Persons with asthma have numerous symptoms with which they have to deal, whether it’s shortness of breath, chest tightness, a noisy chest, or troublesome cough. At the same time nasal allergies can cause nasal congestion or rhinitis, sneezing, or frequent throat clearing from post-nasal drip. In addition, we frequently hear complaints about a hoarse voice. It often comes and goes and can be a considerable frustration, especially for those who do a lot of speaking in their work. The voice quality changes; people notice that their voice doesn’t sound the same, and sometimes it seems to require a greater effort to generate a normally loud voice. What is causing this problem on top of everything else?

There are a number of potential causes, just as in persons without asthma, such as trauma from repetitive coughing, gastroesophageal reflux disease (GERD) causing stomach acid to splash onto the vocal cords, or vocal cords polyps. But an important consideration in persons with asthma is hoarseness as a side effect from use of inhaled steroids. Examples include the inhaled steroids taken alone (fluticasone, budesonide, beclomethasone, mometasone, ciclesonide, and fluticasone) as well as the inhaled steroids taken in combination with long-acting bronchodilator medicines (fluticasone/salmeterol, budesonide/formoterol, and mometasone/formoterol).

We routinely tell our patients to rinse their mouth after using these inhalers in order to prevent a candidal throat infection or thrush. One cannot, however, rinse down to the level of the vocal cords without choking or aspirating. Some of the steroid medication that one inhales settles on the vocal cords on its way down onto the bronchial tubes, with the possibility of causing irritation and voice weakness. This is an adverse side-effect of the steroid medication — an “inhaled steroid-induced laryngitis.” It is more common when the medication is delivered by metered-dose inhaler rather than dry-powder inhaler, and it is probably more common when the dose of medication is higher. It occurs with all of the inhaled steroids, but not commonly with inhaled bronchodilators alone (such as albuterol), so it seems to be an effect of the medication, not the propellant or powder being inhaled.

No one knows exactly in what way the inhaled steroids affect the vocal cords to cause hoarseness. Some have
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thought that they cause a myopathy of the laryngeal muscles; others have thought that there is irritation to the overlying mucosa. Occasionally, one can find candidal infection of the vocal cords. The inhaled steroids do not cause neoplastic changes or permanent injury to the vocal cords.

Inhaled steroids are the cornerstone of long-term asthma treatment. They reduce symptoms, make the airways less hypersensitive to the triggers of asthma, and help to prevent asthma attacks. Their increasingly widespread use is probably the reason for the reduction in asthma hospitalizations and deaths observed in the United States over the last 2 decades. Hoarseness is a frustrating side effect that affects some people who use these highly-effective medicines, even when they are doing everything right in their use.

What can be done? If you can safely omit use of the inhaled steroid for a period of time, voice quality will return to normal. Sometimes it takes only a few days, sometimes longer. One can try adding a spacer to the metered-dose inhaler or switching from a metered-dose inhaler to a dry-powder inhaler for delivery of the inhaled steroid. There is no good evidence that one inhaled steroid has fewer effects on the voice than any other, although there is some theoretic rationale to suggest that ciclesonide (Alvesco) might cause less hoarseness (because it is delivered as a pro-drug that is activated by esterases located mainly in the bronchial mucosa. If hoarseness is severe and persistent, direct inspection of the upper airway by an otolaryngologist is indicated, to exclude alternative diagnoses. Meanwhile, we continue to seek better medicines to treat asthma -- effective and free of frustrating side effects.
Suddenly Unable to Breathe

A young man came to see me the other day complaining of several episodes of frightening shortness of breath. The first episode developed quickly one day when he was leaving work. He had been tired that week, perhaps with early symptoms of a respiratory tract infection or perhaps his allergies were acting up, but his distress seemed to come on “out of the blue.” Quite abruptly, he recalled, he couldn’t breathe. His symptoms improved relatively quickly, such that by the time he arrived home 30 minutes later he felt all better, although frightened by such a severe attack.

He experienced several similar events over the next few weeks, many waking him from his sleep. He had no prior history of asthma, although he had a history of mild seasonal rhinitis. He experienced occasional postnasal drip and had no symptoms of heartburn to suggest gastroesophageal reflux. He had never been told of asthma as a child, and he was a lifelong non-smoker.

