

# Patient Voices

11<sup>th</sup>  
Edition



Diagnostic Odyssey:  
Difficult Journeys  
to Diagnosis

A publication of the American Thoracic Society  
**Public Advisory Roundtable**

 **ATS**  
American Thoracic Society



# ATS

## Public Advisory Roundtable

ATS Patient Voices is published by the American Thoracic Society Public Advisory Roundtable (ATS PAR). Since 2001, ATS PAR has been a core component of the Society and a mutually beneficial partnership wherein organizations that represent persons affected by respiratory diseases, illnesses requiring critical care, and sleep-related disorders collaborate with the ATS to advance their shared educational, research, patient care, and advocacy goals.

The ATS strives to improve health worldwide by advancing research, clinical care, and public health in respiratory disease, critical illness, and sleep disorders. The roots of the ATS reach back to 1905, when a small group of physicians and researchers began sharing information about tuberculosis. Since then, it has grown into an international society with more than 15,000 members.

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# Foreword



**Gregory Downey**  
**MD, ATSF**  
ATS President (2022-2023)



**Karen Collishaw**  
**MPP, CAE**  
CEO, ATS

Since 2001, the ATS Public Advisory Roundtable (PAR) has helped to highlight the patient experience and to weave patients into the fabric of the American Thoracic Society. Along with PAR partners, comprised of various patient advocacy groups, PAR has opened up the Society to include those most personally affected by the diseases we research and treat: the patients and their families.

Throughout the year, PAR and PAR partners promote awareness, public education, advocacy, and research. The outcomes of those presentations are expansive and stretch from inspiring investigators to shaping ATS advocacy programs.

Patient Voices is a particularly important part of this effort. This special edition highlights a critical issue when seeking care, misdiagnosis and

underdiagnosis, by focusing on people who had a long and difficult road to finding an answer. Their stories highlight an imperative in comprehensive medical care: we must listen carefully to the thoughts and concerns of each and every patient. To quote one of this year's patients, "the rarest diseases require the rarest doctors – the ones who take the time to really listen."

The stories illustrate how important collaboration is to quality care, and we're grateful to the contributors for sharing their experiences. We also share in their hope that this edition will raise awareness about lung disease and its far-reaching effects for those impacted.

Thank you for making Patient Voices possible as we work together toward ever more treatments, therapies, and ultimately, cures.

# Chair's Message



**Michele Manion**

Chair, ATS-PAR

Since 2001, the American Thoracic Society leadership has formally partnered with patients and their families through the ATS Public Advisory Roundtable (PAR). PAR continues to be one of the only patient-centered groups woven into the fabric of a medical membership association. As the patient arm of the Society, PAR is a central component of the ATS providing the patient perspective in all aspects of the organization.

Each year, ATS PAR identifies patients to participate in an edition of Patient Voices, with the goal of sharing their journey – to put a “face” to their diagnosis and challenges. As a result, respiratory professionals including physicians, clinicians, scientists, and researchers receive an intimate look into a disease’s impact on patients’ lives. Understanding the patient perspective is essential for Society members to innovate and to advance scientific research toward better patient outcomes.

This edition, ATS Patient Voices 11, was created to highlight the voices of patients who had difficult journeys to diagnosis and had to overcome many hurdles to get vital care. These stories are critical to understanding the complexities that still exist in treatment for those living with these life changing diagnoses.

Today, we continue to align our work with the unique and urgent needs of individuals living with sleep disorders, critical illness, and respiratory diseases. Together, we’re creating a more unified and powerful research and advocacy community. As our patients gain more knowledge and insight about their diseases and the effect on their quality of life, they remain invaluable resources to us all.

It is a great honor and privilege for the ATS PAR to serve as the “patient voice” of the Society.

# Caitlyn & Elizabeth Hilton

## Children's Interstitial Lung Disease



Caitlyn was born at 36 weeks and 3 days, weighing a healthy 5 pounds. About 15 minutes after birth, Caitlyn was blue, floppy and having trouble breathing as she was rushed to the NICU, where she would spend the next 10 days on oxygen, struggling with feeds. As a seasoned mom I could not shake the feeling something was wrong, and I begged the doctors to do an ultrasound to see why Caitlyn would scream every time I sat her up. They agreed, and found a mass in her abdomen, which we were told was likely cancer.

The next day, Caitlyn went into surgery. It was not cancer but a torsed ovary and necrotic fallopian tube, both of which were removed. During the recovery period, Caitlyn experienced what would be the first of several respiratory arrests, which was attributed to her premature birth and small size.

After two weeks, Caitlyn came home. She caught one respiratory virus after another and would land in the PICU requiring oxygen. The first year of her life I remember thinking that she was frequently sick. I discussed my concerns with her doctors and was told it was because she was a preemie.

At 13 months, Caitlyn shut her finger in a door, resulting in a cut that became infected. She was treated with antibiotics, and it healed. Two weeks later, Caitlyn developed a targetoid rash, and was treated for ring worm. She was placed on topical steroids which made the lesions worse. They would heal but reappear every

few days. With these lesions, her eyes would swell, and she would scream for hours each day, vomit, refuse to eat, and often had blood in her urine.

We were referred to a dermatologist, who found small vessel vasculitis. We were sent to a number of specialists who didn't know what to do and after a series of repeat respiratory infections, respiratory failures, and PICU admissions, we sought care out of state at the top children's hospital.



We were desperate for answers. I will never forget the day we met the rheumatologist who said “I believe you. There is absolutely something very wrong, and I promise you we will help.” I felt like I could breathe again! We saw this physician a few times, and she referred us to a pulmonologist.



While we were waiting to see the pulmonologist, then two-year-old Caitlyn got another respiratory infection. I took her to our local hospital, and again, she needed oxygen. I was assured that she was ok, and it was just a case of premie lungs and a cold. She was admitted and despite me repeatedly bringing up that Caitlyn was seeming to struggle to breathe and her O2 saturations were 89-90 percent despite oxygen support, I was told it was fine.

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“We were desperate for answers. I will never forget the day we met the rheumatologist who said ‘I believe you. There is absolutely something very wrong, and I promise you we will help.’”



