Swimming Induced Pulmonary Edema (SIPE)
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[Editor's note: What follows was born of a thread on our reader forum. Information sharing is a good thing, and often helps those with questions locate those with answers. Five real life, first-person stories are told below, interspersed with the best current information on SIPE. Thanks to our two authors, both practicing triathletes, both SIPE-stricken while racing. "Kat" gathered the chronicles. "Trey" explains the condition.]

KAT writes: The first time this happened to me was at the Mooseman half-Ironman race in June 2007. I began experiencing shortness of breath at 750m into the swim. I felt tightness in my chest - almost like an asthma attack, or that my wetsuit was too tight. Then, fluid began to build in my lungs and I developed a slight cough. I ended up doing the 'backstroke' for the last 750m of the swim in order to get to shore. I tried to keep racing and pushed through the complete bike leg, then had to stop at the beginning of the run as I was completely unable to get oxygen and was wheezing. That was 4 hours and 17 minutes into the event. I ended up in an ambulance on oxygen, and was released on site once my breathing improved.

The second time I had it happen was on July 22, 2007 at Ironman USA in Lake Placid. This time, it was much worse. Halfway into the swim, I experienced the same tightness in my chest and shortness of breath. I stopped at the 1km mark into the swim and loosened my wetsuit in hopes it might provide some relief. It didn't. I continued to swim and the gradual build up of fluid in my lungs began. I was struggling to stay above water in the last kilometer of the swim, and luckily there was a kayaker who spotted me in trouble and came to my aid. He could see I was in distress and told me he would stay with me all the way into shore. I swam the last leg of the swim by holding the kayak for air, and taking 10-20 strokes when I had enough oxygen to continue. I could hear the crowd and see the shore in the distance, but couldn't propel my body to get there. I felt as though I might drown. At the swim exit, I began coughing up pink frothy foam. Fortunately, the medical staff identified that there was a problem and pulled me from the race. They administered Lasix and oxygen on site and then transferred me to Saranac Lake Hospital by ambulance. Tests at the hospital confirmed that I had suffered pulmonary edema (fluid in my lungs), that it was not cardiac related. My heart was fine. The pink frothy foam was actually 'blood' that had seeped into my lungs.

Pulmonary edema occurs when proteins and red cells in the blood leak from the capillaries of the lungs into airspaces and other non-vascular lung structures.

“Flooding” of the lung in this way arises when the blood pressure inside the pulmonary capillaries exceeds the ability of the capillary membranes to contain that pressure. A pressure gradient across the capillaries can induce membrane failure when intracapillary pressure is particularly high, alveolar pressure is particularly low, or when the membranes themselves are damaged or have increased permeability.

Pulmonary edema resulting from abnormally high intracapillary pressures is usually caused by heart failure, and is termed “cardiac” or “cardiogenic”, while that caused by high negative airspace pressures, capillary hyperpermeability, or a variety of capillary / membrane stress abnormalities is considered to be "noncardiac" in origin.

Swimming Induced Pulmonary Edema (SIPE) is generally considered to be noncardiac in origin, although my personal bias is that in some people, especially those with underlying hypertension or diabetes, an element of cardiogenic etiology is also present.

Symptoms include marked respiratory distress, wet-sounding popping or crackling in the lungs with breathing, a "junky" rattling cough, and the hallmark; coughing up pink, frothy blood-tinged
sputum. When it occurs during exercise, one of the first sensations is shortness of breath that is substantially out of proportion to the effort being expended. The swimming induced variant of pulmonary edema known as SIPE is believed to occur from a combination of factors that creates what can be thought of as a “perfect storm” that leads to capillary leak. The incidence in triathlon is unknown, but estimates in controlled field military training settings have ranged from two to sixty percent.

The factor most characteristic of SIPE is water immersion; hence the name. Water immersion is believed to be important because water exerts a much larger force on bodily tissues than air does, and this increased pressure from outside the body squeezes blood from the skin, muscle, subcutaneous fat, etc., into the blood vessels of the circulatory system. Within the vascular space, water force compression of peripheral capillaries in the limbs drives a larger volume of the blood into the central circulation, including the large arteries and veins of the chest, such as the aorta and vena cava, the pulmonary artery and pulmonary vein, and the heart and lungs themselves. The weakest link in the central circulation are the pulmonary capillaries. When something has to give in an overloaded central circulation, the pulmonary capillaries are usually it.

**STEVE writes:** I was a competitive swimmer most of my life, including some very intense times in the pool. The first time I ever noticed the rasping, inability to catch my breath, and dramatic loss of energy in my limbs, was in Charleston, South Carolina, my first such incident.

