Exploring the depths of immersion pulmonary edema

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Since first described by Wilmshurst et al. (6) in 1981, immersion pulmonary edema (IPE) has been of more than merely a casual interest to divers and surface swimmers.

The condition and its consequences are of particular concern in military diving communities, where IPE imposes not only significant health risk but also threatens to compromise training and operations (4).

Despite this interest, the scientific literature addressing this condition is scant compared with other forms of pulmonary edema. For example, studies related to high-altitude pulmonary edema (HAPE) exceed those of IPE by at least an order of magnitude. Just some of the factors that have hampered IPE research include the sporadic and currently unpredictable occurrence, its likeness to manifest in remote locations, and the inevitable removal of the IPE victim from the insult (immersion) to render medical attention. As such, the majority of work regarding IPE is limited to case reports or studies performed at shallow immersion.

The work in this issue of the Journal of Applied Physiology by Fraser et al. (1) is the third in a series of studies aimed at confirming ideas about diving physiology as they relate specifically to IPE. The research team should be commended on a cogent study design that executed complex monitoring in a unique hyperbaric environment, and their success testifies to an exceptionally skilled team. At least as worthy of accolades are the volunteer subjects of the study series for their contributions toward more thoroughly understanding this condition. By employing the gold standard pulmonary artery catheter in an immersed hyperbaric environment, the body of work this study series represents has significantly advanced the understanding of IPE encountered in diving environments.

Since its initial description, IPE has been considered more likely to occur in cold water and thought to be associated with some degree of vascular dysfunction. This hypothesis was reasonable and sound given that the thoracic blood volume engorgement seen with immersion has been demonstrated to increase in cold water. This, coupled with evidence of increased forearm vascular resistance in subjects who had suffered from IPE (especially on exposure to cold), and an apparent increase in systemic hypertension, lead one to conclude that some engorgement and vascular dysfunction were at the “heart” of the issue. Subsequently, there were small studies that suggested capillary stress failure, further strengthening the case for pulmonary vascular or cardiovascular dysregulation (2). However, it wasn’t until the first study from this series that significant progress was made toward understanding the cardiopulmonary vascular physiology in immersed cold-water exercise. The study demonstrated an increase in pulmonary artery pressure with exercise in cold water immersion that was attenuated with thermal body protection (5). However, “head only” thermal protection did not attenuate the rise in pulmonary artery pressure.

The techniques described in that first study were then applied to the hyperbaric environment [4.7 atmospheres absolute (ATA)]. Peacher et al. (3) studied the exercising diver in thermoneutral water and explored any potential benefits of breathing oxygen enriched air. To my knowledge this publication represents the most “in-depth” study of pulmonary and vascular physiology in diving environments and brings a tear to the eye of anyone who fondly recalls the halcyon years of basic whole human diving physiology.

In those studies, the overall increase in pulmonary artery pressure was observed, but with quite a range of variability, supporting the notion of individual IPE susceptibility. The pulmonary hypertension Peacher’s group noted in thermoneutral hyperbaric exercising divers was attenuated with oxygen-enriched air and approached the values seen in simple surface (1 ATA) immersion. Interestingly, the pulmonary capillary wedge pressure was unaffected by oxygen tension. These observations support elevated pulmonary vascular pressures as the cause of IPE but leave open the question as to whether susceptible individuals may have an excessive rise in pulmonary artery pressure caused by exaggerated peripheral vasoconstriction or nonhomogeneous pulmonary vasoconstriction, as has been suggested for HAPE.

This work presented by lead author Fraser reporting the third study in this series describes a lack of protection provided by oxygen-enriched air in preventing immersion-related pulmonary hypertension in cold water at 4.7 ATA (1). In addition to the generous scientific value, their work provides practical insight as well. In summing up their body of work with an eye to application, if IPE is a concern the first goal should be to stay warm and consider an oxygen-enriched breathing mixture. And if you are unable to stay warm, the enriched oxygen is not so helpful. This certainly has direct implication for operational divers, especially those in the military.

Beyond delivering an elegant method for testing the pulmonary vascular system in diving and answering some basic whole human physiology questions, these studies also deliver the goods on expanding our questions. And after all, isn’t that the hallmark of good research?

So then, where are we and where do we go from here? What is the cause of the pulmonary vascular variability? Does the variability itself provide insights into other pathological processes in much the same way that research in hypoxic pulmonary vasoconstriction has enlightened the overall field of pulmonary vascular hypertension? What is the mechanism of cold-related pulmonary hypertension and why is that not improved with oxygen enrichment? What other modalities can be examined and tested for decreasing pulmonary pressures in diving? And last but far from least, do actual patients with IPE have an exuberant increase in pulmonary arterial pressures with immersion?
Once again, Fraser should be applauded for this effort, the publication, and the entire group congratulated for the body of work.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

REFERENCES