By the third day I was very concerned. She was no longer able to stay awake and had a high fever. Again, I was told she was fine. A few hours later, she was not fine - they couldn't get her oxygen levels to come past 70 percent despite the 15 liters of oxygen. They called for an ambulance for emergency transport to a children's hospital with a PICU.

This PICU took away all her medication, despite me begging them not to. Within 12 hours, Caitlyn declined even more, and I was told she may not survive. She was on the maximum respiratory support, still struggling to breathe, and had a 108.4-degree fever. She went into respiratory arrest.

Somehow, she pulled through. The day she was discharged I called the pulmonologist and begged them to get her in right away. They got her in the very next day! We walked in and met this amazing physi-

cian who asked me how Caitlyn slept, how often she needed oxygen, and a series of other questions. We went in for a chest x-ray, CT scan, bronchoscopy, and sleep study.

Her chest CT showed interstitial lung disease, but unfortunately her first lung biopsies have not provided us with what type of ILD she has.



Caitlyn's symptoms continue to worsen. She has been put on BiPAP at night, has developed metabolic respiratory lactic acidosis, renal tubular acidosis, and has had a G-tube placed due to failure to gain weight and grow. She has been evaluated for a lung transplant but ultimately was denied. Genetic testing shows nlrp12 and spinocerebellar ataxia 13, but this still has not led to a confirmed diagnosis or treatment plan. Caitlyn's lung disease continues to progress, she is now on five liters of O2 and has frequent illnesses. I am heartbroken that no one can help my little girl and am terrified that I am going to lose her.

## Children's Interstitial Lung Disease

Children's Interstitial and Diffuse Lung Disease (chILD) is a group of rare lung diseases found in infants, children and teens. There are many types of chILD:

- Some types of chILD are genetic and passed through families. Some types are caused by an environmental or infectious trigger. Some have an unknown cause. More and more genetic causes for chILD are discovered as we learn more about genes.
- ChILD symptoms start at different ages. Some types affect babies while others affect older children. Some types of chILD run in families.
- Interstitial lung disease can be hard to detect and even harder to diagnose. Because of this, if your child has symptoms of interstitial lung disease, they should be seen by a pediatric lung specialist doctor (pulmonologist). There is no single test to diagnose chILD, since each type of chILD is different. The lung specialist will choose which tests to order based on your child's symptoms.



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# Conor & Carey Kauffman

## Primary Ciliary Dyskinesia

Conor was born a full-term, 9-pound 4 ounce “healthy” baby boy. His birth was very quick, so his inability to get rid of a tiny bit of congestion seemed, according to the doctor, to be related to some residual amniotic fluid in his system that may not have been ‘squeezed out,’ but nothing to be alarmed about. After staying with us the first night, we were concerned about a grunting noise he was making when trying to breathe. Just as a precaution, he was taken to the nursery to check his oxygen levels. We were told not to worry, and that lots of babies start life with some breathing and oxygen problems. They assured us that it almost always works itself out with no trouble.

Upon discovering his low oxygen level, they took him to the neonatal intensive care unit (NICU) and ordered the chest x-ray that revealed that all of Conor’s organs were completely reversed – a condition called situs inversus. After the



doctors told us that they had never seen a case before, they informed us that there was a 25 percent chance that Conor had an underlying condition called Kartagener Syndrome, which we now know is a subtype of primary ciliary dyskinesia (PCD). From that point, we heard a number of comments from the doctors that were concerning, if not alarming, in light of what would happen a few hours later:

- *“Even if he has it, you won’t need to worry about it until later in his life. It doesn’t manifest itself until later.”*
- *“Donny Osmond has situs inversus totalis – and he’s just fine!”*
- *“Don’t worry – he’ll just have a snottier nose than the other kids. All kids have runny noses.”*

Once we had a name for what he might have, we started frantically researching anything we could find on our phones. From even the little information we could find, we realized that what we were hearing about PCD was not the reality of the disease. It wasn’t even close. It was clear to us that Conor could be on a lifelong mission to prevent lung destruction aside from other PCD-related complications. He clearly wouldn’t just be another “snotty nose kid.”



In fact, I remember my husband saying, “Oh my God – this could be more like cystic fibrosis.” It was a very cold, isolating yet very confusing feeling to realize that these very experienced doctors in a state-of-the-art hospital might not know what we are dealing with. Unfortunately, we were right. Conor passed away the next day, shocking every doctor and nurse involved in his care. His lungs collapsed, causing his heart to shut down, and they couldn’t save him.

The doctors said that they had never seen or heard of anything like it – a perfectly healthy, robust baby boy rapidly deteriorating to the point where he couldn’t be saved - and couldn’t tell us what happened.

“Do you want an autopsy?” they asked. “Yes. And I want him tested for PCD! That’s what he had, I know it,” I replied. “PCD wouldn’t be seen as a ‘cause of death,’” the neonatologist replied, but agreed to do it because I was so serious about it. The autopsy came back citing acute pneumonia which again shocked the doctors who had taken multiple clear x-rays of his chest and had been unable to grow bacteria on tracheal samples when he was alive. They had also given him two antibiotics that would have addressed 90 percent of any infections out there. He tested positive for PCD.

Ultimately, we can get our minds around the cause of death. However, if your cilia don't work, they don't work from day one. ***Conor went into a fight with one arm swinging and he lost.*** He couldn't fight the infection like a 'normal,' healthy baby. We aren't doctors and have no medical training, but that made sense to us.

He's now the first baby on-record having died this way in the world, but we know he's not the first, and not the last. We cannot track what we do not measure.

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“It was a very cold, isolating yet very confusing feeling to realize that these very experienced doctors in a state-of-the-art hospital might not know what we are dealing with.”

I do know that Conor's situation is uncommon, and I'm not sure he could have been saved – maybe he had a more severe form of PCD? Or maybe the pneumonia he got would have been too much for any child? Or maybe if he had gotten through the first couple of days, he'd be here right now slugging away with his daily treatments? But we don't really know, and never will.

Through the PCDF and the voices and talents of everyone touched by this disorder, doctors can be more informed, patients can live healthier lives and perhaps one day PCD will no longer need to be on any radar anywhere in the world.

