Immersion-Induced Mitral Regurgitation
A Novel Risk Factor for Swimming-Induced Pulmonary Edema

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Immersion pulmonary edema, more commonly referred to as swimming-induced pulmonary edema (SIPE), is a well-documented condition believed to be a result of immersion physiologic condition that is characterized by a peripheral-to-central redistribution of blood volume. It disproportionately affects young, healthy athletes with no clinically overt cardiovascular or pulmonary conditions. We present four cases of healthy athletes with previously documented SIPE, who participated in Institutional Review Board-approved clinical studies that examined the pathophysiologic condition and prevention of SIPE. During standard recumbent echocardiography, trivial mitral regurgitation was observed in all four individuals. Acute exacerbation of their mitral regurgitation was observed during immersion with both immersed resting and immersed exercise echocardiography, contributing to the development of SIPE. These observations demonstrate that the occurrence of subclinical or trivial mitral valve regurgitation during dry rest is a novel risk factor for SIPE. We propose the use of immersion echocardiography as a useful investigative tool for otherwise healthy individuals with SIPE and no previously explainable cause.

KEY WORDS: immersion; immersion pulmonary edema; mitral valve insufficiency; swimming-induced pulmonary edema (SIPE)

Pulmonary edema in healthy swimmers or divers (immersion pulmonary edema, which is referred to more commonly as swimming-induced pulmonary edema [SIPE]) was first reported by Wilmshurst et al.1 During immersion, there is a redistribution of blood volume from peripheral tissues to the thoracic cavity that amounts to an estimated increase in heart volume of 50%.2 This blood volume redistribution is associated with increased pulmonary artery and wedge pressures.3 Based on enhanced peripheral vasoconstriction in response to cold in those who experienced SIPE, Wilmshurst et al1 hypothesized that this condition is a form of heart failure that is induced by the combination of cold exposure and immersion-related cardiac volume overload. Since then, >300 cases of SIPE have been reported; many of the individuals are young and exceptionally physically fit.4-6

In susceptible individuals, the increase in pulmonary vascular pressure during immersed or submersed exercise is greater than in the general population.7 The cause of
this disproportionate increase in SIPE-susceptible individuals is unknown, although some cases have been attributed to ventricular dysfunction,8 including Takotsubo cardiomyopathy4,5,9 and established valve disease.4 We present here another possible cause of SIPE: immersion-related mitral regurgitation (MR) in four individuals where trivial MR that was observed under dry resting conditions (recumbent, left side down) increased in severity during immersion in cold water.

Case Reports
All four subjects who experienced at least one diagnosed episode of SIPE were in otherwise good health and had no evidence of underlying cardiovascular or pulmonary disease. These individuals were among 16 subjects with histories of SIPE who were studied with immersed echocardiography under two protocols approved by the Duke University Medical Center IRB (Duke IRB protocol #s Pro00003158, Pro00019996 and Pro00100971). After informed consent, they underwent echocardiography in the recumbent position under standard dry resting conditions followed by rest and up to 40 minutes of mild exercise on an electrically braked cycle ergometer while immersed to the neck in 20°C water without or with an oral fluid load of 2 L Pedialyte (Abbott Laboratories). All grading of MR severity that are discussed in this case series was performed with transthoracic echocardiography with color flow Doppler imaging. Qualitative and quantitative parameters that were used to describe the severity were adapted from the American Society of Echocardiography 2017.10

Pulmonary edema was assessed with the use of symptoms, lung auscultation, change in spirometry (Vyaire Medical), chest radiography, and, in one case, pulmonary ultrasound imaging performed with a Lumify 1-4 MHz phased-array probe (Philips Healthcare) with a Samsung Galaxy Tab S3 tablet (Samsung) and the technique described by Volpicelli et al.11 The technique used during immersion is shown in Figure 1. Cases are summarized in Table 1.

Case 1
A physically fit 51-year-old white woman experienced her first episode of SIPE while windsurfing wearing a wetsuit. She fell off her board into the water (water temperature 14°C); within minutes, she reported feeling short of breath and having difficulty swimming. Once on shore, she began coughing pink frothy sputum and was admitted to a local hospital where her initial BP was 173/85 mm Hg, pulse rate 72 bpm, and blood oxygen saturation (SpO2) 85% on room air. Chest radiograph confirmed pulmonary edema. After recovery, follow-up stress echocardiogram revealed only trivial MR.

Prior to our study, her baseline measurements were BMI 20.4 kg/m², BP 112/88 mm Hg, and pulse rate 66 bpm.

