Cardiovascular Concerns in Water Sports

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KEYWORDS

• Diving • Triathlon • Immersion • Swimming-induced pulmonary edema
• Immersion pulmonary edema • Decompression sickness • Arterial gas embolism

KEY POINTS

- Excess dyspnea and cough during the swimming phase of a triathlon is likely caused by swimming-induced pulmonary edema.
- Divers who develop extensive pulmonary barotrauma on ascent from depth may develop shock and pulseless electrical activity of the heart because of obstruction of the central circulation with air.
- Adults with congenital, valvular, and acquired heart disease may require special training to avoid adverse hemodynamic effects of water immersion and underwater exercise.
- Divers and swimmers with a long QT syndrome may develop ventricular tachycardia when swimming or diving.

Although there are a variety of water sports that have effects on the cardiovascular system, this article is confined to the sports that are affected by two important factors involved with water exposure. These include the physiologic effects of immersion on the cardiovascular system and the effects of exposure to increased pressure that include the physical effects of pressure based on the principles of Boyle's law (direct pressure effects[1,2]), and the effects of increased pressure on kinetics of gases in tissues and organs based on Henry's law (indirect pressure effects[3]). In some exposures all three effects may combine to cause abnormal stresses on the circulatory and the respiratory systems. To understand these various physiologic effects, it is necessary to provide a brief review of the physics of immersion and diving.

DEPTH AND PRESSURE

Pressure, defined as force/unit area, is related to the weight of a fluid column by the formula: pressure = height·density. Thus, pressure in water is directly related to depth below the surface. **Table 1** shows the relation between depth in seawater and

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Table 1
Relationship between depth in seawater and ambient pressure

	feet	atm	mm Hg	psi
Sea level	0	1	760	14.7
	33	2	1520	29.4
Depth in seawater	66	3	2280	44.1
	99	4	3040	58.8
	132	5	3800	73.5
	165	6	4560	88.2

pressure. In seawater, an increase in pressure of 1 atm (14.7 psi; 100 kPa) occurs for each 33 feet of seawater (FSW). Because of the slightly lower density of freshwater, the depth to achieve 1 atm pressure is 34 feet. Because of the fixed relationship between height and pressure, pressure is often expressed in units of length (millimeters of mercury, FSW, and so forth).

BOYLE'S LAW

For a fixed mass of an ideal gas, Boyle's law states that the product of pressure and volume ($P \cdot V$) is constant.[1] Thus a volume of gas at the surface of seawater at a pressure of 1 atm absolute pressure (ATA) is reduced to one-half of the original volume at 33 FSW (2 ATA). Conversely, a volume of gas at 33 FSW doubles as it rises from 33 FSW to the surface. This relationship has important implications related to lung volumes during excursions underwater.

HENRY'S LAW

This law states that the volume of dissolved gas in a liquid is proportional to the partial pressure of the gas.[3] Gas partial pressure is determined by the product of the ambient pressure and the proportion of the gas in the gas mixture. In air (79% nitrogen) at 33 FSW for example, partial pressure of nitrogen is $0.79 \cdot 2$ ATA = 1.58 ATA. This relationship is particularly applicable to decompression sickness (DCS) that results from supersaturation of inert gas in tissues and water with the risk of formation of a gas phase (bubbles) and tissue injury.

IMMERSION

When an individual is immersed in water, the vertical fluid column produces a pressure that counters the intravascular pressures that result from the height of the blood column below the heart. This effect results in an estimated 600 to 700 mL of blood to shift from the veins into the central circulation.[4] This volume is accommodated by expansion of the pulmonary vasculature, and by increased stroke volume based on the Starling relationship.[4,5] In individuals with reduced systolic or diastolic ventricular function, this shift can result in an increase in left ventricular end diastolic pressure with subsequent pulmonary venous congestion and pulmonary edema. Stickland and colleagues[6] demonstrated that exercise-trained individuals show a lower pulmonary capillary wedge pressure at high exercise loads compared with untrained individuals. Their data suggest that dyspnea related to extreme exercise is related to increased pulmonary capillary pressure and interstitial lung edema that impairs gas exchange. The work of West and colleagues[7,8] in race horses performing extreme exercise supports this hypothesis. He studied several horses that developed exercise-induced pulmonary hemorrhage when racing at maximum performance (**Fig. 1**). When added to