## Primary Ciliary Dyskinesia

Primary ciliary dyskinesia (PCD) is a rare, inherited, genetic disorder of motile (moving) cilia. Cilia are tiny hairlike structures on the cells in the body. Motile cilia perform an important role in the nose, ears, and airways within the lungs, working to remove unwanted inhaled particles and germs. PCD causes frequent respiratory infections starting at a very early age that result in lifelong, progressive lung, sinus and ear disease. People with PCD benefit from early diagnosis and treatment to hopefully limit permanent lung damage.

- PCD is an inherited disorder, meaning that people born with the disease receive a mutated (abnormal) gene from both parents. In PCD, mutations in the genes responsible for building cilia and controlling their function result in cilia that do not work effectively.
- Dyskinesia, or impaired movement, is the most common ciliary defect seen in PCD. Other defects may lead to not having enough cilia on each airway cell, which can also cause the clinical symptoms seen in PCD.



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# Erica Courtenay -Mann

## Sarcoidosis

A decade ago, I was sick. I went to the doctor and told her “I feel really, really tired.” This was the second time I had been in her office in six months. She looked at my neck and said, “Your neck is not supposed to be that big,” to which I replied, “I’m just getting fat. It’s baby weight.” It wasn’t just baby weight. My baby was actually two years old, going on three. At that point, I knew I was really sick. They looked down my throat and saw two large growths. By the time I was on the table for the surgery, those two growths had multiplied to six. Little did I know they were granulomas. Here I was a working Black American mother with a sweet little toddler that woke up from a laryngoscopy with one less vocal cord and a Sarcoidosis diagnosis, tasked with finding out what Sarcoidosis was and what this meant for me, my life, and my motherhood.





They told me I had Sarcoidosis, but what I heard at first was that I was going to die. I knew nothing of Sarcoidosis, but I knew everything about the barriers to high quality care Black Americans faced in the health care system. They told me I had Sarcoidosis and all I could hear was that I was fighting a disease that “shape shifted” to each individual, and that I was not going to win this fight.

Sarcoidosis turned my life upside down for a long while. As a mom to a three-year-old, I was really scared. I’m whole-heartedly convinced the diagnosis was the death knell to my marriage. The diagnosis put me into a long period of fear, denial, and depression. I didn’t know where to turn for help, at first. Then, I had my first flare. It took me down for three days. First, I lost some mobility in my legs. Then, when I finally got back home with my daughter, I lost mobility in my hands. In desperation, I had to call my co-parent to come get our daughter, because I couldn’t care for her at that moment, a moment where I started to wonder how I would manage this disease. I was in Texas far away from my relatives, with a small support system, with work and parenting responsibilities, and trying to navigate this new life I had with Sarcoidosis.



No choice or knowledge of clinical trials contributed to my fears after my diagnosis. African American women may have an understandable distrust of the healthcare system and an understandable distrust of clinical trials, and this is only compounded when a patient has experienced non-equitable care empirically, when clinical trials are limited, or when providers and clinical trials do not accommodate the work logistics and financial needs of the patient. This was the case for me. They told me I had Sarcoidosis and now I had to work overtime to reclaim a new baseline with my health outcomes and my socio-emotional outlook on living with a chronic disease. I'm really glad I had many specialists and my primary care physician to help see me through.

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“The diagnosis put me into a long period of fear, denial, and depression.”

Over the past decade, I have educated myself about Sarcoidosis. I have worked with my healthcare providers to come up with a plan. I have moved from fear of Sarcoidosis to walking hand-in-hand with Sarcoidosis. I knew I had to fight to live every time I heard my daughter laugh, or she was excited about an achievement at school, or something brought her joy. It is in those moments I dig so very deep to stay focused on managing this disease.

I feel it is my duty to raise awareness about Sarcoidosis and its impact on Black American women. I feel it is my duty to raise awareness that encourages providers to stop using a one-size-fits all approach to treating Sarcoidosis. Black women are three times more likely to develop Sarcoidosis than white women and men, and are more likely to be hospitalized, or even die from sarcoidosis when compared to other groups. As a Black American woman living with Sarcoidosis, I want providers to see me as more than a data point. I want to be seen as a partner in a process that seeks to improve the quality of life of those living with Sarcoidosis.

## Sarcoidosis

Sarcoidosis is a disease of unknown cause in which inflammatory cells clump together and form tiny lumps of cells in various organs and tissues of the body. Sarcoidosis most often affects the lungs and its hilar lymph nodes but can also involve other areas of the body including the eyes, skin, sinuses, liver, kidneys, brain and heart.

- When sarcoidosis affects the lungs (pulmonary sarcoidosis), the disease can reduce the amount of air the lungs can hold and cause abnormal stiffness, called “restriction,” of the lungs. This results in breathing problems that can interfere with daily activities.
- Since sarcoidosis can affect one or more parts of the body, the signs and symptoms depend on the tissue/organs involved. Some people with the disease do not have any symptoms and it may be noticed by chance when they are being seen for other problems. Other people may be hard to diagnose because the symptoms they have are not very specific. But certain clinical features such as the erythema nodosum, rash or eye findings may lead a healthcare provider to suspect sarcoidosis.



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# Jeanie Hanley

## Cystic Fibrosis



I was diagnosed with Cystic Fibrosis late in life at 33, along with three of my eight siblings. In my childhood I was frequently diagnosed with sinusitis and bronchitis that always came back after antibiotic therapy. CF wasn't well recognized in people of color or different ethnicities, such as my Mexican-American heritage. I decided to become a doctor to find out what was wrong with my sister, Theresa, and me. She was the sickest of all of us.

Up to and throughout medical school, I would have sinus surgeries, two episodes of pneumonia and partially collapsed lungs (atelectasis) and too many courses of antibiotics. I was given the diagnosis of "atypical allergies and asthma" that was refractory to the usual asthma treatments except for oral steroids, which led to multiple courses, sometimes for months at a time.

During my medical training, I realized that the progressive respiratory issues plaguing my siblings and I was much more serious than just asthma. A microbiology class exercise where we coughed into an agar plate showed I grew the bacteria *Pseudomonas*. The professor took a look at the agar, a look at me, and deemed I looked fairly healthy so it must be a contaminant and not to worry about it. In another class, we had been learning about CF and how *Pseudomonas* is a common bacteria in CF lungs. Knowing my family's health issues, I did worry.

