

IPE?

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A healthy 33-year old military diver with no significant past medical history and 350+ logged dives leaves the surface for a routine SCUBA requalification dive. The planned maximum depth is 45 feet; bottom time is to be no longer than 40 minutes. The water temperature is 51° F (11° C), and the outgoing tide is producing a moderate current. Five minutes after beginning the dive, he surfaces and appears to be in distress. He complains of mild dyspnea (difficulty breathing) and is assisted back to the dive boat, where he is given oxygen via mask. He reports that his dyspnea began shortly after he reached the bottom at 42 feet; he has no other complaints. The other divers are recalled and brought back onto the boat, which immediately departs for the standby hyperbaric chamber. On disembarking the boat at the chamber facility, the diver expectorates a small amount of pink, frothy sputum. He continues to complain of dyspnea but refuses help walking and denies any other problems. He is evaluated by the diving physician at the chamber and found to be neurologically intact. His chest x-ray shows diffuse pulmonary edema (accumulation of fluid in the lung tissues) with no other abnormalities noted. His equipment is examined later and determined to be functioning properly. He has 2850 psi left in his 80 cubic foot tank.

What happened to this diver, and what's the treatment?

If a diver reports difficulty breathing, our minds might jump to the “obvious” answer that, because he’s been diving, he has some sort of compressed-gas injury like pulmonary decompression sickness (“chokes”) or a lung overinflation injury. In this case, we’ll need to look a little deeper, so to speak. Right away we can rule out pulmonary DCS; his bottom time isn’t long enough to support that diagnosis. Also, considering that he surfaced before he planned to, it’s likely that he experienced problems on the bottom, an assumption that’s backed up by his own report.

He wasn’t out of air, nor did he have an equipment malfunction, either of which could have caused him to aspirate water. He doesn’t have any heart problems, which are a common cause of pulmonary edema.

What, then, is the source of our diver’s problems?

Some fascinating physiological changes take place when the body is immersed in water. The effect of gravity, which on the surface tends to pull our blood toward the lower portions of our bodies, is all but neutralized. Cold temperatures cause the arteries in our extremities to constrict, which directs blood toward our vital organs. So, when we jump into the water, especially cold water, a sizable amount of blood is quickly shifted to our core. In some people, for reasons which are still under investigation, this phenomenon results in a

dramatic increase in the pressure inside the tiny capillaries of the lung, called *pulmonary capillaries*. This condition is known as *pulmonary hypertension*, or high blood pressure in the pulmonary circulation. This elevated pressure drives fluid into the lung tissue and produces *immersion pulmonary edema*, or IPE.

Pulmonary edema occurs in the tissue of the delicate air sacs known as *alveoli*. This tissue is normally so thin that gases pass through it and into the pulmonary capillaries without difficulty. If the alveolar membrane becomes thickened with fluid, oxygen and carbon dioxide won't move across it as easily. The diver's blood can be deprived of oxygen, a condition known as *hypoxemia*, and may also accumulate carbon dioxide in a condition called *hypercapnia*. The excess fluid can also leak into the alveoli and cause a productive cough. The sputum may be frothy and, if trace blood is present, it may be tinged pink. Severe pulmonary edema may lead to respiratory failure and death.

It's not just divers who are subject to immersion pulmonary edema. The condition has also been documented in swimmers (1). At Duke, an ongoing study is investigating the physiology behind immersion pulmonary edema. Though the study is not complete, it appears that genetics may play a role in the development of IPE in individual swimmers. In general, the precipitating factors appear to be cold water immersion combined with exercise (2).

A swimmer or diver who develops IPE may be short of breath, have a productive cough, and suffer from acute respiratory failure. Anyone suspected of having IPE should be transported to the closest available hospital emergency department as soon as possible. Prior to evacuation, properly trained personnel should give 100% O₂ via mask and be prepared to support ventilations. In the hospital, a physician will order a chest x-ray and may give a diuretic medication like furosemide (Lasix) to help remove excess fluid. Recompression therapy is not indicated in a diver with IPE, since it's not a compressed-gas injury. However, difficulty breathing while submerged may induce panic and cause a rapid ascent, which could precipitate an arterial gas embolism and/or decompression sickness. This is a complex situation that requires advanced medical management; the diver must be emergently evacuated via the fastest possible means.

Immersion pulmonary edema is a relatively rare disorder, but when it does occur, prompt recognition and treatment can have a significant influence on the outcome.

References:

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(2) Wester, T.E., Cherry, A.D., Pollock, N.W., Freiburger, J.J., Natoli, M.J., Schinazi, E.A., Doar, P.O., Boso, A.E., Alford, E.L., Walker, A.J., Moon, R.E. (2008). Effects of Head and Body Cooling on Hemodynamics During Immersed Prone Exercise at 1 ATA. *Undersea Hyperb Med.*, 35, 4.