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I have been practicing emergency medicine for 30 years. In 1994 I invented an imaging system for teaching intubation, the procedure of inserting breathing tubes. This led me to perform research into this procedure, and subsequently teach airway procedure courses to physicians worldwide for the last two decades.

So at the end of March, as a crush of Covid-19 patients began overwhelming hospitals in New York City, I volunteered to spend 10 days at Bellevue, helping at the hospital where I trained. Over those days, I realized that we are not detecting the deadly pneumonia the virus causes early enough and that we could be doing more to keep patients off ventilators — and alive.

On the long drive to New York from my home in New Hampshire, I called my friend Nick Caputo, an emergency physician in the Bronx, who was already in the thick of it. I wanted to know what I was facing, how to stay safe and about his insights into airway management with this disease. “Rich,” he said, “it’s like nothing I’ve ever seen before.”

He was right. Pneumonia caused by the coronavirus has had a stunning impact on the city’s hospital system. Normally an E.R. has a mix of patients with conditions ranging from the serious, such as heart attacks, strokes and traumatic injuries, to the nonlife-threatening, such as minor lacerations, intoxication, orthopedic injuries and migraine headaches.

During my recent time at Bellevue, though, almost all the E.R. patients had Covid pneumonia. Within the first hour of my first shift I inserted breathing tubes into two patients.

Even patients without respiratory complaints had Covid pneumonia. The patient stabbed in the shoulder, whom we X-rayed because we worried he had a collapsed lung, actually had Covid pneumonia. In patients on whom we did CT scans because they were injured in falls, we coincidentally found Covid pneumonia. Elderly patients who had passed out for unknown reasons and a number of diabetic patients were found to have it.

And here is what really surprised us: These patients did not report any sensation of breathing problems, even though their chest X-rays showed diffuse pneumonia and their oxygen was below normal. How could this be?

We are just beginning to recognize that Covid pneumonia initially causes a form of oxygen deprivation we call “silent hypoxia” — “silent” because of its insidious, hard-to-detect nature.

Pneumonia is an infection of the lungs in which the air sacs fill with fluid or pus. Normally, patients develop chest discomfort, pain with breathing and other breathing problems. But when Covid pneumonia first strikes, patients don’t feel short of breath, even as their oxygen levels fall. And by the time they do, they have alarmingly low oxygen levels and moderate-to-severe pneumonia (as seen on chest X-rays). Normal oxygen saturation for most persons at sea level is 94 percent to 100 percent; Covid pneumonia patients I saw had oxygen saturations as low as 50 percent.

To my amazement, most patients I saw said they had been sick for a week or so with fever, cough, upset stomach and fatigue, but they only became short of breath the day they came to the hospital. Their pneumonia had clearly been going on for days, but by the time they felt they had to go to the hospital, they were often already in critical condition.

In emergency departments we insert breathing tubes in critically ill patients for a variety of reasons. In my 30 years of practice, however, most patients requiring emergency intubation are in shock, have altered mental status or are grunting to breathe. Patients requiring intubation because of acute hypoxia are often unconscious or using every muscle they can to take a breath. They are in extreme duress. Covid pneumonia cases are very different.

A vast majority of Covid pneumonia patients I met had remarkably low oxygen saturations at triage — seemingly incompatible with life — but they were using their cellphones as we put them on monitors. Although breathing fast, they had relatively minimal apparent distress, despite dangerously low oxygen levels and terrible pneumonia on chest X-rays.

We are only just beginning to understand why this is so. The coronavirus attacks lung cells that make surfactant. This substance helps the air sacs in the lungs stay open between breaths and is critical to normal lung function. As the inflammation from Covid pneumonia starts, it causes the air sacs to collapse, and oxygen levels fall. Yet the lungs initially remain “compliant,” not yet stiff or heavy with fluid. This means patients can still expel carbon dioxide — and without a buildup of carbon dioxide, patients do not feel short of breath.

Patients compensate for the low oxygen in their blood by breathing faster and deeper — and this happens without their realizing it. This silent hypoxia, and the patient’s physiological response to it, causes even more inflammation and more air sacs to collapse, and the pneumonia worsens until oxygen levels plummet. In effect, patients are injuring their own lungs by breathing harder and harder. Twenty percent of Covid pneumonia patients then go on to a second and deadlier phase of lung injury. Fluid builds up and the lungs become stiff, carbon dioxide rises, and patients develop acute respiratory failure.

