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Swimming-Induced Pulmonary Edema in Endurance Athletes: Pathophysiology, Diagnosis, Acute Management, and Return to Sport – A Narrative Review

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Abstract

Introduction and purpose. Swimming-induced pulmonary edema (SIPE) is an acute form of immersion-related pulmonary edema that can occur in otherwise healthy swimmers and triathletes. Because symptoms develop in the water and may progress rapidly, failure to recognize SIPE creates an immediate drowning risk. This review summarizes current evidence on epidemiology, pathophysiology, diagnosis, acute treatment, recurrence and return to sport.

Materials and methods. A narrative literature review was conducted using PubMed/MEDLINE and relevant consensus or position statements, with literature considered through August 2026. Search concepts included swimming-induced pulmonary edema, immersion pulmonary edema, open-water swimming, triathlon, pulmonary edema, lung ultrasound, hemodynamics, risk factors, treatment, recurrence and return to sport. Priority was given to systematic reviews, larger observational cohorts, hemodynamic and diagnostic studies, treatment studies and long-term follow-up data. Because this was a narrative review, study selection was guided by clinical relevance and evidential strength rather than a formal systematic-review protocol.

Results. SIPE appears to result predominantly from hydrostatic pulmonary edema caused by immersion-related central blood-volume redistribution, exercise, cold-induced vasoconstriction and individual susceptibility to elevated pulmonary vascular pressures. Typical features are acute dyspnea, cough, crackles, hypoxemia and sometimes hemoptysis during or shortly after swimming. Lung ultrasound can support rapid diagnosis. Initial management consists of immediate exit from the water, oxygen when required and escalation to positive airway pressure in selected patients. Recurrence is clinically important, and cardiovascular risk factors or structural disease should be considered after an episode.

Conclusion. SIPE should be considered whenever an endurance athlete develops unexplained respiratory distress during open-water swimming. Event-side recognition, appropriate supportive treatment and structured post-event evaluation are central to reducing drowning risk and guiding a safe, individualized return to swimming.

Keywords: swimming-induced pulmonary edema, immersion pulmonary edema, open-water swimming, triathlon, lung ultrasound, pulmonary circulation, return to sport

1. Introduction

Open-water swimming and triathlon place the cardiovascular and respiratory systems in an unusual physiological environment. Exercise is performed horizontally, often in cold water, while hydrostatic pressure redistributes blood from the periphery toward the thorax. In most athletes these changes are well tolerated. In a susceptible minority, however, acute pulmonary edema develops during or shortly after immersion. The condition is commonly termed swimming-induced pulmonary edema (SIPE), while immersion pulmonary edema (IPE) is often used as the broader term encompassing swimmers and divers [1-3].

SIPE is clinically important for a reason that is easy to overlook when the athlete is first seen on land: the critical phase begins in the water. Dyspnea, cough and hypoxemia can impair swimming ability and create a secondary drowning risk before medical assessment is possible. Presentations range from relatively mild breathlessness to severe hypoxemic respiratory distress with frothy or blood-stained sputum [4, 5].

SIPE is being recognized more often as participation in mass open-water events has expanded. Large recreational cohorts now provide event-specific incidence estimates, while invasive hemodynamic studies support a predominantly pressure-driven mechanism in susceptible individuals [6-8]. Long-term follow-up has also shown that recurrence is clinically relevant [9].

The aim of this narrative review is to integrate these findings into a clinically useful framework for endurance-sport medicine. The discussion focuses on epidemiology, mechanisms, individual and environmental risk factors, event-side diagnosis, differential diagnosis, acute management, evaluation after recovery and decisions about return to swimming.

2. Epidemiology and Clinical Burden

Early SIPE literature consisted largely of military series and case reports, which made the true frequency difficult to estimate. A field study of strenuous swimming in military trainees demonstrated that pulmonary edema could recur and that the syndrome was not limited to isolated anecdotal cases [10]. Later, Adir and colleagues described 70 young, healthy male trainees with SIPE, establishing a recognizable clinical pattern that included acute dyspnea, cough and hypoxemia [11]. Chest radiographs obtained 12-18 h after symptom onset were normal in that series, underscoring how rapidly radiographic abnormalities may resolve.