At my next doctor's visit, I mentioned this finding and asked if I could have CF. Unequivocally, he said no way – that I was too healthy-looking and too old, since the usual diagnosis occurs before seven years of age. He added that it only occurs in those with Northern European backgrounds, not in Mexican-Americans/Hispanic population. I learned I was the wrong ethnicity. He did diagnose me with “Medical Student Syndrome,” found in medical students who think they have every disease they learn about.

In later training in Pediatrics and then in my Allergy subspecialty, I hoped other colleagues, allergists and pulmonologists could help me on my quest to figure out the underlying cause of my issues. Eventually I was diagnosed with ABPA for which I took more courses of steroids. However, it didn't explain my constellation of symptoms or those of my sister or other two siblings who also had recurrent respiratory infections and significant GI issues. Everyone I asked about CF said it wasn't possible for the same reasons I'd heard before.

When I didn't recover fully after a course of antibiotics, oral steroids, and daily asthma inhalers, I was told that I was being a "typical doctor" and not taking the medicines as prescribed. I was desperate to get better and did all that was prescribed in the hopes that it would help, but I was on my own.

After more episodes of atelectasis and hemoptysis, I had extensive workups to find another cause for the bronchiectasis that is common among people with CF, yet all tests and biopsies were negative. I had learned from my mother that one of my sisters had a negative sweat chloride test when she was a child. Being the gold standard for diagnosis for CF, it presented a sliver of doubt. I also learned that my brother discovered he had bilateral absence of the vas deferens (CBAVD) and couldn't have children. The mountain of evidence was growing when considering myself and my family's progressive symptoms, yet I still couldn't prove it.



In 1994, I attended an allergy conference and presented my family to a CF specialist (shout out to Richard Moss, MD, of Stanford) who encouraged me to obtain genetic testing, which had recently become available, on my entire family.

At Christmas time, I swabbed the buccal mucosa of all my siblings and asked a geneticist to order the testing, which checked for six CF mutations. The results showed that my mom, five sibs and I carried one F508delta mutation. I was very excited and presented the results to the geneticist. Yet he didn't bat an eyelash as he stated it was the normal carrier frequency in the community. He felt he was reassuring me by saying I was too old and too healthy-looking to have CF. It did give him pause, though, that the F508del mutation could occur in someone of Mexican-American descent.

Soon after, I came across a publication by a prominent researcher about two sisters diagnosed with CF as adults and whose sweat chloride was negative. I was incredulous and contacted the researcher. His research lab immediately offered to test the blood of my entire family including my parents. Again, at Christmas time, I drew all my siblings and parents' blood and sent them off. After two months, the lab confirmed that four of us had CF due to two CFTR mutations - F508delta and a rarer Class IV mutation.



Rather than being devastated, my sister Theresa and I were excited to start receiving the correct treatment and hopefully improve the quality of our lives. My other two siblings found the diagnosis harder to accept and didn't seek treatment at a CF Center for many years.

Only after this diagnosis would I learn that I could've been diagnosed with CF without genetic testing. Although my sweat chloride would be negative just like my sister's, another test for CF, usually performed at research centers like Stanford, called a "nasal potential difference" was found to be abnormal.

Unfortunately, the lifelong lack of proper treatment affected all four of my siblings and me. It led to more progressive bronchiectasis. Theresa had the worst disease and despite the appropriate CF therapies, she passed away eight years after being diagnosed from Cepacia syndrome – a CF complication and fatal bacterial lung infection that spread throughout her body. It's painful to think that an earlier diagnosis for her might have helped her live long enough to receive CFTR modulators that have helped me so much.





I also try not to predict what would have happened had I received proper treatment earlier. I was very sick during my allergy training, working full time, requiring constant antibiotics and steroids, nebulizing during commutes and at home, trying desperately to clear secretions. My CF has led to two bronchial artery embolization procedures to stem the flow of life-threatening hemoptysis, multiple sinus surgeries and yearly hospitalizations up until several years ago with the advent of CFTR modulators.

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“Unfortunately, the lifelong lack of proper treatment affected all four of my siblings and me. It led to more progressive bronchiectasis.”

My quality of life improved by leaps and bounds when I was fortunate enough to qualify for CFTR modulators, life-saving pills that jumpstart the mutated CFTR proteins and force them to function. They have improved my health to levels I hadn't known since I was a child. Even though I have to continue with all daily CF nebulizer and inhaled treatments to this day, my new level of health has enabled me to return to work as an allergist in private practice after over a decade on medical leave.

Spreading the word that CF does not discriminate and can occur in people of all colors, all ages, all countries and even in those with negative sweat chlorides is critical. Unfortunately, about 10 percent of all those with CF do not qualify for CFTR modulators due to having rarer mutations that modulators can't correct. Many are people of color and of non-European ethnicities, who may not be detected at birth by newborn screen. I suspect that the combination of my CF mutations would not have led to CF being diagnosed at birth by newborn screening. For this reason, I'm certain there are children and adults walking around with undiagnosed CF who will need the medical community's help to diagnose them.

## Cystic Fibrosis

Cystic Fibrosis occurs when a person inherits a mutated (abnormal) copy of the CFTR (cystic fibrosis transmembrane conductance regulator gene) from each parent. It is an autosomal recessive disease meaning only people with two CFTR mutations have the disease. While there is no cure, life expectancy has steadily improved in the United States. Some other facts about Cystic Fibrosis are:

- There are now more adults than children with CF in the United States.
- Newborn screening for CF done on blood samples can identify most children before one month of age, which allows for early treatment and disease monitoring.
- CF individuals have abnormally thick mucus, which blocks the airways (obstruction) and leads to repeated infections and damaging inflammation in the lungs. Treatments are directed at trying to prevent and treat these problems.



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# Jon Hagstrom

## Alpha-1 Antitrypsin Deficiency



I have Alpha-1 Antitrypsin Deficiency, “Alpha-1” for short, a rare genetic disease that can affect the lungs and/or the liver. I want to share my own experience with this condition, as it highlights the human cost of late detection, which is all too common for people with my disease.