By the time patients have noticeable trouble breathing and present to the hospital with dangerously low oxygen levels, many will ultimately require a ventilator.

Silent hypoxia progressing rapidly to respiratory failure explains cases of Covid-19 patients dying suddenly after not feeling short of breath. (It appears that most Covid-19 patients experience relatively mild symptoms and get over the illness in a week or two without treatment.) A major reason this pandemic is straining our health system is the alarming severity of lung injury patients have when they arrive in emergency rooms. Covid-19 overwhelmingly kills through the lungs. And because so many patients are not going to the hospital until their pneumonia is already well advanced, many wind up on ventilators, causing shortages of the machines. And once on ventilators, many die.

Avoiding the use of a ventilator is a huge win for both patient and the health care system. The resources needed for patients on ventilators are staggering. Vented patients require multiple sedatives so that they don’t buck the vent or accidentally remove their breathing tubes; they need intravenous and arterial lines, IV medicines and IV pumps. In addition to a tube in the trachea, they have tubes in their stomach and bladder. Teams of people are required to move each patient, turning them on their stomach and then their back, twice a day to improve lung function.

There is a way we could identify more patients who have Covid pneumonia sooner and treat them more effectively — and it would not require waiting for a coronavirus test at a hospital or doctor’s office. It requires detecting silent hypoxia early through a common medical device that can be purchased without a prescription at most pharmacies: a pulse oximeter.

Pulse oximetry is no more complicated than using a thermometer. These small devices turn on with one button and are placed on a fingertip. In a few seconds, two numbers are displayed: oxygen saturation and pulse rate. Pulse oximeters are extremely reliable in detecting oxygenation problems and elevated heart rates. Pulse oximeters helped save the lives of two emergency physicians I know, alerting them early on to the need for treatment. When they noticed their oxygen levels declining, both went to the hospital and recovered (though one waited longer and required more treatment). Detection of hypoxia, early treatment and close monitoring apparently also worked for Boris Johnson, the British prime minister.

Widespread pulse oximetry screening for Covid pneumonia — whether people check themselves on home devices or go to clinics or doctors’ offices — could provide an early warning system for the kinds of breathing problems associated with Covid pneumonia. People using the devices at home would want to consult with their doctors to reduce the number of people who come to the E.R. unnecessarily because they misinterpret their device. There also may be some patients who have unrecognized chronic lung problems and have borderline or slightly low oxygen saturations unrelated to Covid-19.

All patients who have tested positive for the coronavirus should have pulse oximetry monitoring for two weeks, the period during which Covid pneumonia typically develops. All persons with cough, fatigue and fevers should also have pulse oximeter monitoring even if they have not had virus testing, or even if their swab test was negative, because those tests are only about 70 percent accurate. A vast majority of Americans who have been exposed to the virus don’t know it. There are other things we can do as well to avoid immediately resorting to intubation and a ventilator. Patient positioning maneuvers (having patients lie on their stomach and sides) open up the lower and posterior lungs most affected in Covid pneumonia. Oxygenation and positioning helped patients breathe easier and seemed to prevent progression of the disease in many cases. In a preliminary study by Dr. Caputo, this strategy helped keep three out of four patients with advanced Covid pneumonia from needing a ventilator in the first 24 hours.

To date, Covid-19 has killed more than 40,600 people nationwide — more than 10,000 in New York State alone. Oximeters are not 100 percent accurate, and they are not a panacea. There will be deaths and bad outcomes that are not preventable. We don’t fully understand why certain patients get so sick, or why some go on to develop multi-organ failure. Many elderly people, already weak with chronic illness, and those with underlying lung disease do very poorly with Covid pneumonia, despite aggressive treatment. But we can do better. Right now, many emergency rooms are either being crushed by this one disease or waiting for it to hit. We must direct resources to identifying and treating the initial phase of Covid pneumonia earlier by screening for silent hypoxia. It’s time to get ahead of this virus instead of chasing it. ENDS