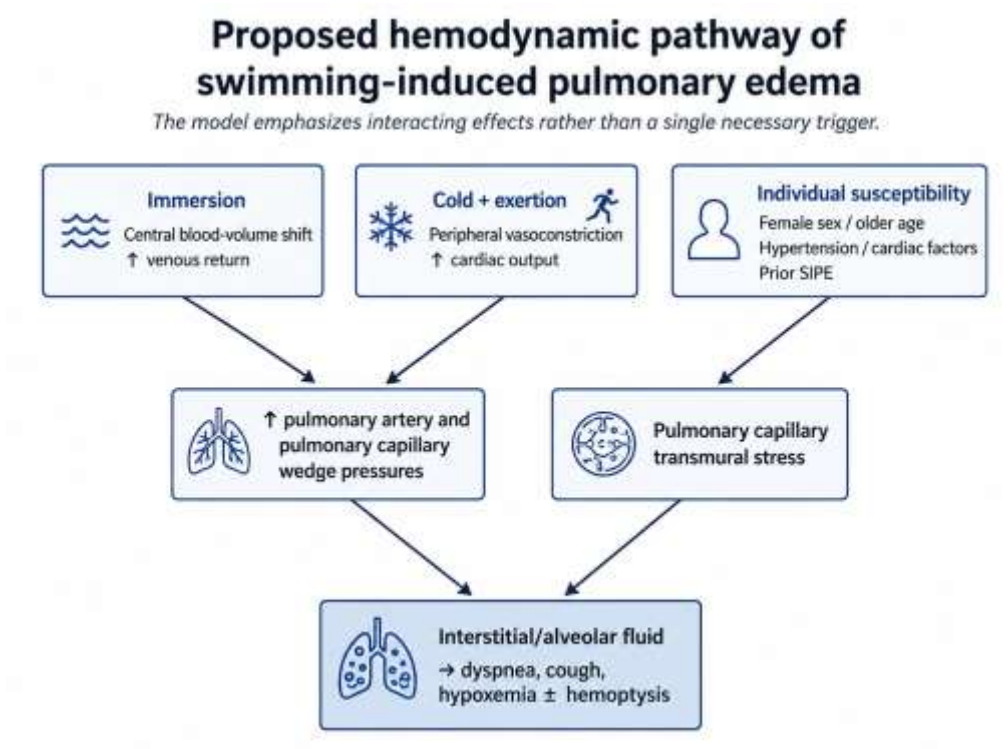
Population data from recreational endurance sport are now stronger. In a Swedish cohort covering 47,600 open-water swimming distances, the overall incidence of SIPE was 0.44%. Incidence was substantially higher in women than in men and rose with age [6]. These figures

are event-specific rather than universal, but they demonstrate that SIPE is not vanishingly rare in large open-water populations.

Military populations can show a much higher burden because exposure is intense and repeated. In a prospective observational study of 2,117 US Navy Special Warfare candidates, 106 SIPE cases were identified, corresponding to 5.0% of candidates during the observation period; repeat episodes also occurred [12]. The difference from civilian event estimates illustrates how case definition, water conditions, training intensity, population selection and repeated exposure influence apparent incidence.

A 2018 systematic review found substantial heterogeneity across published incidence and prevalence estimates and concluded that quantitative pooling was not appropriate [13]. This remains an important limitation: a diagnosis based on symptoms alone will not be directly comparable with one verified by lung ultrasound or radiography. Modern event studies using standardized clinical criteria and lung ultrasound have therefore been particularly valuable.

Figure 1. Proposed hemodynamic pathway of swimming-induced pulmonary edema (SIPE).