<table>
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<th>Case</th>
<th>Fluid Load</th>
<th>Swimming-Induced Pulmonary Edema Symptoms</th>
<th>Symptom Onset Time After Exercise Start, min</th>
<th>Lung Auscultation Findings</th>
<th>Lowest SpO₂, %</th>
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TABLE 1  Summary of Collected Data Following Head-Out Immersed Exercise in 20°C Water
Spirometry (Vyaire Medical) was normal for her age, sex, and race. Her baseline echocardiographic study showed trivial MR; on immersion while at rest, her MR had increased to moderate. During initial exercise, MR further increased to severe and remained severe during the extent of the exercise. Clinically, however, she remained asymptomatic, and her vital capacity immediately after exercise was unchanged. After 24 hours, the study was repeated after oral preload. While fluid-loaded, her resting dry study showed severe MR that remained severe on immersion. Within 1 minute of exercise, the patient reported fatigue, dyspnea, and inability to maintain workload; exercise was stopped immediately. Lung examination at this time revealed bilateral crackles, and pulse oximetry was 92%. Within 2 hours after stopping the exercise, her symptoms completely resolved, and spirometry returned to normal.

Selected views from her ultrasonograph are shown in Figures 2 and 3. During both immersion encounters, the subject’s left ventricle ejection fraction, wall motion, and other detailed functional indexes, including left ventricular global longitudinal strains, remained normal. Of interest is the inferior vena cava diameter: with immersion, because of central blood redistribution, the inferior vena cava dilates slightly and loses its inspiratory dynamic collapse, as previously described in athletes, and is presumed to be a physiologic response to increased flow and pressure during exercise.12

This same individual had earlier participated in a separate study that examined the effects of immersion on pulmonary arterial pressure. After Institutional Review Board approval and having provided informed consent, she performed 6 minutes of cycle ergometer exercise immersed in cold water (20°C). Invasive measures of mean arterial pressure, central venous pressure, and mean pulmonary artery and wedge pressures were obtained. Results are shown in Table 2.

Figure 2 – Case 1: Serial parasternal long axis views show progressive increase in mitral regurgitation (arrows) among A, dry rest (trivial), B, immersed peak exercise (severe), and C, immersed recovery (even more severe). Ao = aorta; LA = left atrium; LV = left ventricle. Arrows show the mitral regurgitant jet.

Figure 3 – A-D, Case 1: A and C, Subcostal long axis expiratory and B and D, inspiratory through the inferior vena cava at dry rest (A and B) and exercise (C and D). Note the normal respiratory dynamic movement of the inferior vena cava diameters (distance between the opposite arrows) from A, expiration and B, inspiration. With immersion, the inferior vena cava dilates slightly but loses its respiratory dynamic collapse from C, expiration to D, inspiration. IVC = inferior vena cava. Arrows depict the IVC diameter.
Case 2

A 50-year-old white female scuba diver reported an episode of SIPE while attempting an open ocean dive wearing a 5-mm thickness wetsuit with a hood and using a rebreather apparatus. Water temperature at maximum depth of 200 feet of sea water was 10.9°C. After 17 minutes of progressive coughing and difficulty breathing while at maximum depth, she ascended and went to a local hospital where pulmonary edema was confirmed via chest radiography. Exercise echocardiography 3 weeks after this episode showed very mild aortic regurgitation, trivial-to-mild MR, and trivial tricuspid regurgitation without pulmonary hypertension and no other abnormalities.

At our facility, her baseline measurements were BP of 119/78 mm Hg, pulse of rate 55 bpm, SpO₂ of 97% on room air, and BMI of 27.9 kg/m². Spirometry and electrocardiography results were normal. Stress echocardiography showed no exercise-induced wall motion abnormalities. She performed head-out immersed cycle ergometer exercise in 20°C water with external workload of 75 watts. Echocardiography with the use of the same probe and machine as in Case 1 showed that severe MR developed immediately after immersion (Fig 4). After 19 minutes, she began experiencing symptoms similar to her previous episode of SIPE; after 23 minutes of exercise, she was unable to continue. Chest radiograph showed mild pulmonary edema. After 20 minutes, SpO₂ returned to 97% on room air. She remained asymptomatic for 2 hours and repeat spirometry and SpO₂ on room air and follow-up examination results the next day were normal.

Case 3

A 44-year-old white woman experienced SIPE during a triathlon, where approximately 200 m into the swim, she experienced fatigue, productive cough with blood-tinged sputum, and “rattling” in her chest. She was forced to abandon the swim and seek medical treatment. Months later, she experienced these symptoms again under similar circumstances and reported to a local hospital where a chest radiograph showed pulmonary edema. Further workup after the resolution included normal spirometry and negative methacholine challenge.

Her baseline measurements were BMI of 26.4 kg/m², BP of 106/50 mm Hg, pulse rate 54 bpm, and SpO₂ 99% on room air. Spirometry and echocardiography results were normal. Stress echocardiography showed no exercise-induced wall motion abnormalities, no valvular stenosis,
and trivial mitral, tricuspid, and pulmonic regurgitation with ejection fraction \(>55\%\).

