rare in breath-hold divers unexposed to a compressed air source. The history of a possible case of CAGE in a previously healthy breath-hold diver is described here; and the differential diagnoses are discussed.

Clinical history

A fifteen year old male, from Munda in the Solomon Islands, made frequent repetitive breath-hold dives over a period of three and a half hours to spear fish. His maximum depth was approximately 8 m. On surfacing from his last dive, he developed a sudden severe headache, dizziness, blurred vision, and numbness and weakness of all four limbs. He was unable to stand or walk and had to be carried from the water by his father.

The symptoms persisted, and he was admitted to the Helena Goldie Hospital, Munda, the following day. He had no previous history of medical problems (specifically, no history of pulmonary or neurological illness), and had been completely well prior to and during his breath-hold dives. There was no history of exposure to a compressed air source, and he was the only person in the water at the time that the

incident occurred. There was no history of marine envenomation.

On examination, the patient had a well built muscular physique. He was apyrexial with a respiratory rate of 20, heart rate 64 bpm and blood pressure of 100/70 mmHg. There were no rigors, neck stiffness or depression of conscious level. There were no skin lesions, rash, or signs of envenomation. Hearing was normal, but there was vertigo and blurred vision bilaterally. Visual fields were grossly normal, and there was no evidence of nystagmus. There were no clinical signs of pulmonary barotrauma (pneumothorax, pneumopericardium or subcutaneous emphysema). There was movement of all four limbs, but with profound weakness and numbness.

X-Ray equipment was available, but its use was limited due to practical and financial considerations. In view of these restrictions and the absence of clinical evidence of pneumothorax, a chest X-Ray was not performed.

Following discussion between the doctors at the base hospital and the Divers Emergency Service in Adelaide, the findings were consistent with a diagnosis of cerebral arterial gas embolism (CAGE). For financial and logistical reasons, it was impossible to arrange retrieval from the remote location to a hospital with a hyperbaric facility. He was, therefore, kept supine with continuous surface oxygen via a Hudson mask at a flow rate of 4 l/min, and given 3 litres of 0.9% saline intravenously over 8 hours. Due to the lack of adequate cardiac monitoring, it was not possible to administer an intravenous lignocaine infusion.

The headache, blurred vision and neurological changes resolved progressively and completely within 24 hours of commencing surface oxygen therapy. A blood film showed the presence of falciparum malaria, which was treated with a 2 day course of quinidine and Fansidar (sulfadoxine/pyrimethamine). There were no sequelae, and the patient made a full recovery.

Discussion

The presentation of a catastrophic neurological event of sudden onset and equally sudden resolution in a previously healthy young male breath-hold diver is highly unusual. The differential diagnoses to consider in this case were: cerebral malaria, unrecognised marine envenomation, decompression sickness (DCS), cerebral arterial gas embolism (CAGE) and some other unrecognised neurological or psychiatric illness.

CEREBRAL MALARIA

The finding of malarial parasites on the patient's blood film is not remarkable as chronic infection with *Plasmodium falciparum* is endemic in the population of the Solomon islands, with a prevalence of around 30%. Cerebral malaria is associated with several days of non-specific feverish symptoms, followed by impairment of consciousness, generalised convulsions, and coma which persists for 24 to 72 hours.[2] Neurological examination usually reveals symmetrical upper neuron dysfunction with generalised extensor spasms and decorticate or decerebrate rigidity or opisthotonus. Supportive management and administration of intravenous quinine dihydrochloride or quinidine gluconate are required. The clinical presentation of our subject and rapid recovery with surface oxygen would suggest that cerebral malaria was not the cause of the symptoms in this case.

ENVENOMATION

Venomous species found in the vicinity of the Solomon Islands include sea snakes, cone shells, stone fish, blue ringed octopus and jellyfish (including *Chironex* and *Irukandji* species). Although muscle weakness and peripheral numbness may be a feature of envenomation by these organisms; they all cause skin lesions which may be seen on careful examination, and envenomation by cone shells, stonefish, scorpion fish and *Chironex* is associated with severe pain. Painless envenomation may occur from sea snakes, blue ringed octopus, or *Irukandji*; however, autonomic nervous system involvement is a prominent feature of both blue ringed octopus and *Irukandji* envenomation, and the myotoxins in sea snake venom cause characteristic myalgia and myoglobinuria.[3]

A number of cases of severe prolonged neurological deficit of sudden onset ("sea stroke") have recently been described in divers off the coast of North Carolina, USA , and are thought to be due to brainstem infarction as the result of envenomation by an as-yet-unknown marine organism.[4]

None of the above explain the course of the symptoms observed in our subject.