When questioned more about his difficulty breathing, he was quite clear: he simply could not get air in or out of his chest. It was not that it was hard to empty the air from his chest, he said, it was that no air would move at all. He was given an albuterol inhaler to try, but found it difficult to use and in truth had not tried it.

He reported only minimal cough, no sputum production. He had not experienced wheezing, but recalled a respiratory sound that he made as his episodes gradually resolved. His wife thought that she too had heard a breathing noise, particularly when he tried to breathe in. They have two cats at home but noted no increased likelihood of symptoms when around the cats. In the absence of these attacks, he felt well and was able to work out at the gym without limitation due to his breathing. His only medications were vitamin D and glucosamine chondroitin.

His chest examination was normal. Chest X-ray was normal. Spirometry performed at a time when he felt well was likewise normal. And the question was: is this asthma?

Asthma causes symptoms that come and go. Between attacks one can feel entirely well with a normal chest exam and normal lung function. However, the history that this young man offered was atypical in several ways, including no prior history of asthma; sudden severe attacks that came on without warning and resolved within a few minutes without treatment; and his sense that during these spells it was not hard to breathe, but impossible to breathe at all – no air movement in or out at all. As the episode abated, there came an inspiratory sound; and when asked if he could localize the site of his distress, he offered that he thought his problem was more in his throat than in his chest.

The diagnosis? Not asthma but laryngospasm – an alternative and more plausible explanation for these sudden attacks of difficulty breathing. Imagine that some irritant triggers the vocal cords to suddenly adduct and tightly obstruct the upper airway. One cannot breathe (or talk), and it feels as though one...
were about to suffocate to death. One tries to inhale or exhale, but no air can pass the closed glottis. After what seems like an eternity but is probably well less than one minute, the laryngeal spasm begins to abate. As the vocal cords begin slowly to move apart, one can start to get air passed, with an inspiratory sound that we recognize as stridor. At first, air enters the lungs with increased resistance through the narrowed upper airway, but over several seconds, as the laryngeal muscles further relax and the vocal cords abduct fully, normal breathing is restored. The entire event is over in a minute or two, and no medication is needed (or likely to help). An inhaled bronchodilator might be more irritating to the larynx and should probably be avoided.

What causes some people to develop laryngospasm is not known. Our young man had a normal ENT examination with direct laryngoscopy to exclude a structural abnormality of the glottis. His laryngeal sensitivity developed without prior trauma (such as a recent intubation) or other explanation. Potential triggers that may set off spasm of the sensitized larynx include mucus draining from the posterior pharynx, acid refluxed from below, cough with secretions expectorated at high velocity, or oro-pharyngeal aspiration.

Laryngospasm is to be distinguished from vocal cord dysfunction (VCD) syndrome, in which signals from the cerebral cortex cause inappropriate narrowing (not closure) of the vocal cords, often on exhalation. The resulting expiratory wheezing through the narrowed glottis can mimic the wheezing of asthma. VCD is typically the result of major psychologic/psychiatric stresses, a variant of Munchausen’s syndrome. Laryngospasm is a localized reflex action of the laryngeal muscles.

Preventing provokers of laryngospasm, such as laryngopharyngeal reflux, is an important treatment, especially in persons with frequent night-time episodes. Other management strategies that have been described include “rescue breathing” techniques taught by speech-language therapists; application of forward and upward pressure behind the earlobes and in front of the mastoid processes in what has been described as the “laryngospasm notch” (Shinjo T, et al., *J Anesth* 2013; 27:761-3); use of neuromodulating drugs such as amitriptyline or gabapentin; and, in desperate circumstances, botox injections into the larynx.

In most instances, however, simply coming to understand the mechanism of the event is key to dealing with it: one needs to try to stay calm, attempt small breaths in through the nose, and perhaps visualize relaxation and separation of the vocal cords. Knowing that the spasm of the larynx will pass in a matter of seconds and that there will be no long-term harmful effect are the reassurances that we have to offer. Distinguishing these episodes from asthma attacks is also crucially important. Treatment with bronchodilators and corticosteroids will not bring relief or prevent episodes of laryngospasm. It only confuses the issue, obscures the diagnosis, and likely frustrates the sufferer.