Source: own elaboration based on the reviewed literature

3. Pathophysiology: Why the Lung Becomes Vulnerable During Immersion

The central physiological event in immersion is redistribution of blood volume toward the thorax. Classic human immersion experiments demonstrated increased central blood volume and cardiac filling during head-out water immersion [14]. In a swimmer, this occurs while cardiac output is simultaneously increased by exercise. Cold exposure adds peripheral vasoconstriction, which can further increase venous return and systemic afterload.

The pulmonary circulation is therefore exposed to a combination of high flow and elevated filling pressures. The most direct evidence comes from invasive hemodynamic work in SIPE-susceptible individuals. During immersed exercise, Moon and colleagues found higher mean pulmonary artery pressure and pulmonary artery wedge pressure in susceptible subjects than in controls at comparable cardiac output [7]. These observations support a hydrostatic mechanism in which pulmonary capillary pressure rises sufficiently to drive fluid into the interstitium and alveoli.

The same study provides an important mechanistic clue rather than a routine treatment recommendation. Sildenafil reduced pulmonary vascular pressures during immersed exercise in SIPE-susceptible subjects [7]. This finding suggests that an exaggerated pulmonary vascular response is modifiable, but the study was small and was not designed to establish sildenafil as standard prophylaxis for endurance athletes.

Experimental studies of immersed exercise also show that pulmonary artery pressures can become substantial even in healthy individuals, especially under conditions combining immersion, cold and altered breathing mechanics [15, 16]. The concept of pulmonary capillary stress failure is therefore plausible: sufficiently high transmural pressure may disrupt the normally tight alveolar-capillary barrier and permit both fluid and, in some patients, erythrocytes to enter the alveolar space.

Not every susceptible athlete has an obvious resting abnormality. Cardiopulmonary testing performed after recovery from SIPE has been normal in some military subjects when assessed in dry conditions [17]. This helps explain why routine pre-participation evaluation may fail to identify risk. The abnormal physiology can be conditional—appearing when immersion, exertion and individual cardiovascular response occur together.

Cardiac factors warrant particular attention in older recreational athletes. Immersion-induced mitral regurgitation has been demonstrated in SIPE-susceptible individuals, suggesting that abnormal left-sided filling pressures may contribute in selected patients [18]. This does not imply that SIPE is primarily caused by structural heart disease; rather, it supports targeted cardiovascular assessment after severe, recurrent or otherwise atypical episodes.

4. Risk Factors and Predisposing Conditions

Risk factors for SIPE can be divided into individual susceptibility and exposure-related triggers. Earlier literature repeatedly implicated hypertension, previous SIPE, cold water, heavy exertion, tight wetsuits and excessive pre-event fluid intake, although the quality of evidence varied [2, 13, 19]. The distinction between a plausible mechanism and a proven independent risk factor is important, particularly when counseling athletes.

Some of the most informative recreational data come from the Swedish open-water program. Incidence increased with age and was higher in women [6]. A 2026 case-control analysis of 258 SIPE cases and 2,117 controls reported associations with female sex, older age, hypertension and heart disease and also identified asthma, limited open-water swimming experience, recent respiratory tract infection and previous respiratory symptoms during open-water swimming as potential risk markers [20]. Because the study is observational, these findings should not be interpreted as proof of causality.

Hypertension is biologically credible because an exaggerated vasoconstrictor response and increased left-sided filling pressures can amplify the hemodynamic effects of immersion. Long-term observations from early diving and swimming cohorts also linked immersion pulmonary edema with later hypertension [21]. In middle-aged and older recreational athletes, an episode of SIPE should therefore prompt assessment of blood pressure and consideration of occult cardiovascular disease rather than being attributed solely to environmental exposure.

Cold water is frequently present but is not an absolute requirement. It can increase peripheral vasoconstriction and central blood pooling, but SIPE has been described outside stereotypically cold conditions. Likewise, a wetsuit may contribute through external compression, yet wetsuit use is difficult to separate from water temperature and event type in observational datasets.