As the condition is genetic, I’ve had it since birth. But there is no newborn screening in place to catch it, and I made it through early childhood without any symptoms that might have precipitated an early diagnosis.

As was common in the late 60s and 70s, I grew up surrounded by second-hand smoke. Had we known about my condition, that might have been avoided, or maybe my parents would have discovered their own elevated risk of lung disease and quit smoking - they both carried a single allele for the disease, resulting in higher risk for lung and liver disease than normal, though far less

than I had with my two alleles. In my teenage years I added first-hand smoke to the mix, a habit I maintained through my 30s. That certainly would not have happened if I knew about the special risks I had as a person with Alpha-1.

When I was 13, I had blood work done which showed significantly elevated liver enzymes. Today that might have led to testing for my condition. In the early 80s there was even less awareness of the disease, and I went undetected.

In my 20s, an aunt who had never smoked died relatively young from lung issues. Under a more rigorous detection regime, that might have set off a cascade of testing in the family, warning me before it was too late.

Finally, in my late 30s my breathing was bad enough that I decided to see a pulmonologist. I then had lung function (FEV1) of only 29 percent of normal. Based on my age and the severity of lung damage I had, my pulmonologist tested me on my first visit. I remember him saying “There’s this odd disease you probably don’t have but I’m going to test you for it anyway...”

I was lucky to have been diagnosed on my first-ever visit to a pulmonologist. The average lung-affected Alpha sees three different medical professionals and goes six to seven years between first reporting lung issues and finally getting diagnosed.

Upon testing positive for Alpha-1, I started weekly intravenous infusions of the Alpha-1 Antitrypsin protein – then and now the only therapy approved for Alpha-1 lung disease. It does not cure the disease, and it doesn't reverse existing lung damage, but it can slow the further decline of lung function. But by the time I started it, too much damage had already been done. I can't know for sure whether augmentation slowed my decline but if so, it didn't slow it much - within six years of diagnosis I was referred for a lung transplant.

Such was my disease course - the bulk of my decline happened before I even knew I had the disease. Even if a cure had existed and had been given to me immediately upon diagnosis, at 29 percent lung function, I still would have lived a very constrained life, with many normal activities beyond my reach.



My story is not atypical. Indeed, most people with my condition never get diagnosed at all. Some liver-affected Alphas get diagnosed at autopsy. This is unacceptable considering testing has been available for decades, and technologies such as direct-to-consumer genetic testing is only making it easier.

This is a problem we need to fix. At the individual patient level, time is critical. This disease can move quite fast, as my own story confirms. In 2010, I had a high energy, demanding lifestyle, working at an investment bank, and splitting my time between London and New York. Nobody looking at my life from the outside would have ever thought of me as sick.

A mere three years later I was officially disabled. I no longer carried my passport and briefcase. Instead, I dragged an oxygen concentrator with me

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“Indeed, most people with my condition never get diagnosed at all.”

everywhere I went, including to my son's kindergarten orientation. He called it "Daddy's special air". I had to hire someone to play catch with him because I couldn't do it myself. That tremendous change happened in just three years, between 2010 and 2013. But it continues. Three years after that, my lung function was at 14 percent as I was wheeled into an operating room for a bi-lateral lung transplant. I walked out of the hospital with a six-year life expectancy and a seven-year-old boy waiting at home. We need to do better for the Alphas who come after me. And one of the ways to do that - earlier detection - is something we can do now.

## Alpha-1 Antitrypsin Deficiency

Alpha-1 antitrypsin deficiency is a genetic condition that decreases lung protection resulting in an inherited form of emphysema. People with the condition, also known as AAT Deficiency or alpha-1 antitrypsin deficiency, do not have enough of a protein called alpha-1 antitrypsin (AAT) in their blood. This protein is made in the liver, and it protects the lungs so they can work normally.

- Alpha-one antitrypsin deficiency is an inherited condition. Every person inherits two AAT genes—one from each parent. Inheriting two abnormal AAT genes causes very low levels of AAT in the blood.
- AAT deficiency lung symptoms usually appear after age 30 but may emerge earlier or much later. The first symptom is usually shortness of breath during daily activities. Other symptoms include wheezing and decreased ability to exercise.
- AAT deficiency only can be detected through blood tests. One type of blood test measures the body's level of AAT. If the AAT level is lower than normal, your healthcare provider may order a genotype or a phenotype blood test.



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# Pat Owens

## Chronic Obstructive Pulmonary Disease (COPD)



I am 78 years old and have very severe COPD. I have had asthma since I was six years old. They wouldn't let me run and play or do sports, for fear I would have an asthma attack, so growing up I would sit and watch my friends and siblings play.

I was diagnosed with Chronic Bronchial Asthma in 1998. I was put on disability and had to quit working. I smoked for 42 years and with the help from my Lord I quit (cold turkey) after having a bout with pneumonia. I was put on oxygen in 2002. In 2007, I was diagnosed with exercised induced asthma and emphysema (the doctors say I have "three whammies"). In 2016, I had pneumonia. I was put on a ventilator for four days and spent two weeks in the hospital. After coming home, with exercise, diet, and breathing exercises I went off oxygen for two years. I now only use it with exertion and when I have flare ups.



I live by myself so it is very important to take care of myself so I can continue to stay as independent as possible. When I go for a walk, I use my umbrella stroller, like the ones they use for children. I put my oxygen M9 tank and my purse in it, because the M9 tanks weigh 10lbs alone. When I take my groceries into my house, again I use my stroller. I also stock up on my groceries for when I might be sick and can't go to the store. It takes a while to clean my little house, but I get it done, though not all at once.

It's very important to have family and friends to be able to depend on, and I also have several hobbies to focus on when I get sick. I do love my Bible study and do it just about every day. I also have six wonderful children, along with their awesome spouses, 28 grandchildren, and 10 great grandchildren. None of them live close to me. They all live in either cold states (my lungs don't like the cold) or in higher elevations (my lungs don't like that, either). They come to visit me, and I talk to at least one every day. If I need them, I just call, and they are here. I also have some wonderful friends that help me any time I need it. My Lord has blessed me so much as to giving me the tools and knowledge of how, what, and where to go to get through flare ups and everything else in my life.