DCS AND CAGE

The presentation of a severe neurological deficit in a previously healthy subject which is related to diving, is of sudden onset, and resolves on treatment with oxygen, is strongly suggestive of decompression sickness (DCS) or cerebral arterial gas embolism (CAGE). Typically, the neurological deficit associated with CAGE is profound, resembling an embolic stroke, occurs immediately on surfacing and is often associated with loss of consciousness. The neurological symptoms of DCS are less severe, with a latent period of several minutes to 48 hours after surfacing, and with gradual progressive onset of pain or paraesthesia. DCS frequently also involves joints, skin

and lungs. CAGE may resolve after several hours, due to spontaneous passage of bubbles through the cerebral circulation, and tends to improve or resolve completely with surface oxygen. DCS is more persistent, migratory, and has variable and poor response to surface oxygen alone. The clinical course in this case was more consistent with CAGE than DCS; however it may be difficult to distinguish clinically between CAGE and DCS and the two pathologies may co-exist. However the management is similar, being oxygen and early recompression. Consequently, a unified diagnosis of acute neurological decompression illness (DCI) has been proposed.[5]

DCS IN BREATH-HOLD DIVERS

The occurrence of DCS in repetitive breath hold divers unexposed to a compressed gas source has been well documented.[6-11] The clinical manifestations range from intellectual impairment to vertigo and nausea, paralysis, unconsciousness and death.[6]

Nitrogen becomes dissolved in tissues as a function of time and depth as dictated by Dalton's Law of partial pressures and Henry's Law of gas solubility. Rapid ascent results in bubble nucleation and growth in the tissues and vessels. On a single breath-hold dive, it is virtually impossible to acquire sufficient tissue nitrogen loading to cause significant bubble formation on ascent. However, deep repetitive breath-hold dives with insufficient surface interval between dives may result in cumulative nitrogen loading of tissues, with consequent DCS.[8,12]

No-decompression limit tables have been calculated for repetitive breath-hold divers which predict that repeated breath-hold dives to a depth of 66 feet (20 m) can be made safely, provided that the surface interval is greater than or equal to the bottom time for each dive.[13] However, surface intervals of half the bottom time result in a high risk of DCS after only 2 hours of repetitive breath-hold diving.

Although the maximum depth and total duration of the series of dives was known in the case described above, information about the duration of individual dives and surface intervals was unfortunately not available. With a history of repetitive dives over a 3 hour period, it is possible that the subject had acquired a significant inert gas load. However, his maximum depth was only 8 m which makes it difficult to explain the severity and rapidity of onset of symptoms on the grounds of venous and tissue bubble formation alone.

PATHOPHYSIOLOGY OF CAGE

If compressed gas is breathed at depth and the diver ascends, the gas in the lungs will expand by Boyle's law as the ambient pressure falls. If it is unable to escape due to breath holding or bronchospasm, the increase in volume and pressure as the gas expands may cause the lungs to rupture. Gas can then escape into the pleural cavity, causing pneumothorax; into the mediastinum and soft tissues of the neck, causing surgical emphysema; into the pericardial cavity, causing pneumopericardium; and into the pulmonary arterioles, causing arterial gas embolism. The term pulmonary over-pressurisation syndrome (POPS) has been used to describe this sequence of pulmonary over-inflation, rupture, and escape of gas into extra-alveolar locations.[1]

It is not known whether disruption of the alveolar membrane occurs due to barotrauma, volutrauma (shearing forces between adjacent tissues of heterogeneous compliance), or a combination of both.[14,15] It has been suggested that the alveoli rupture when the transthoracic pressure gradient exceeds 10 kPa;[16] however studies on human cadavers have demonstrated a " bursting threshold " of as little as 73 mmHg (9.7 kPa).[17] It is believed that mechanical disruption of the alveolar-arterial barrier allows pressurised gas to enter the arterial circulation; however, this does not explain the observation that CAGE and pneumothorax are found together in less than 5% of divers.[18,19]