Overhydration is often mentioned because increased circulating volume could further raise cardiac filling pressures. The practical advice to avoid aggressive pre-race fluid loading is reasonable, but the evidence base is weaker than for age, sex, hypertension or previous SIPE. Athletes should not replace one extreme with another; avoiding deliberate pre-event fluid loading while maintaining appropriate hydration for the prevailing conditions is a more defensible approach.

Table 1. Risk factors and the strength of the current clinical signal.

Factor	Current interpretation	Clinical use
Previous SIPE / prior compatible symptoms	Consistent and clinically important recurrence signal.	Ask explicitly before open-water events and during post-event review.
Female sex and older age	Supported by large recreational cohorts.	Raises pre-test probability; not sufficient for diagnosis.
Hypertension / heart disease	Supported by cohort and mechanistic evidence.	Measure BP and consider cardiovascular evaluation after SIPE.
Asthma / recent respiratory infection	Emerging evidence, including 2026 case-control data.	May justify closer assessment, especially with recurrent symptoms.
Cold water / intense exertion	Strong physiological plausibility; common exposure pattern.	Useful for risk reduction but absence does not exclude SIPE.
Tight wetsuit / excessive fluid loading	Plausible, repeatedly suggested, but less securely quantified.	Discuss as potentially modifiable contributors rather than proven causes.

5. Clinical Presentation

The characteristic presentation is acute respiratory difficulty beginning during swimming or very soon after leaving the water. Cough and dyspnea are the dominant symptoms. Frothy sputum, hemoptysis or blood-tinged sputum may occur, and hypoxemia can be substantial [4, 11, 12]. Some athletes describe a sudden inability to maintain pace, a sensation that breathing has become disproportionately difficult, or panic that develops only after respiratory symptoms begin.

Physical examination is variable. Crackles support pulmonary edema but their absence does not rule it out. In the Navy Special Warfare cohort, a notable proportion of affected candidates had a normal initial chest examination, while others had crackles, wheeze or rhonchi [12]. Wheezing should therefore not automatically redirect the diagnosis toward asthma.

The course is often rapid after exit from the water, which can create false reassurance. Historically, SIPE was described as resolving within 24 to 48 hours [4]. More recent follow-up data suggest that symptoms may persist longer in a substantial minority: 38% of respondents

reported symptoms lasting more than two days, although most ultimately reported preserved or improved general health and physical activity [9].

The most dangerous consequence may occur before any of these findings are documented. An athlete who becomes hypoxemic and breathless while still in open water is at risk of being unable to self-rescue. For event organizers, recognition by lifeguards, kayakers and safety personnel is therefore as important as recognition by clinicians in the medical tent.

6. Event-Side Diagnosis

SIPE remains a clinical diagnosis supported by objective evidence of pulmonary edema. The most useful event-side study to date evaluated 160 swimmers presenting with acute respiratory symptoms during a large Swedish open-water event. Lung ultrasound confirmed pulmonary edema in 102. Oxygen saturation of 95% or lower was strongly associated with edema, while the combination of low saturation and crackles was highly specific [8].

Lung ultrasound is particularly attractive in event medicine because it is portable and can identify interstitial edema without moving a symptomatic athlete to radiology. B-lines should still be interpreted in context; they demonstrate increased extravascular lung water but do not by themselves prove that immersion caused it. The Swedish study also found that edema could be focal or unilateral, which is a useful warning against expecting a perfectly symmetric textbook pattern [8].

Chest radiography can support the diagnosis and remains useful when the athlete is transferred to hospital. The Navy cohort described interstitial radiographic changes and other features consistent with increased central vascular volume [12]. CT is rarely required simply to prove SIPE, but may be appropriate when pulmonary embolism, alternative parenchymal disease or another diagnosis remains a serious concern.