I wish medical personnel would explain more to their patients about the ways they can help themselves. Some people give up when they get a diagnosis, and don't do anything for themselves because it's hard to breathe. They are not told that exercise gets easier as they continue with it. I have gotten most all of my knowledge of this dreaded disease through my own research and the COPD Foundation, and I am one of the Arizona State Captains for the COPD Foundation.

It is very important to keep busy and do what you can. I exercise in my home when I can't go out because of the weather or if I have a flare up. I have joined the gym for the use of more equipment. I keep busy as I can, I volunteer in the community I live in to help people with whatever I can do for them. I also do whatever I can to give people knowledge of COPD. I can't fly anymore because the pressure in the cabins on the airplanes is set at about 8000 ft elevation and I can't do high altitudes even with oxygen, so when I travel, I put my O2 in my car and away I go. I haven't traveled since COVID-19 began but hope to again when things calm down. I feel at my age I deserve to do whatever I can and want to do!

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“I wish medical personnel would explain more to their patients about the ways they can help themselves. Some people give up when they get a diagnosis, and don’t do anything for themselves because it’s hard to breathe.”

## Chronic Obstructive Pulmonary Disease (COPD)

Chronic Obstructive Pulmonary Disease (COPD) is a preventable and treatable lung disease. People with COPD must work harder to breathe, which can lead to shortness of breath and/or feeling tired. Some other facts about COPD are:

- Although the most common cause of COPD is tobacco smoke, there are several other factors that can cause or make COPD worse, including environmental exposures and genetic (inherited) risk.
- Common symptoms of COPD include feeling short of breath while resting or when doing physical activity, cough, wheezing, fatigue, and/or mucus production that does not go away.
- Some general classes of medications to treat COPD include those that aim to widen the airways (bronchodilators), reduce swelling in the airways (antiinflammatory drugs, such as steroids), and/or treat infections (antibiotics).



**Learn more**

ATS Patient Education Series

# Rebecca Stanfel

## Sarcoidosis



Getting diagnosed with sarcoidosis when I was 34 years old—and a brand-new mother—left me reeling. An urgent care doctor in my small Montana city told me I probably had lymphoma after seeing my abnormal CT scan. It was a very long two weeks until I got an appointment with a pulmonologist across the state. Still, when I learned that I had some immune disease instead of lymphoma, I felt hopeful.

A few weeks later, while I was out for a neighborhood walk pushing baby Andrew in his stroller, I passed out. I had no idea how long I had lain in the street, out cold, with my child unattended.

My pulmonologist referred me to a research hospital a couple of states away. I had a battery of tests: a cardiac MRI, a signal average EKG, an echocardiogram. The electrophysiologist (EP) told me I had cardiac sarcoidosis and that I could

“drop dead at any moment.” I still get gooseflesh and a pain deep in my gut when I even think of his words now, 18 years later, and picture Andrew waiting for me back at the hotel with my parents.

The question that my medical team wanted to address was whether I should have a defibrillator implanted immediately and be started on high-dose steroids, or just start on steroids. At the time, the protocol was to use a right heart catheterization to help decide. If the EP could stimulate (potentially fatal) ventricular tachycardia while I was safely being monitored in the surgical lab, they would implant the defibrillator then and there. If not, I was judged not to be at any real risk and wouldn't need a defibrillator. I passed my cath lab test that day and began a years' long course of high-dose steroids (80 mg. and above).

However, my heart didn't get the message that all was well. I spent the next few years passing out without warning. Each of these episodes of syncope required I return to the cath lab that was a long day's drive from my home.

I began what felt like an increasingly absurd and surreal roster of tests. A different EP theorized that perhaps exertion combined with my underlying cardiac sarcoidosis caused my issues so, I went into my fourth right heart cath unmedicated, and was handed light dumbbells midway through the procedure and instructed to carefully do some chest pressed and biceps curls while they tried to stimulate v-tach. As usual, my heart behaved itself in the lab.

I hadn't yet learned the most important question any patient should ask when being scheduled for a test. Will the results of this test change my treatment or prognosis? I had good insurance and wanted to be a good patient. I believed that if I followed all the doctors' instructions, I would get to live to see my son grow up.

What followed were tilt table tests, blood gas tests, EMGs, more heart cath, PET scans, MRIs. I gained 100 pounds on prednisone, and none of these tests provided a definitive answer. More than one doctor blithely suggested I get a heart biopsy. By then I'd learned enough to ask questions and discovered that even such an extreme procedure wouldn't provide definitive results.

I changed doctors. Early in my first appointment he asked how my defibrillator was managing my cardiac sarcoidosis.

Defibrillator?

I had been caught between protocols. Newer research showed that all my hours in the cath lab without getting v-tach were just that—hours. He told me I was lucky not to have died.

A week later, I had an AICD implanted in my chest, and I've grown accustomed to it pacing and occasionally shocking my heart back into normal rhythms. It saved my life.

I don't blame those doctors who had me lifting weights while in surgery. With so few cardiac sarcoidosis patients, they did the best they could with the limited data they had. I'm extremely fortunate not to have ended up a data point disproving their theory.

But I am left with strong feelings about all the testing I endured. The irony is that in all my cath lab exams, in being flipped around on a tilt table and spun around in a chair with flashing lights until I vomited is that I was both over and under treated. In my doctors' quest for surety, my life ended up at stake.



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“In my doctors’ quest for surety, my life ended up at stake.”

Testing and then re-testing patients with diseases like mine often don’t bring surety, only more questions (that require more tests). My goal isn’t to appear, anonymous, in a medical journal. Now I am a patient advocate for the Foundation of Sarcoidosis Research. I want to live as fully as I can with sarcoidosis. For this to happen, I need my doctor to ask herself how the test will improve the patient’s treatment and quality of life before ordering the test.

## Sarcoidosis

Sarcoidosis is a disease of unknown cause in which inflammatory cells clump together and form tiny lumps of cells in various organs and tissues of the body. Sarcoidosis most often affects the lungs and its hilar lymph nodes but can also involve other areas of the body including the eyes, skin, sinuses, liver, kidneys, brain and heart.