The race started with a .6 mile swim in Atlantic ocean, no waves, water was warm - mid 70's. Three-tenths of a mile into swim, my arms felt like lead, my breathing became raspy. Another tenth of a mile later, I was holding the kayaks every 20 feet or so, trying to catch my breath. Another tenth, and I stopped moving and started sinking under the water. I was grabbed by a lifeguard who saw me go under and taken to shore - I was conscious, blood coming from my mouth. I spent 90 minutes on the shore while they tried to get my oxygen levels up. They did not leave for the hospital because they thought I would go into cardiac arrest at any moment. In the emergency room, they gave me Lasix and Heprin drips, took x-rays and blood tests. Twelve hours after being pulled from the water, the diagnosis was congestive heart failure - they readied me for an Angiogram. Twenty-four hours after, the angiogram showed I had no heart problems. They moved me to get a full body CT scan to find the clots. Forty-eight hours afterward, they pulled all the drips out of me, told me they had no idea what had happened, and let me walk out of the hospital.

Following the Charleston race, I had extensive cardio-pulmonary testing done, including a stress echo. Nothing was found. The cardiac specialist told me the incident in was likely a one-time occurrence. In preparation for my next tri, I did extensive pool training, and the night before the race I put on my wetsuit and went for a 25-minute swim in the Pacific Ocean, about 2 miles from where the start of the race would be the next morning. Everything was fine.

Two-hundred yards in I felt great, was at the head of the group, no problems. Just after I felt very tired, very fast, no rasping, but could not catch my breath, I reached for the kayak. After another 50 yards I pulled down my wetsuit top to try to get more air, and realized I was in trouble again. I called for the boat, and was taken from the water with blood coming from my mouth and nose. They wanted to call the ambulance, I told them a lie: it is just asthma. I did not want all the same tests. I got home, took a diuretic, went to bed.

Water immersion can't be the whole story, though, because if it were, people couldn't swim and certainly couldn't dive deep, where pressures are much higher than at surface swimming. Therefore, other contributing causes have to be present for SIPE to occur. Several of these other causes have been proposed in the medical literature, but no specific pattern has been identified. It appears, given our current limited knowledge, that assembly of a critical mass of any of the known risk factors can bring on an episode, and this varies by person and maybe even by episode.
Exercise appears to be an important determinant. All SIPE cases I am aware of have occurred during exercise (except SCUBA diver's pulmonary edema, but that's a bit different because of the much larger water pressures involved), and most of it is during strenuous exercise, as in the case of racing or time trial swimming. It has also been described with water running.

Cold causes peripheral capillary constriction in excess of what water pressure already causes. An influential early report of the syndrome suggested that people who develop SIPE have blood vessels that are abnormally reactive to cold. This finding has not been reproducible in other attempts to replicate it.

Overhydration can lead to short term expansion in plasma volume, especially if it's done so close to exercise start that the kidneys don't have time to take off the excess. Expanded plasma volume forced into the central circulation by water pressure could cause capillary breach. Similarly, habituation to hot weather training causes marked sustained plasma volume increases, and this could be troublesome by the same mechanism.

Trauma to the pulmonary capillaries can occur with unusually brutal training, large positive-to-negative pressure swings mid-race, significant water aspiration (breathing water, not swallowing it), maldistribution of pulmonary blood flow due to postural abnormalities, blood clots, etc., and capillary stretch or torsion. Racehorses are known to develop pulmonary edema from capillary trauma, although they are somewhat different from humans in that they are bred specifically to have hearts too big for their bodies. An argument could be made that human triathletes are self-selected to be at increased risk of high-flow capillary trauma on the grounds that outrageous cardiac output is needed for competition at the highest levels, but SIPE seems to affect people at all levels of the sport, so this relationship is unclear. SIPE does usually seem to develop within the first few minutes of a given effort, and there may be some brief period of highly augmented pulmonary capillary pressures that exists until the various systems all get entrained into a coordinated rhythm.

Hyponatremia, shock and other neurohumoral abnormalities can make the capillaries hyperpermiable and therefore leaky without the need for increased intracapillary pressure. What if any role this plays in SIPE is unclear at the present time.

Though not so much of an issue for triathletes, a more upright position in the water, swimming supine (backstroke) and swimming one lung down (sidestroke) without switching sides have all been implicated in the development of SIPE in Navy SEALS and similarly trained special forces commandos.

