**STATUS ASTMATICUS**

**Definition**

Status asthmaticus is a prolonged severe attack of asthma that is unresponsive to initial standard therapy, is characterized especially by dyspnea, dry cough, wheezing, and hypoxemia, and that may lead to respiratory failure. The attack can last up to 24 hours. It is the extreme form of an asthma exacerbation that can result in hypoxemia, hypercarbia, (abnormally elevated carbon dioxide saturation in the blood) and secondary respiratory failure.Status asthmaticus is considered a medical emergency. **It** does not respond to standard treatments of [bronchodilators](https://en.wikipedia.org/wiki/Bronchodilators) (inhalers) and this should be prevented from happening through patient compliance with controller medications (e.g. steroid inhalers) in an outpatient setting.

Status asthmaticus can vary from a mild form to a severe form with bronchospasm, airway inflammation, and mucus plugging that can cause difficulty breathing, carbon dioxide retention, hypoxemia, and respiratory failure.

## Signs and symptoms

An exacerbation (attack) of asthma is experienced as a worsening of asthma symptoms with breathlessness and cough (often worse at night).

In acute severe asthma, breathlessness may be so severe that it is impossible to speak more than a few words.

* On examination, the respiratory rate may be elevated (more than 25 breaths per minute)
* Heart rate may be rapid (110 beats per minute or faster).
* Reduced oxygen saturation levels
* Auscultation of the lungs with a stethoscope may reveal reduced air entry and/or widespread wheeze.[[2]](https://en.wikipedia.org/wiki/Acute_severe_asthma#cite_note-BTS-2)
* The peak expiratory flow can be measured at the bedside; in acute severe asthma the flow is less than 50% a person's normal or predicted flow.
* Very severe acute asthma (termed "near-fatal" as there is an immediate risk to life) is characterized by a peak flow of less than 33% predicted, oxygen saturations below 92% or cyanosis (blue discoloration, usually of the lips), absence of audible breath sounds over the chest ("silent chest"), reduced respiratory effort and visible exhaustion or drowsiness. Irregularities in the heart beat and abnormal lowering of the blood pressure may be observed.[[2]](https://en.wikipedia.org/wiki/Acute_severe_asthma#cite_note-BTS-2)

## Pathophysiology

Inflammation in asthma is characterized by an influx of eosinophil’s during the early-phase reaction and a mixed cellular infiltrate composed of eosinophils, mast cells, lymphocytes, and neutrophils during the late-phase reaction. The simple explanation for allergic inflammation in asthma begins with the development of a predominantly helper T2 lymphocyte–driven, as opposed to helper T1 lymphocyte–driven, immune milieu, perhaps caused by certain types of immune stimulation early in life. This is followed by allergen exposure in a genetically susceptible individual.

Specific allergen exposure (e.g., [dust mites](https://en.wikipedia.org/wiki/Dust_mite)) under the influence of helper [Th2 helper T cells](https://en.wikipedia.org/wiki/T_helper_cell#Th1.2FTh2_model) leads to [B-lymphocyte](https://en.wikipedia.org/wiki/B-lymphocyte) elaboration of [immunoglobulin E](https://en.wikipedia.org/wiki/Immunoglobulin_E) (IgE) antibodies specific to that allergen. The IgE antibody attaches to surface receptors on airway mucosal mast cells. One important question is whether atopic individuals with asthma, in contrast to atopic persons without asthma, have a defect in mucosal integrity that makes them susceptible to penetration of allergens into the mucosa.

Subsequent specific allergen exposure leads to cross-bridging of IgE molecules and activation of mast cells, with elaboration and release of a vast array of mediators. These mediators include [histamine](https://en.wikipedia.org/wiki/Histamine); [leukotriene’s](https://en.wikipedia.org/wiki/Leukotriene) C4, D4, and E4; and a host of [cytokines](https://en.wikipedia.org/wiki/Cytokine). Together, these mediators cause bronchial smooth muscle constriction, vascular leakage, inflammatory cell recruitment (with further mediator release), and mucous gland secretion. These processes lead to airway obstruction by constriction of the [smooth muscles](https://en.wikipedia.org/wiki/Smooth_muscle), edema of the airways, influx of inflammatory cells, and formation of intraluminal mucus. In addition, ongoing airway inflammation is thought to cause the airway hyperactivity characteristic of asthma. The more severe the airway obstruction, the more likely ventilation-perfusion mismatching will result in impaired gas exchange and [low levels of oxygen in the blood](https://en.wikipedia.org/wiki/Hypoxemia).

## Diagnosis

**DIAGNOSIS**

* History and physical examination.
* **Pulmonary function studies** Pulmonary function tests are a broad range of tests that measure how well the lungs take in and exhale air and how efficiently they transfer oxygen into the blood.
* Spirometry measures how well the lungs exhale.
* Lung volume measurement detects restrictive lung diseases. In this set of diseases, a person cannot inhale a normal volume of air. Testing the diffusion capacity (also called the DLCO) permits an estimate of how efficiently the lungs transfer oxygen from the air into the bloodstream.
* Peak expiratory flow rate. Decreased.
* Functional Residual Capacity (FRC), Total [Lung](javascript:void(0);) Capacity (TLC), and Residual Volume (RV) are increased because air is trapped within the lungs.
* Chest X-ray.
* Pulse oximetry and ABG. Pulse oximetry usually reveals low oxygen saturation. ABG results often show some degree of hypoxemia, with elevated partial pressure of arterial carbon-dioxide in severe cases.
* Allergy skin testing if indicated.
* Blood level of eosinophils amd IgE.

