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Neurological hazards associated with scuba diving

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Letter to the Editor

Recreational scuba diving has become a popular activity these days. But, it is associated with its share of medical complications which includes some important neurological hazards.

There are four main types of neurological involvement:

1) **Dive-Related Barotrauma** – It refers to tissue damage that occurs when some gas-filled body space (e.g.middle ear, lungs) fails to equalize its internal pressure to accommodate the changes in ambient pressure[1]. In accordance to Boyle's law, the volume of a gas varies inversely with pressure. While descent, with ambient pressure increasing, the volume of gas-filled spaces decreases unless internal pressure is equalized. If the pressure is not equalized by larger volume of gas, the space will be filled by tissue engorged with fluid and blood. This process affects the middle ear, external auditory canal, sinuses and teeth.

   Barotrauma to the middle or inner ear can occur during the descent or ascent phases of the dive[2]. Middle ear barotrauma of descent is the most common type of diving injury and may involve hemorrhage and rupture of the tympanic membrane with symptoms including acute onset of pain, vertigo and conductive hearing loss. In severe cases (usually during ascent), increased pressure in the middle ear can cause reversible weakness of the facial nerve and Bell's palsy (facial baroparesis)[3]. Inner ear barotrauma also can develop in patients with middle ear barotraumas with symptoms including acute onset of vertigo, sensorineural hearing loss, tinnitus, nausea and emesis.

   Pulmonary barotrauma is the most severe form of barotrauma and occurs during ascent [2]. As the ambient pressure is reduced during ascent, gas inside the lungs expands in volume [4]. If the expanding gas is not allowed to escape by exhalation, the alveoli and surrounding tissues tear. Several forms of pulmonary barotrauma can develop, including mediastinal emphysema, subcutaneous emphysema, pneumothorax and arterial gas embolism. Arterial gas embolism is the most dangerous form of pulmonary barotrauma and accounts for nearly one fourth of the fatalities per year among recreational divers [5]. A large proportion of air bubbles can reach the brain, occlude blood vessels and cause stroke-like events. The most common signs and symptoms of arterial gas embolism are neurologic, although pulmonary symptoms may also be present. Almost two thirds of patients with arterial gas embolism have alterations of consciousness (i.e., coma or obtundation). Seizures, focal motor deficits, visual disturbances, vertigo and sensory changes are also common. Spinal cord lesions occur less frequently [1].

2) **Decompression sickness** - It is caused by the release of inert gas bubbles (usually nitrogen) into the bloodstream and tissues after ambient pressure is reduced[2]. Decompression sickness is classified into type I and type II. In type I decompression sickness, symptoms are usually mild and may manifest as fatigue or malaise (i.e., constitutional decompression sickness) or may be more specific, involving the skin, muscles and joints[6]. Type II decompression sickness is usually more severe and can affect the vestibular apparatus, lungs and the nervous system. The formation of bubbles affects the brain, spinal cord, cranial and peripheral nerves, and the neural vasculature. Nitrogen bubbles injure nervous tissues by mechanical disruption, compression, vascular stenosis or obstruction, and activation of inflammatory pathways [6]. Cerebral decompression sickness (30 to 40 percent of cases) usually involves arterial circulation, while spinal cord decompression sickness (50 to 60 percent of cases) involves obstruction of venous drainage and the formation of bubbles within the cord parenchyma [7]. The incidence of decompression sickness among recreational scuba divers is estimated to be one case per 5,000 to 10,000 dives [8]. In addition to the rate of ascent and dive profile, other factors may influence the risk of decompression sickness, including fatigue, hypothermia, increased age, dehydration, alcohol intake, obesity, female gender and patent foramen ovale [2]. Neurologic decompression sickness can present with a wide spectrum of symptoms. The most severe presentation is partial myelopathy referable to the thoracic spinal cord with patients complaining of paresthesias and sensory loss in the trunk and extremities, tingling or constricting

sensation around the thorax, ascending leg weakness ranging from mild to severe, pain in the lower back or pelvis and loss of bowel and/or bladder control [6]. The neurologic examination often reveals monoparesis or paraparesis, a sensory level and sphincter disturbances. However, neurologic examination also may be normal. Pathologic features within the spinal cord include hemorrhagic infarctions, edema, bubble defects, demyelination and axonal degeneration [7]. Cerebral decompression sickness can occur alone or in combination with spinal decompression sickness and manifests as an alteration of mentation or confusion, weakness, headache, fatigue,gait disturbance, diplopia or visual loss. The neurologic examination may show hemiparesis, dysphasia, gait ataxia, hemianopsia and other focal signs. Behavioral and cognitive aspects of cerebral decompression sickness may be persistent or slow to improve [6]. The pathologic features are similar to those of spinal decompression sickness, although not as pronounced. The diagnosis of neurologic decompression sickness is clinical and should be suspected in any patient with a recent history of diving who has a consistent presentation. MRI demonstrates high-signal lesions of the brain and spinal cord in 30 to 55 percent of cases which suggests ischemia, edema and swelling. The lesions do not enhance with contrast. The initial management of neurologic decompression sickness is similar to that of arterial gas embolism and decompression illness, and requires transport to a recompression facility [2]. Recompression therapy reduces the size of bubbles, allowing easier re-absorption and dissipation, and increases the nitrogen gradient to expedite off-gassing. The majority of recreational divers with neurologic decompression sickness have excellent recovery after prompt recompression therapy.

3) **Headache** – It is a common symptom in divers with numerous benign cases including exacerbation of tension or migraine headaches, exposure to cold, mask or sinus barotrauma, sinusitis and a tight face mask. Dangerous causes of headache include cerebral decompression sickness, contamination of the breathing gas with carbon monoxide, arterial gas embolism, severe otic or sinus barotrauma with rupture, and oxygen toxicity [2]. If headache occurs in a patient with potential arterial gas embolism or decompression sickness, it should be considered an emergency, because it suggests the presence of intracerebral bubbles. This type of headache usually develops within minutes of ascent. Immediate use of 100 percent oxygen and of recompression therapy is indicated.

4) **Oxygen toxicity** - Most likely cause of oxygen toxicity is diving with oxygen enriched air (i.e.Nitrox). Nitrox is a breathing mixture that contains more than 21 percent oxygen (usually 32 to 36 percent), and allows extended bottom time. When diving with Nitrox, the diver is at risk of oxygen toxicity if the maximum oxygen depth limit and/or the oxygen time limit is exceeded. In general, the higher the oxygen content in the Nitrox mixture, the shallower the dive to minimize the potential for oxygen toxicity. Symptoms develop at depth without warning and consist of focal seizures, vertigo, nausea and emesis, paresthesias, visual constriction and respiratory changes [9]. Generalized seizures (which may be even fatal) or syncope can also occur in 5 to 10 percent of patients. The cause of oxygen toxicity to the nervous system mainly involves oxygen free radical formation, as well as reduction of the inhibitory neurotransmitter, gammaaminobutyric acid. Treatment consists of reducing oxygen exposure and dive depth and, if necessary, managing seizures.

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