During the study, she exercised at a workload of 150 watts after oral preload. After an exercise time of 6 minutes, she experienced dyspnea and cough and was unable to continue. Echocardiography (Philips Healthcare) showed moderate MR (Fig 5) while she was immersed during exercise. Immediately after exercise, \(\text{SpO}_2\) was 92%; crackles were heard on auscultation; FVC had decreased from 3.62 L to 3.00 L, and \(\text{FEV}_1\) had decreased from 2.85 L to 2.29 L. Chest radiograph showed interstitial pulmonary edema, and B-lines were seen on ultrasound imaging. Reevaluation 103 minutes after exercise showed \(\text{SpO}_2\) 97%, and spirometry had returned to preexercise baseline.

**Case 4**

A 39-year-old white woman was scuba diving to 50 feet of sea water depth in cold water (approximately 18°C). After 10 minutes, she noticed that it was difficult to breathe and slowly surfaced and boarded the boat. During exhalation, she reported “whistling and gurgling,” coughing, and a “sensation of drowning.” Abnormal breathing sounds lasted 3 to 4 hours; the

| TABLE 2 | Summary of Hemodynamic Measurements for Case 1 During Submersion in 20°C Water |
|---------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|
| Condition | Heart Rate, beats/min | Mean Arterial Pressure, mm Hg | Central Venous Pressure, mm Hg | Pulmonary Arterial Pressure, mm Hg | Pulmonary Arterial Wedge Pressure, mm Hg |
| Dry rest | 46 | 107 | 6 | 18 | 12 |
| Submersed rest | 77 | 130 | 1 | 20 | Not obtained |
| Submersed exercise | 134 | 141 | 5 | 33 | 17 |

See Reference 7 for details of the procedure.
cough lasted for 12 hours. She reported that, for the past several years after a day of diving in cold water, she would experience what she described as several episodes of "a deep, wheezy voice" that was accompanied by a hoarse cough. At our facility, her BMI was 22.8 kg/m²; her BP was 120/84 mm Hg; her pulse rate was 45 bpm, and her SpO₂ was 99% on room air. She performed head-out immersed cycle ergometer exercise in 20°C water for 40 minutes with a workload of 90 watts and oxygen consumption rate of 2.1 L/min. At baseline, she had mild MR that did not change during immersed exercise. No symptoms or signs of SIPE occurred. A second immersed exercise the next day was performed after oral preload; MR increased to moderate (Fig 6). After 25 minutes, she reported chest tightness, and the exercise was stopped. Lung auscultation revealed basal crackles. SpO₂ was 95%; chest radiography revealed mild pulmonary edema.

Control Subjects

The cases of the other 13 individuals with a history of SIPE who were studied in the same manner are summarized in the Supplementary Table. In this group, without fluid loading and during immersion, one person’s condition increased from trivial to mild MR. One person without significant MR experienced SIPE symptoms. A subset of eight individuals were also studied after fluid loading. Of these, MR increased from trivial to mild in only one individual. Five individuals experienced SIPE symptoms (four trivial MR, one mild MR).

In addition, 47 individuals with no SIPE history who had been preselected with a tricuspid regurgitation jet were studied; of these, 14 individuals were selected after fluid loading. Of this group, the maximum MR grade was mild (Supplementary Table). During immersion, one individual’s condition increased from none to mild, and the conditions of two individuals increased from trivial to mild. Only one of the 48 individuals experienced SIPE symptoms, with only trivial MR during immersed exercise after fluid loading. This person’s propensity to experience SIPE was previously unknown to him.

Discussion

Several potential predisposing cardiac conditions have been described that include hypertension and coronary artery disease. Of 292 cases of SIPE reviewed by Peacher et al, six individuals had evidence of preexisting established valvular disease, which included mitral valve prolapse or regurgitation. Fluid loading is a common practice before military training exercises and triathlons and may be an additional risk factor. With the use of echocardiography, the three cases described earlier did not have significant valve disease during dry rest (either morphologic or functional); however, we hypothesize that, in these individuals, the central redistribution of blood during immersion caused sufficient dilation of the mitral annulus or alteration of the geometry of the mitral apparatus to induce hemodynamically significant MR. This concept was suggested in a series of children who experienced transient MR during acute glomerulonephritis. After resolution of the glomerulonephritis, the MR resolved, which was hypothesized to be due to transient volume overload during the oliguric phase. To our knowledge, hemodynamically significant MR that occurs as a result of immersion has not been reported previously.

The four individuals described in this report had exceptional physical fitness and no other previously proposed predisposition for SIPE. Ventricular expansion from blood redistribution in addition to prehydration in cases 1, 3, and 4 distorted the mitral apparatus that created MR and a mechanism for SIPE development. The observation that this is not present in all SIPE-susceptible individuals is consistent with SIPE having many possible predisposing factors.

Conclusions

These cases demonstrate major exacerbation of mitral valve regurgitation during immersion in cold water. We conclude that subclinical mitral valve regurgitation at rest that worsens during immersion represents a novel and measurable risk factor for SIPE. Echocardiography during head-out immersion is a straightforward technique that requires only an immersion tank. We suggest the utility of immersion echocardiography as a useful investigation for individuals with a history of SIPE with trivial or mild MR with no other plausible cause.

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Additional information: The Supplementary Table can be found in the Supplemental Materials section of the online article.
References