- When sarcoidosis affects the lungs (pulmonary sarcoidosis), the disease can reduce the amount of air the lungs can hold and cause abnormal stiffness, called “restriction,” of the lungs. This results in breathing problems that can interfere with daily activities.
- Since sarcoidosis can affect one or more parts of the body, the signs and symptoms depend on the tissue/organs involved. Some people with the disease do not have any symptoms and it may be noticed by chance when they are being seen for other problems. Other people may be hard to diagnose because the symptoms they have are not very specific. But certain clinical features such as the erythema nodosum, rash or eye findings may lead a healthcare provider to suspect sarcoidosis.



**Learn more**

ATS Patient Education Series



# Sarah Jones

Vasculitis



Before I begin to tell parts of my story, I have to say that my heart is full of gratitude for the health care team that I currently have. Their hard work and dedication are appreciated far beyond the gifts, notes, and flowers that I can bestow upon them. My heart is full.

And.

It has taken two years to assemble them.

With two Master's Degrees, one in Public Administration with a health care focus, and one in Strategic Design, I have always lived my life steering toward positive outcomes. No one would ever call me a shrinking violet, but the process of diagnosis and post-diagnosis care has tested that.

For 12 months I struggled to get an answer to my sudden severe asthma, expansive new allergies and recurring bronchitis, pneumonia, and hives. The process was demoralizing, exhausting and painful.

On three separate appointments, a pulmonologist diagnosed me with GERD and prescribed erythromycin. His only recommendation was surgery for a hiatal hernia that didn't exist. (And I don't have GERD.)

Following that I began to see an asthma/allergy doctor. He said my recurring pneumonia was due to allergies. Four visits to him resulted in only antibiotics, and steroids. I questioned the hives, telling him that according to my own research, they looked like vascular hives. This was never addressed.

During my eighth case of bronchitis, I went to the allergy/asthma office – again. The NP I saw was the first one to take my blood pressure, and she promptly sent me to the Emergency Room. My BP was 210/117, I had a cough and shortness of breath. The doctor did a COVID-19 test, x-ray and sent me home. When I inquired about my BP – he said, “It is just nerves, go home and just take it again in the morning.” I did. It was 224/128; my BP would continue to be unmanaged for the next eight months. This was the exact same emergency room that my brother went to with a BP of 230/120 and was promptly admitted and tested for days. In my opinion, sexism in health care is alive and well.

Several weeks later, I had bronchitis for the ninth time. This time we drove to a new ER, but again, the COVID-19 test defined my care. The doctor was furious. He said, “We just can’t help people who need testing and evaluation during a pandemic.” He was quick to prescribe another round of antibiotics and steroids, and I was out the door.



Upset, I began studying my lab and EKG history. If the doctors were not going to look at it, I was. I noted my abnormal EKGs, elevated D-Dimer, chronic shortness of breath, cough, bronchitis, vascular hives, and my eosinophils at three times the normal range. Several weeks later my bronchitis returned and was clearly pneumonia – again.

Desperate, and armed with my history, my wife drove me two hours to the medical center in a nearby city. We were not doing this dance with doctors that would not listen and did not care.

James Komara, D.O, the ER physician at the Mayo Clinic, was the first person to really listen. I can still hear his voice, “You are a very sick woman. We will figure this out.” It was the first time I had felt seen and taken seriously. He diagnosed pneumonia, noted the extensive inflammation in the lungs, flagged the eosinophils, injected steroids for hives, admitted me and ordered a skin biopsy. Mayo’s curious culture tested every system and ordered a skin biopsy, which led to the diagnosis of EGPA.

Finally.

Diagnosis is not the end of the story for a person with a rare disease, but it is an essential step in the journey of dealing with a life-threatening disease.

In March 2021 I woke up with a 103 fever and pulse/ox in the 80s. That week in the hospital was the first hospitalization since receiving the EGPA diagnosis. We showed up and immediately asked for a rheumatology consult due to my condition and gave everyone information on EGPA. The hospitalist refused the rheumatologist request and had the pulmonologist stop in. He proclaimed that I was fine.

On day four, it took my refusal to take any more medications and a complaint to the hospital against the hospitalist to get a rheumatological consult. The rheumatologist knew to order ABGs, which then resulted in a diagnosis. This five-day hospital stay could have been one to two days if they had listened to us.

In the last several months I saw my new pulmonologist. My lungs looked good, but I was still short of breath. He took that opportunity to tell me at least six times I was overweight. I was humiliated. I had been on steroids for 16 months, of course I had gained weight. My wife, exasperated, asked him if my breathing issues could be tied to the fact I could not breathe through my nose. That comment led to a three-and-a-half-hour sinus surgery and a clear nasal passage, for the first time in years. The EGPA was directly responsible for the sinus issues.

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“Diagnosis is not the end of the story for a person with a rare disease, but it is an essential step in the journey of dealing with a life-threatening disease.”

To the doctors reading this, thank you. Thank you for caring and showing up every day! And please, look at more than labs and scans. Please listen closely and respectfully with a curious mind.

Eighteen months into this disease, I am thrilled to have doctors who partner with me, listen, and seek to understand. It helps the daunting future seem less formidable.

## Vasculitis

Vasculitis is a general term that refers to inflammation of the blood vessels. It is used to describe a family of nearly 20 rare diseases, characterized by narrowing, weakening, or scarring of the blood vessels, which can restrict blood flow and damage vital organs and tissues. Vasculitis can affect any of the blood vessels of the body, including arteries, veins, and capillaries. Symptoms depend on the organs and tissues affected and can vary from person to person. Early diagnosis and treatment are extremely important to avoid potentially life-threatening complications.

- Vasculitis is classified as an autoimmune disorder, which occurs when the body's natural defense system mistakenly attacks healthy tissues. Triggers may include infection, medication, genetic or environmental factors, allergic reactions, or another disease. However, the exact cause is often unknown.
- Most forms of vasculitis are chronic, with periods of relapse and remission. In addition, medications used to treat vasculitis carry the risk of side effects, so follow-up medical care is essential.