ECG and cardiac biomarkers are not diagnostic tests for SIPE, but they may be appropriate when chest pain, arrhythmia, significant cardiovascular risk or an atypical clinical course raises concern for another cardiac process. Echocardiography is particularly relevant when valvular disease, ventricular dysfunction or abnormal filling pressures are suspected [18]. Minor biomarker abnormalities after strenuous exercise should be interpreted in clinical context rather than treated as proof of acute coronary syndrome.

Table 2. Diagnostic tools in suspected SIPE.

Tool	Potential value	Limitation
Pulse oximetry	Rapid severity assessment; SpO ₂ ≤95% supported diagnosis in a large event cohort.	Affected by perfusion, motion and cold extremities.
Auscultation	Crackles increase suspicion and can be assessed immediately.	Normal examination does not exclude SIPE; wheeze may occur.
Lung ultrasound	Rapid demonstration of interstitial/alveolar edema; portable.	Operator dependent; B-lines are not specific to SIPE.
Chest radiograph	Documents edema and helps assess alternatives such as pneumonia.	Requires transport/equipment; abnormalities may resolve rapidly.
ECG	Assesses arrhythmia/ischemia and alternative cardiac causes.	Often nonspecific in isolated SIPE.
Troponin / NT-proBNP	Useful when myocardial injury or cardiac contribution is a concern.	Mild elevations may occur after strenuous exercise and SIPE.
Echocardiography	Assesses ventricular/valvular disease and post-event susceptibility.	Usually not required for immediate event-side diagnosis.

7. Differential Diagnosis

Acute dyspnea in a swimmer should not be labeled SIPE simply because the athlete was immersed. Aspiration, exercise-induced bronchoconstriction, anaphylaxis, acute coronary syndrome, arrhythmia, pulmonary embolism, infection and panic can produce overlapping symptoms. The history of symptom onset relative to immersion, objective hypoxemia and evidence of pulmonary edema help narrow the differential.

Aspiration is particularly important because coughing and radiographic opacities may overlap with SIPE. A witnessed choking or submersion event favors aspiration, but the two processes can coexist if SIPE causes distress in the water. Persistent fever or evolving focal infiltrates later in the course should prompt reconsideration of infection or aspiration-related lung injury. Exercise-induced bronchoconstriction can cause cough, wheeze and chest tightness, but does not explain pulmonary edema on lung ultrasound or radiography. Conversely, wheeze can occur in SIPE [12]. A known history of asthma therefore modifies probability but should not end the diagnostic process.

Cardiac disease becomes increasingly relevant with age. Three triathletes with an apparent SIPE presentation were subsequently diagnosed with Takotsubo cardiomyopathy [22]. This small case series is best interpreted as a diagnostic warning rather than an estimate of frequency: persistent chest pain, ECG abnormalities, marked biomarker elevation or significant echocardiographic dysfunction should shift attention toward a primary cardiac disorder.

Pulmonary embolism is uncommon in this setting but must remain in the differential when hypoxemia is persistent or disproportionate, particularly with pleuritic pain, thromboembolic risk factors or symptoms not resolving after removal from the water. SIPE usually improves relatively quickly; a course that does not fit should trigger diagnostic reassessment.

8. Acute Management

The first management step is immediate removal from the water. Continuing to swim despite progressive dyspnea increases both pulmonary stress and drowning risk. Once on land, management follows standard ABCDE priorities, with particular attention to oxygenation and work of breathing.

Supplemental oxygen is appropriate for hypoxemic athletes. Many cases improve rapidly with rest, warmth and oxygen after immersion ends. There is no evidence that routine diuretics are required in otherwise stable SIPE, and empiric medication should not distract from respiratory support and assessment for an underlying cardiac problem.

Positive airway pressure is a logical escalation when hypoxemia or respiratory distress persists. In a prospective observational study from the Swedish open-water program, prehospital CPAP or a positive expiratory pressure device was feasible and associated with improvement in oxygen saturation and clinical findings [23]. The study does not establish superiority over all other supportive strategies, but it supports the practical use of non-invasive positive pressure in selected patients.