Wetsuit wearing is associated with SIPE, and it may be physiologically related, but wetsuits are so common in triathlon that their effect is difficult to separate from immersion. Several anecdotal reports exist of people feeling short of breath and clawing at their wetsuit tops so they can breathe, but this seems likely to be due to the feeling produced by the constricting neck of the garment as distress sets in. It makes more sense to me physiologically that if wetsuits are contributing to the problem, they are doing it by the same mechanism as the water: by compressing the lower extremities and forcing the blood volume towards the central circulatory apparatus.

Diastolic Dysfunction hasn't received much attention in the small (but growing) SIPE literature, but it is a well documented cause of dry-land pulmonary edema and classic “flash” pulmonary edema due to heart failure or extreme high blood pressure. Chronic (even mild) high blood pressure, diabetes and a number of other medical conditions can interfere with the ability of the heart's left ventricle to relax normally between beats. This inefficient relaxation can increase the pressure required to fill the ventricle for the next systolic ejection. Since blood returning to the heart from the lungs is what fills the left heart chambers, increased left heart filling pressures can cause increased pressures upstream in the pulmonary capillaries. Several recent reports in the
literature have indicated that some degree of diastolic dysfunction can be present in endurance athletes, and the apparent connection between high blood pressure and/or diabetes and SIPE in anecdotal reports suggest that these conditions may be contributory. An important feature of this mechanism - unlike many of the others - is that it is amenable at least in theory to treatment.

RAYLENE writes: The first time I experienced pulmonary edema with swimming was while wearing a wetsuit for the first time, training on the Wildflower triathlon race course. I had had pneumonia once, so I immediately knew what that feeling was. My boyfriend got me out of the water, and then I got on the bike and managed to do most of the Wildflower course. But I finally couldn't breathe biking up the hills anymore, so the boyfriend biked ahead and got the car. Everyone said it was just a reaction to wearing the wetsuit and being in cold water. I think everyone thought it was an asthma attack. I felt better in a couple of days - I found the web pages about the Navy Seals, and I decided I just needed to get used to swimming in cold water.

I bought my own wetsuit and gradually started swimming in the Pacific Ocean. Everything went well until my first triathlon.

I warmed up well, without any problems. Then, getting to the first buoy, I started to hear the rattling. I persevered, went off course because I couldn't think straight - no blood in the brain - and finally made it to shore. The doctor checked my pulse and decided I was ok, so I tried the bike ride. How I made it to the top of the hill I'll never know, because I can't remember much of it. I do remember lying on the sidewalk by my bike, gasping for breath.

My chip and bike were taken away from me, and I was sent to the medical tent. I was treated with an inhaler, even though the doctor watched me cough up pink foam for about 20 minutes. I went home.

SIPE, clearly, is not caused by any single factor. This is good and bad. It's bad because there's not a one-shot target for cure. It's good because there are multiple targets for prevention. While we will never be able to eliminate all the precipitating causes as long as triathlon involves a water event, we can take the three-legged stool approach. Take one leg away and the stool can't stand.

Of the suite of nine putative risk factors already mentioned, two are reasonable targets for prevention.

The first, hydration, is low hanging fruit. If you've had prior problems with SIPE and don't have the other modifiable factor below, don't be overly aggressive with hydration prior to the swim. If you pee a 20-30 second stream of clear urine at least once after your first-morning void prior to the swim you are well enough hydrated to do well in the water. Any deficit coming out will be small and you can catch it up on the bike. If you're going to take salt or other electrolyte supplements, don't start them until you're out of the water. As with everything, know your body, your mileage may vary, consult your doctor, and so on and so forth.

A class of drugs known as angiotensin converting enzyme II (ACE-II) inhibitors have been shown to improve ventricular relaxation in people with diastolic dysfunction and they have a high likelihood of being helpful in hypertensive triathletes with SIPE. Some suggestion has arisen recently that these compounds may be helpful in preventing exercise-induced pulmonary edema in Frankenstein-hearted racehorses, even though horses in their prime don't generally have diastolic dysfunction. ACE-II inhibitors also cause dilation of the small vessels of the lungs and may therefore play a role in reducing transcapillary pressures in high-demand situations even in the absence of diastolic dysfunction, so there may be some element of "right drug, wrong reason", with their use. I am stopping short of recommending them in humans without documented high blood pressure. Use of these medications should be considered only under medical supervision.
RED writes: I participated in this past Ironman Wisconsin and I experienced PE, possibly SIPE. Here is a brief recap of how things developed on that day. The 1:14 swim was okay but a bit slower than I had hoped, but with no breathing problems experienced that I can remember. My doctors asked me if I swallowed any water, but I didn't recall any choking or gagging during the swim.