**Learn more**

Vasculitis Foundation

# Thespena Panos

Lymphangi leiomyomatosis  
(LAM)



For seven years, I knew something was wrong. I had a hard time breathing and started coughing up blood clots with exertion or exercise. I went to several pulmonologists, but no one could find anything. On a family trip to Greece in 2014, I had a severe episode while swimming. I couldn't breathe, I became dizzy and almost passed out. I spent two days in bed unable to move. The doctor thought it was an asthma attack and gave me an inhaler. When I came back home, I had a CT scan, and everything seemed normal. I saw another pulmonologist who told me there was nothing wrong, and that I just had to lose weight. With despair, I tried to explain that I had had these issues even when my weight was normal. How could I exercise when I couldn't breathe and kept coughing up blood clots? I distinctly remember him saying, "I don't know what to tell you...just lose weight." I left heartbroken and ashamed.

Deep down I knew this was more than a weight issue, but after a couple of weeks of being down on myself, I decided to get a personal trainer. During my initial assessment, I could only climb a step for about a minute before having to sit down gasping for air. The oximeter showed my oxygen level was 82. I sat until my breathing returned to normal and the oximeter showed 98. The trainer thought the oximeter was broken, but I knew something was severely wrong. I bought my own oximeter, monitored myself and kept a detailed log of all my mysterious health issues.

It wasn't until late 2016 when an internal medicine doctor, a good friend of mine, truly listened to my cries for help. I went to see him for what I thought was a bad bout of bronchitis. After an extensive conversation, he walked out of the exam room, passed a room with a pregnant woman waiting to see him, and suddenly remembered reading a question about LAM the year before when he took his recertification exam. He admitted me to the hospital the following week for tests, and a CT scan showed I had hundreds of cysts in my lungs. Turns out, there had been cysts on my 2014 scan, but they were missed. I was sent down for a bronchoscopy and the procedure went terribly wrong. I ended up on a ventilator.



The first thing I read was that life expectancy was 8-10 years. I was devastated at the thought that I may only have a year or two left, especially being a single mother to my two beautiful daughters on my own, Sophia and Eva. Until I knew more details, I decided to hold off telling them the name of the disease because they would have immediately turned to the internet. Despite my fears, my first job was still to protect my daughters and make sure they were living their best lives.



I learned about the LAM Foundation and called them for support. They gave me a wealth of information, resources, clinical trials and treatment options, and the latest medical research. They also referred me to the closest LAM clinic in a nearby state. However, my insurance wouldn't allow me to cross state lines to see the LAM specialist.

After continually getting denied, I contacted my congressman to help me fight the state's department of insurance to allow me to be seen at the out-of-state medical center that could provide me with effective treatment. After four months of appeals, I finally was approved for a bronchoscopy, but it ended up being inconclusive, so the next step was an open lung biopsy. In May 2017, it was FINALLY confirmed that I have lymphangioleiomyomatosis, LAM.



I was put on a drug that was approved by the FDA to treat LAM just seven years ago and is helping women live longer than 8-10 years with a better quality of life. Unfortunately, LAM has already destroyed my lungs to a point where I will continue to need supplemental oxygen most of the time, for the rest of my life.

The hardest part for me is knowing that if I had been diagnosed sooner, I would not require oxygen today. It took me a few years, but I can finally talk about my diagnosis without crying. I went from deep despair to renewed hope and a determination to live life to the fullest. I am truly blessed to have my family and friends by my side, especially my daughters, my fiancé, and my parents. They've been my rock, my strength, my hope...and I am so grateful for all their love and encouragement. This meandering journey of misdiagnosis or delayed diagnosis MUST stop. We need new therapies and greater awareness about LAM. We need to find a cure!

## Lymphangiomyomatosis (LAM)

Lymphangiomyomatosis, also known as LAM, is a rare lung disease that mainly affects women, usually during their childbearing years. LAM is caused by mutations in the tuberous sclerosis complex (TSC) genes. These mutations lead to growth of abnormal cells that spread by the blood stream and make their way into the lungs. Once in the lungs, these cells create holes in the lung tissue (called cysts) that can weaken breathing and the ability to take up oxygen.

- Elevated VEGF-D levels can help confirm the diagnosis of LAM without needing a lung biopsy.
- LAM causes multiple air-filled holes, called cysts, in the lungs. Often these cysts can rupture and cause air to leak outside of the lung, leading to lung collapse
- There is a possibility that pregnancy may lead to progression of LAM, so consult your doctor if you are pregnant or considering pregnancy



**Learn more**

ATS Patient Education Series

# Epilogue



**Courtney White**

Senior Director, Patient Outreach,  
Equality and Engagement

For those in roles with high patient-engagement, such as clinicians, or patient advocates, the patient is never far from mind.

No matter how many medical advancements we make, we will always have things to learn from patients. The experience of living through, or living with, a disease like many of our patients have faced, has made them well-equipped to remind us of the realities of survival.

Taking the time to read their stories reminds us that to them and to their communities, they are not an asthma patient, or a COPD patient, or even a lung cancer survivor. They are a parent, a friend, or a neighbor who has asthma, or COPD, or who has beaten lung cancer. Their disease does not define them, even when it does define their daily lives.

We remain grateful to the patients who share their stories with us, and who remind us that life with these diseases is more than possible – it's critical. They remind us that every milestone is important: every treatment that makes their lives a bit more normal, every intervention that makes breathing a bit easier, allows them to focus less on their disease, and more on their lives. By hearing their stories, we can inform our own work – where do they see a need for innovation? What do they see from their proximity to the disease?

Patient Voices is a great way to remind ourselves of patients' expertise in their own disease and treatment, and once a year isn't enough. That's why the ATS, in conjunction with PAR partners, dedicates specific patient education weeks to individual diseases throughout the year. During those times we bring patient advocacy groups together with expert clinicians and researchers to shed light on disease and treatment and facilitate a public conversation. We talk about the existing state of treatment, as well as where treatments are headed. By connecting our members and PAR partners, the ATS not only highlights the patient experience, but also encourages collaboration as researchers are able to connect with the many resources our partners offer, from grants to patient registries.

Thanks to input from all stakeholders including, patients, families, clinicians, scientists, and researchers, we can continue to move forward, together. ●

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# Patient Voices

A publication of the American Thoracic Society  
**Public Advisory Roundtable**

“I wish medical personnel would explain more to their patients about the ways they can help themselves. Some people give up when they get a diagnosis, and don’t do anything for themselves because it’s hard to breathe.”

**-Pat Owens**