

Immersion pulmonary edema may cause swimming deaths during triathlons

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Heart abnormalities linked to immersion pulmonary edema were present in a greater-than-expected proportion of triathletes who died during the competition's swim portion, according to a study led by researchers at Duke Health.

The findings, published Aug. 29 in the journal *BMJ Open Sport & Exercise Medicine*, are based on an analysis of autopsy reports of people who died in the United States and Canada between 2008 and 2015 while participating in triathlons. Triathlon competitions involve consecutive races of swimming, bicycling and running.

Study investigators identified 58 deaths during the time period, with 42 of those occurring during the swim. Researchers examined autopsy reports on 23 of the 42 deaths.

Richard Moon, M.D., the study's first author and a professor of anesthesiology and medicine at the Duke University School of Medicine, said he and co-investigators reviewed the autopsy reports to identify whether conditions were present that may have made the [athletes](#) susceptible to immersion [pulmonary edema](#) (IPE, in the study abbreviated as IPO based on the Greek spelling of oedema).

IPE, also known as swimming-induced pulmonary edema, occurs when the lungs suddenly fill with body fluids during activities in cold water, such as swimming and diving. IPE can lead to difficulty breathing, wheezing and confusion, which can be serious and even fatal.

While IPE can occur in healthy individuals, Moon said its onset is often seen in those with left ventricular hypertrophy (LVH), a condition where the heart muscle becomes thickened or heart mass increases. LVH typically occurs in people with high blood pressure and is a marker for susceptibility to IPE. A mildly enlarged heart—commonly referred to as athlete's heart—can also develop among endurance athletes, although athlete's heart is not believed to predispose to swimming-induced pulmonary edema.

Moon and co-researchers compared figures from the autopsy reports in the current study to data from previous studies, including one published in 1997 led by Pamela Douglas, M.D., professor of medicine at Duke. Douglas' investigation looked at the prevalence of LVH in 225 athletes who completed a triathlon in Hawaii from 1985-95.

"Among the autopsy reports of the deceased triathletes, we found a much higher prevalence of LVH than the healthy athletes in Dr. Douglas' study," Moon said. "The degree of enlargement was also much greater in the triathletes who died."

Specifically, the 16 triathletes who died from unclear causes had higher measures of heart wall thickness. The autopsy reports showed abnormal cardiac septal thickness in 67 percent of the deceased athletes and abnormal posterior wall thickness in 50 percent. In the Douglas study, it was one percent and one-half percent, respectively.

While it cannot be definitively concluded that IPE was the cause of the triathlete deaths during swimming, Moon said the autopsy reports demonstrate a plausible link. He recommended that potential triathletes and their health-care providers be aware of the known connection between LVH and the potential risk of immersion pulmonary edema.

"The message is that if people have untreated hypertension or they're

known to have ventricular hypertrophy, they need to get evaluated and treated before they embark on this sport," Moon said.

Provided by Duke University Medical Center

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