Severely unstable patients require emergency transport and advanced respiratory support as needed. The threshold for transfer should be lower when hypoxemia persists, symptoms are

severe, the diagnosis is uncertain, chest pain or arrhythmia is present, or the athlete has significant cardiovascular disease.

Unlike exertional heat stroke, SIPE has no single time-critical disease-specific intervention analogous to cold-water immersion cooling. The management priority is therefore to stop exposure, support oxygenation and identify the subset in whom SIPE is a manifestation of or is complicated by another cardiopulmonary disorder.

9. Recurrence and Long-Term Course

Recurrence is one of the most important issues for athletes because the next episode may again begin far from shore. Earlier series already suggested recurrence, and systematic reviews identified previous SIPE as a major practical risk marker [10, 13]. The best contemporary follow-up data come from 165 Swedish cases identified at a large open-water event.

At approximately 30 months, 28% of participants reported recurrent respiratory symptoms during subsequent open-water swimming [9]. 58% had not returned to open-water swimming, which complicates interpretation and may lead to underestimation of recurrence among athletes who re-expose themselves. Asthma was independently associated with symptom duration beyond two days and with recurrent symptoms in that cohort.

The same study found that 93% of participants reported unchanged or improved general health and 85% reported unchanged or improved physical activity, which is reassuring. SIPE should therefore not be presented as inevitably causing chronic cardiopulmonary impairment. The main long-term problem is uncertainty about susceptibility and recurrence rather than established progressive lung damage in most athletes.

Normal dry-land pulmonary function after recovery does not eliminate recurrence risk [17]. This reinforces the idea that SIPE is an exposure-dependent hemodynamic phenomenon. A swimmer may appear entirely normal in clinic and still reproduce abnormal pulmonary vascular pressures under the combined stress of immersion and exercise.

10. Evaluation After an Episode

Post-event evaluation should be proportional to severity, age, recurrence and cardiovascular risk. At minimum, the clinician should document the circumstances of the episode, water temperature, distance, intensity, wetsuit use, fluid strategy, recent infection, previous similar symptoms, medical history and blood pressure.

A first episode in a young athlete who recovers rapidly and has no concerning history is different from recurrent SIPE in a 55-year-old swimmer with hypertension. In the latter situation, ECG and echocardiography are reasonable, and further cardiology assessment may

be appropriate. Structural disease, valvular dysfunction and coronary disease should not be missed simply because the episode occurred in water.

The extent of cardiac testing after an episode should depend on the clinical presentation. Persistent symptoms, ischemic chest pain, arrhythmia or abnormal echocardiography require conventional cardiovascular evaluation. The immersion-induced mitral-regurgitation study provides a specific example of a potential susceptibility mechanism that may only become apparent during immersion [18].

For divers, a 2024 joint position statement from SPUMS and the UK Diving Medical Committee recommends specialist evaluation after immersion pulmonary edema and takes a cautious position on future compressed-gas diving [24]. Competitive swimming is not identical to diving, but the statement is valuable because it emphasizes the search for cardiovascular predisposition and the seriousness of recurrence.

11. Prevention and Risk Reduction

Preventive recommendations rest on a weaker evidence base than diagnostic recommendations. Avoiding aggressive pre-event fluid loading, extreme cold exposure and an excessively restrictive wetsuit is physiologically reasonable after SIPE, but these strategies have not been validated in randomized prevention trials. Blood pressure and cardiovascular disease should be assessed and treated according to standard clinical practice.

A gradual warm-up may theoretically reduce abrupt hemodynamic stress, although high-quality clinical evidence is lacking. Likewise, acclimatization and greater open-water experience may be beneficial, but the 2026 association between limited open-water exposure and SIPE should not yet be interpreted as proof that training exposure prevents the condition [20].