## Treatment

After confirming the diagnosis and assessing the severity of an [asthma](http://emedicine.medscape.com/article/296301-overview) attack, direct treatment toward controlling bronchoconstriction and inflammation.

Interventions include [intravenous](https://en.wikipedia.org/wiki/Intravenous) (IV) medications (e.g. [magnesium sulfate](https://en.wikipedia.org/wiki/Magnesium_sulfate)), aerosolized medications to dilate the airways (Broncho dilation) (e.g., [albuterol](https://en.wikipedia.org/wiki/Albuterol) or [ipratropium bromide/salbutamol](https://en.wikipedia.org/wiki/Ipratropium_bromide/salbutamol)), and [positive-pressure](https://en.wikipedia.org/wiki/Positive-pressure) therapy, including [mechanical ventilation](https://en.wikipedia.org/wiki/Mechanical_ventilation). Multiple therapies may be used simultaneously to rapidly reverse the effects of status asthmaticus and reduce permanent damage of the airways. Intravenous [corticosteroids](https://en.wikipedia.org/wiki/Corticosteroid)[[3]](https://en.wikipedia.org/wiki/Acute_severe_asthma#cite_note-3) and methylxanthines are often given. If the person with a severe asthma exacerbation is on a mechanical ventilator, certain sedating medications such as [ketamine](https://en.wikipedia.org/wiki/Ketamine) or [propofol](https://en.wikipedia.org/wiki/Propofol), have Broncho dilating properties.

Fluid replacement

Hydration, with intravenous normal saline at a reasonable rate, is essential. Special attention to the patient's electrolyte status is important.

Hypokalemia may result from either corticosteroid use or beta-agonist use. Correcting hypokalemia may help to wean an intubated patient with asthma from mechanical ventilation. Hypophosphatemia may result from poor oral intake and is also an important consideration when weaning such patients.

**Antibiotics**

Patients are administered antibiotics only when they show evidence of infection (eg, pneumonia, sinusitis). In some situations, sinus imaging using computed tomography (CT) scanning or plain radiographymay be essential to rule out chronic sinusitis.

**Oxygen monitoring and therapy**

Monitoring the patient's oxygen saturation is essential during the initial treatment of status asthmaticus. Arterial blood gas (ABG) values are usually used to assess hypercapnia (carbon dioxide toxicity) during the patient's initial assessment. Oxygen saturation is then monitored via pulse oximetry throughout the treatment protocol.

Oxygen therapy is essential, with hypoxia being the leading cause of death in children with asthma. Oxygen therapy can be administered via a nasal canula or mask, although patients with dyspnea often do not like masks. With the advent of pulse oximetry, oxygen therapy can be easily titrated to maintain the patient's oxygen saturation above 92% (>95% in pregnant patients or those with cardiac disease).

In the event of significant hypoxemia, non-rebreathing masks may be used to deliver as much as 98% oxygen. Tracheal intubation and mechanical ventilation are indicated for respiratory failure.

**Leukotriene modifiers**

Leukotriene modifiers are useful for treating chronic asthma but not acute asthma. This treatment may be beneficial if used via a nebulizer, but it remains experimental. Most studies have examined intravenous use, such as that by Dockhorn et al.[[16](javascript:void(0);)]

**ICU admission criteria**

Indications for ICU admission include the following:

* Altered sensorium
* Use of continuous inhaled beta-agonist therapy
* Exhaustion
* Markedly decreased air entry
* Rising PCO 2 despite treatment
* Presence of high-risk factors for a severe attack
* Failure to improve despite adequate therapy

**Surgery**

Status asthmaticus is generally managed by means of medical therapy, with some exceptions. For example, thoracostomy is indicated in pneumothoraces.

Some children may have asthma that is primarily exacerbated by gastroesophageal reflux disease. Some patients can be treated with a combination of antireflux (eg, proton pump inhibitors) and histamine 2 (H2)–receptor antagonist agents. However, surgery, such as Nissen fundoplication (surgical procedure to treat GERG) is occasionally required.

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**Diet**

Some children with asthma may have episodes triggered by food allergies. Consultation with a nutritionist may be necessary to provide appropriate dietary management.

### Nursing Management

The main focus of nursing management is to actively assess the airway and the patient’s response to treatment. The nurse should be prepared for the next intervention if the patient does not respond to treatment.

* Constantly monitor the patient for the first 12 to 24 hours, or until status asthmaticus is under control. [Blood](https://nurseslabs.com/blood-anatomy-physiology/) pressure and cardiac rhythm should be monitored continuously during the acute phase and until the patient stabilizes and responds to therapy.
* Assess the patient’s skin turgor for signs of [dehydration](https://nurseslabs.com/diarrhea/); fluid intake is essential to combat [dehydration](https://nurseslabs.com/diarrhea/), to loosen secretions, and to facilitate expectoration.
* Administer IV fluids as prescribed, up to 3 to 4 L/day, unless contraindicated.
* Encourage the patient to conserve energy.
* Ensure patient’s room is quiet and free of respiratory irritants (eg, flowers, tobacco smoke, perfumes, or odors of cleaning agents); no allergenic pillows should be used.