The bike started out great and I felt pretty strong on the first loop. The second loop was another story. I probably went out a bit hard, but breathing problems complicated things even more. Somewhere between miles 70-80 I started experiencing shortness of breath, I was coughing a lot too and spitting the fluids that the coughing generated. I saw that there was some color in my phlegm, but going fast on the bike I couldn't tell if it was just tinged from the orange Gatorade I was drinking or foods I was eating.

I finished the bike and got changed for the run. I was feeling a bit out of it and had difficulty focusing mentally. Immediately after starting the run I knew something was wrong as my coughing had gotten worse and I was now sure that I was coughing up blood. I made it to mile 2 and found some medics to ask their advice. It was a tough decision after talking with them for a couple minutes because they can't help you unless if you concede to drop out of the race. I decided to try and go another mile, taking it easy. By this point I was unable to run because any deep breathing brought on fits of coughing. After a couple miles of walking I realized that I would be able to finish, but I wouldn't be able to run. I was able to go for short :30 second spurts at a jogging pace before I had to catch my breath and stop the coughing. I finally finished in 12:51 after a 5:37 mara-walk.

I went to the med tent afterwards to get checked out. After getting checked by a few different people they suggested I go to the ER after hearing "wetness" in my lungs. I was reluctant at first but conceded, rounded up my gear and headed to a local hospital in Madison. After telling my story ump-teen times to different doctors and nurses and getting blood work done they wanted me to stay for observation and to get an ECG (echo cardiogram - ultrasound of my heart) in the morning.

After the ultra-sound on Monday morning and several hours of waiting they told me that all my symptoms point to pulmonary edema, possibly induced during the swim.

SIPE was first described in highly trained young men with no measurable health problems. The occurrence of an attack does not imply in any way that the sufferer has heart or lung disease, or any other underlying illness for that matter.

Still, not everyone appears to be susceptible to it, which means it likely does not occur at random, but to date no test has been identified that can predict who is likely to have an episode and who isn't.

The good news about SIPE, to the extent that there is any, is that it appears to be largely self-limiting. When people get out of the water and stop exercising, it seems to get better mostly on its own. Supplemental oxygen is sometimes needed, and in severe cases diuretics such as furosemide (Lasix) may be necessary to draw down excess plasma volume.

A couple of cohort studies have been done now in closely observed military recruits, and no one has died or become disabled long term by SIPE in an observed episode. This does not mean, of course, that underlying health problems will not make a SIPE attack more serious, nor does it mean that hypoxemia and disorientation couldn't lead to drowning.

BURT writes: Very early on in the Ironman Kentucky swim I inhaled quite a bit of water into my lungs. I actually think this happened multiple times as my lung capacity felt diminished and resulted in quicker breaths. Once I hit the bike and my torso was in a prone position I began to have that gurgling sensation in my breathing. Throughout the bike I would get coughing fits and
actually would cough up 2-3 ounces of frothy fluid at a time. I could literally just let it run out of my mouth like I was spitting water.

I didn't feel the effects on my breathing while on the bike. I tried to keep my heart rate and breathing down but I'm sure there was an impact. By the time I hit the run my lung capacity felt like it was about a fourth of normal. I'd run 100 meters and be gasping for air, unable to catch a breath. As I continued the run I started paying more attention to what I was coughing up. There was definitely blood in it. I'm not sure if this was from my throat being so raw that it was bleeding or from the trauma to my lungs or both.

When I hit the finish line one of the volunteer nurses sat me in a chair for a few seconds and walked away. As I was sitting there I got another coughing fit and spit up about 4 ounces of blood and mucus. The nurse thought it was vomit and wanted to take me to the medics, assuming I was dehydrated. I told her it wasn't vomit. I was coughing up water and blood from my lungs.

If you've had a SIPE episode, you're at increased risk compared to your peers of having another one. Not everyone does, but recurrence is fairly common. If you do have a SIPE episode, whether racing or training, you should consider yourself done for the day. No good can come from trying to push through it, and it will not get better as long as you're in the water. If you get pulmonary edema in the water, get the hell out. You may survive if you push on, but you won't finish well that day, and it may take you longer to recover.

The decision about whether to pursue an aggressive work-up after the acute phase has run its course is a personal one. Very few physicians are familiar with the condition, and this may lead to incorrect attribution of the symptoms to other processes. It likely also will lead to extensive work-up that doesn't show anything abnormal, although the prospect that some other serious predisposing factor could be uncovered in a detailed work-up may make it worthwhile. If hypertension is present, proper treatment may have a significant impact on the risk of future episodes.

More research is needed.