Sildenafil is the most discussed pharmacologic preventive strategy because invasive hemodynamic data show reduced pulmonary vascular pressures in SIPE-susceptible subjects [7]. That mechanistic signal is intriguing but insufficient for routine prophylactic prescribing. The risks, contraindications and regulatory context of medication use in athletes require individualized medical assessment.

Counseling about prevention should make this uncertainty clear. Individual recurrence risk cannot currently be estimated with precision, so shared decision-making is more appropriate than a universal rule regarding future participation.

12. Return to Swimming and Endurance Sport

Return to sport after SIPE remains poorly standardized. Unlike many musculoskeletal conditions, there is no validated test showing that an athlete's susceptibility has resolved. Complete clinical recovery is essential, and persistent dyspnea, resting hypoxemia, abnormal lung findings or unexplained exercise intolerance should prompt further evaluation rather than immediate return.

Risk stratification should follow clinical recovery. Recurrent SIPE, severe hypoxemia, syncope, significant hemoptysis, hypertension, known cardiac disease or an abnormal post-event cardiac evaluation all justify a more cautious approach. The athlete should understand that recurrence can occur despite normal testing on land [9, 17].

For lower-risk athletes who choose to return, a graded strategy is reasonable: begin in controlled conditions, avoid solitary open-water swimming, use accessible safety support, avoid extreme cold and maximal early effort, and stop immediately if unexplained breathlessness or cough develops. These recommendations are pragmatic rather than trial-validated.

Return-to-sport counseling should also distinguish swimming from diving. The physiological stresses overlap, but compressed-gas diving introduces additional breathing-resistance and pressure-related factors. Guidance that advises against further compressed-gas diving after IPE cannot simply be transferred unchanged to recreational swimming [24].

13. Implications for Event Medical Planning

SIPE is an event-safety problem as much as a diagnostic problem. Medical teams may see the athlete only after rescue, so water-safety personnel should be trained to recognize swimmers who suddenly slow, become upright in the water, repeatedly stop, cough or appear unable to coordinate breathing. Early extraction may prevent progression to drowning.

At larger events, pulse oximetry, oxygen and the ability to provide positive airway pressure are useful resources. Portable lung ultrasound can add diagnostic confidence when trained operators are available [8, 23]. A predefined pathway for transfer is important for athletes with persistent hypoxemia, cardiovascular symptoms or uncertain diagnosis.

Data collection should be standardized. Recording water temperature, wetsuit use, distance, symptom onset, SpO₂, auscultation, lung ultrasound findings, treatment and disposition would improve comparability between events and help refine future risk estimates. Current epidemiologic uncertainty is partly a consequence of inconsistent case definitions.

14. Discussion

The evidence base for SIPE has developed considerably since the early case-report literature. Large recreational cohorts, military surveillance, lung-ultrasound studies, invasive hemodynamic experiments and long-term follow-up support a coherent clinical model in which immersion and exercise expose a susceptible pulmonary circulation to elevated hydrostatic pressures [6-9].

Several clinically important uncertainties remain. First, incidence is highly context dependent. A 0.44% incidence in a Swedish open-water event and a 5.0% burden in Navy Special Warfare candidates should not be treated as conflicting estimates; the populations and exposures are fundamentally different [6, 12]. Second, risk-factor data are improving but remain observational. Female sex, age, hypertension and previous symptoms have increasingly consistent support, whereas wetsuit compression and overhydration remain plausible but less precisely quantified. Earlier triathlon-specific data also illustrate the difficulty of separating exposure-related triggers from individual susceptibility [25].

It is also plausible that SIPE encompasses more than one clinical phenotype. Some athletes appear to have an exaggerated pulmonary vascular response despite normal dry-land testing, while others have hypertension, valvular disease or other cardiovascular abnormalities that become important during immersion [7, 18, 26]. Future research may benefit from separating predominantly vascular-susceptibility SIPE from episodes in which identifiable cardiac disease is a major contributor. A broader review of environment-induced pulmonary edema supports the concept that similar hydrostatic mechanisms can emerge under different combinations of environmental and cardiovascular stress [27].