CAGE may also occur when bubbles of venous or tissue origin enter the arterial circulation via a right-to-left shunt due to an atrial or ventricular septal defect, patent foramen ovale or pulmonary arteriovenous fistula. The term "paradoxical embolism", which has been used to describe this mechanism, is a misnomer as the mechanisms involved are exactly what would be predicted from a basic understanding of the physiological principles involved.[20]

Approximately 25% of the population have a "probe-patent" foramen ovale which is functionally closed under normal conditions, but may open if right atrial pressure is raised, permitting right-to-left shunting to occur, with arterialisation of venous emboli and subsequent CAGE.[15] This may occur in divers on release of a forced Valsalva manoeuvre when clearing the ears, or straining to lift a heavy object,[21] or due to hydrostatic pressure on the thorax when a diver is partially immersed in the vertical position.[22]

No heart murmurs suggestive of a gross right-to-left shunt were found in our subject. However it is possible that he may have had a small shunt or probe-patent foramen ovale, which could only be diagnosed with the aid of echocardiography, and could have led to arterial embolisation of bubbles of venous or tissue origin.

CAGE IN BREATH-HOLD DIVERS

CAGE and POPS may occur in breath-hold divers who perform a breath hold ascent after taking a breath from

a SCUBA diver's alternative air source at depth.[23] However they may also occur in breath-hold divers who have not been exposed to a compressed air source.[9,24,25]

The mechanism is uncertain, as in theory, the diver should return to the surface with no more air in the lungs than when the dive began. It is hypothesised that local air trapping may occur during the dive with subsequent expansion and pulmonary rupture on ascent.[26,27] Healthy lungs are close to their elastic limit when held at total lung capacity (TLC) and it has been suggested that lung rupture may actually occur before the dive due to the shearing forces caused by maximal inhalation.[28] It is also possible that the displacement of blood into the thorax on submersion causes a reduction in compliance which makes the lung more likely to rupture. Diseased lungs are probably more prone to rupture, as they may have poorly communicating gas-filled spaces, weakened areas due to subpleural bullae or blebs, or areas of heterogeneous compliance due to global or focal fibrosis, which may generate and focus shearing forces.[28]

A number of techniques employed to extend the depth or duration of breath-hold dives result in increased intrathoracic pressure, which may predispose to POPS, CAGE, "paradoxical" embolism and syncope due to decreased venous return to the heart.

The increasing ambient pressure on descent causes blood to be redistributed from the periphery to the intrathoracic vascular bed, reducing vital capacity (VC) and limiting maximum diving depth. To prevent this, breath hold divers often voluntarily increase intrathoracic pressure by performing a maximal inspiration to TLC prior to descent. They may then further increase intrathoracic pressure by a technique known as "buccal pumping" or "lung packing" in which additional mouthfuls of air are forced into the lung by swallowing whilst maintaining an open glottis. This technique may increase the VC by up to 39% by forcing intrathoracic blood into the periphery; however the technique is extremely dangerous, resulting in a dramatic rise in airway pressures of up to 5.72 kPa.[29]

To prolong the duration of the dive, some divers swallow repeatedly or perform a Valsalva manoeuvre while at depth which can delay the conventional breaking point by modifying chest wall mechanoreceptor activity. However this may also result in a potentially dangerous increase in intrathoracic pressure.

Our subject had no history of pre-existing lung disease and did not report the use of pre-dive maximal inspiration, lung-packing, or Valsalva manoeuvre at depth during his series of dives.

OTHER CAUSES

It is possible that the patient may have had a transient ischaemic attack or some other catastrophic spontaneously resolving neurological event; however there is no reason for this to occur in a healthy young subject. Psychiatric illness or malingering could present in this way, however there was no preceding history of psychiatric illness nor secondary gain to be derived.

Conclusion

II would appear that the most likely explanation for his symptoms was that the breath-hold diver experienced CAGE due to pulmonary barotrauma or systemic "paradoxical" embolisation of bubbles formed as a consequence of nitrogen loading in the tissues and venous circulation from repeated breath hold dives.

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