Diagnostic evidence has also become more robust. The 2020 lung-ultrasound study is particularly relevant because it was performed during a real mass-participation event rather than in a laboratory [8]. Nevertheless, its criteria should be externally validated in other water temperatures, event formats and populations. Point-of-care ultrasound is operator dependent, and an algorithm developed in a well-resourced Swedish event may not translate directly to every race. The 2026 scoping review similarly identifies heterogeneity in diagnostic criteria and highlights the importance of pragmatic approaches that remain usable in resource-limited settings [28].

Treatment remains an area in which the evidence is comparatively sparse. Immediate exit from the water and supplemental oxygen for hypoxemia are supported by physiology and clinical experience. Prehospital positive airway pressure has encouraging observational data but has not been tested in randomized comparative trials [23]. Pharmacologic prevention with

sildenafil remains mechanistically interesting, yet the available hemodynamic study is too small to justify routine prophylaxis [7]. A 2025 treatment-focused review reaches a similar conclusion, emphasizing supportive care and the absence of a routinely validated pharmacologic preventive strategy [29].

Return-to-sport decisions are still poorly standardized after SIPE. The 30-month follow-up study provides valuable recurrence data, but many participants avoided subsequent open-water swimming, creating exposure bias [9]. Prospective evidence defining a safe, reproducible return protocol is still lacking. This concern is echoed in clinical commentary focused on the questions athletes most often ask after SIPE—diagnostic certainty, recurrence risk and prevention [30]. A recent triathlon-focused review likewise concludes that recurrence prevention and return-to-racing advice remain based largely on observational evidence and expert interpretation [31].

This review also has limitations. It is narrative rather than systematic, and the SIPE literature uses overlapping terminology that includes swimming-induced and immersion pulmonary edema. Studies of divers were included selectively when they clarified immersion physiology or post-event cardiovascular assessment, but diving-specific mechanisms cannot always be extrapolated to surface swimming. Much of the evidence remains observational, and several recommendations for prevention and return to swimming are therefore based on physiological rationale and expert judgment rather than randomized trials.

Despite these limitations, the available evidence supports a clear clinical message: sudden respiratory distress during open-water swimming warrants medical assessment and should not be attributed to poor conditioning or panic without considering SIPE and other cardiopulmonary causes. SIPE should be considered early, particularly when cough, crackles, hypoxemia or hemoptysis accompany symptoms. The athlete should be removed from the water, treated according to the severity of respiratory compromise, and subsequently assessed for factors that may influence recurrence.

15. Summary and conclusions

Swimming-induced pulmonary edema is an uncommon but clinically important cause of acute respiratory distress in endurance athletes. Immersion-related central blood-volume redistribution, exercise, cold-induced vasoconstriction and individual cardiovascular susceptibility interact to raise pulmonary vascular pressures and promote hydrostatic edema.

The diagnosis is primarily clinical, supported by hypoxemia and evidence of pulmonary edema. Lung ultrasound is particularly useful in event medicine when expertise is available. Immediate removal from the water is essential; oxygen and non-invasive positive pressure may be required

according to severity. Persistent symptoms, severe hypoxemia, chest pain, arrhythmia or an atypical course should prompt hospital assessment and investigation for alternative or contributing cardiopulmonary disease.

Recurrence is common enough to matter in return-to-sport counseling. After recovery, evaluation should focus on previous episodes, hypertension, cardiovascular disease, recent respiratory infection and the circumstances of exposure. Return to open-water swimming should be individualized, with explicit discussion of uncertainty and recurrence risk rather than a universal clearance rule.

Declaration of the use of generative AI and AI-assisted technologies in the writing process

During the preparation of this manuscript, the author used ChatGPT (OpenAI) to assist with language refinement, improvement of clarity and readability, and manuscript structuring and formatting. After using this tool, the author critically reviewed and edited the content as needed and takes full responsibility for the final content of the manuscript.

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