

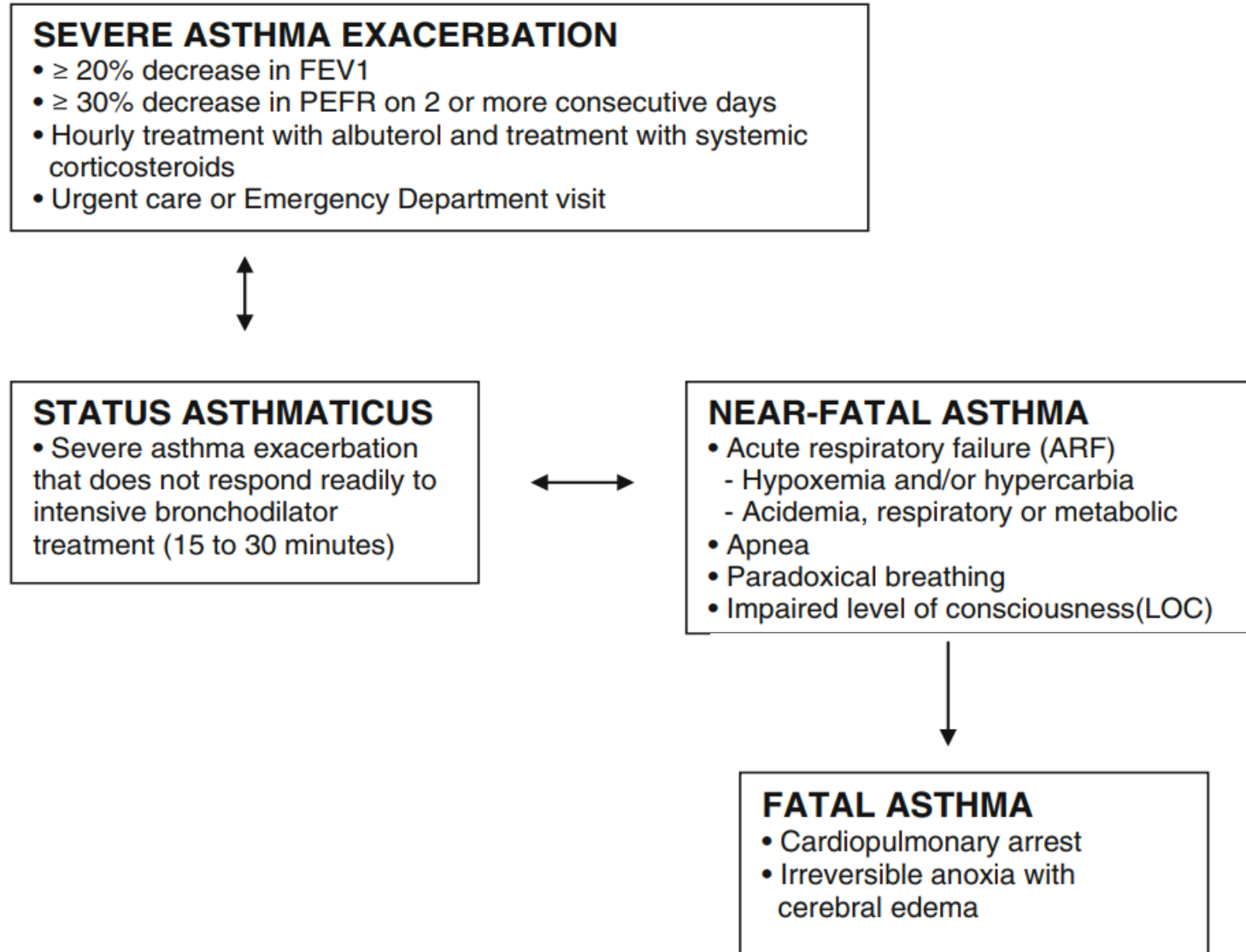
# Update in Asthma management in ICU

1-Triage of Patients to the ICU

2-Assessing severity in acute asthma exacerbations

3-TREATMENT GOALS

**Fig. 1** Deterioration of severe asthma exacerbation: status asthmaticus and near-fatal asthma with suggested ICU treatment and management



# Triage of Patients to the ICU

**Absolute** criteria for triaging are lacking

**Guidelines** recommend that patients must be monitored in an ICU if

PEFR <200 l/min,

pulsus paradoxus >15 mmHg,

use of accessory muscles of respiration

<10% improvement in PEFR

- Approximately 25% of patients are hospitalized and of these 5% to 10% require the ICU.  
Mortality rates for NFA patients requiring mechanical ventilation vary from 0% to 22%.
- The in-hospital mortality rate for all asthmatics is between 1% to 5%, but for critically ill asthmatics that require intubation, the mortality rate is between 10% to 25% primarily from anoxia and cardiopulmonary arrest.

## Assessing severity in acute asthma exacerbations

- **Mild** – normal alertness, slight tachypnea, expiratory wheezing, a mildly prolonged expiratory phase (inspiratory-to-expiratory ratio of 1:1 rather than the normal 2:1), minimal accessory muscle use, oxygen saturation of **>95** percent.
- **Moderate** – normal alertness, tachypnea, wheezing throughout expiration with or without inspiratory wheezing, an inspiratory-to-expiratory ratio of approximately 1:2, significant use of accessory muscles, oxygen saturation that is typically 92 to 95 percent.
- **Severe** – inability to repeat a short phrase, extreme tachypnea, inspiratory and expiratory wheezing, an inspiratory-to-expiratory ratio exceeding 1:2, very poor aeration, significant use of accessory muscles, and an oxygen saturation that is typically **<92** percent.
- **Signs of impending respiratory failure-** cyanosis, inability to maintain respiratory effort, depressed mental status (lethargy or agitation), pulse oxygen saturation **<90** percent, respiratory acidosis

## Assessing severity in acute asthma exacerbations

**PIS** 1 to 6 = mild, PIS 7 to 11 = moderate, or PIS  $\geq 12$  = severe  
 underestimate the degree of illness in an older child

Score	Respiratory rate		Wheezing	Inspirator y/ expiratory ratio	Accessory muscle use	Oxygen saturation
	<6 years old	$\geq 6$ years old				
0	$\leq 30$	$\leq 20$	None*	2:1	None	99 to 100
1	31 to 45	21 to 35	End expiration	1:1	+	96 to 98
2	46 to 60	36 to 50	Entire expiration	1:2	++	93 to 95
3*	>60	>50	Inspiration and expiration	1:3	+++	<93

**TABLE 2.3** Classification of Asthma Severity in Youths Older Than 12 Years and in Adults

Components of Severity		Classification of Asthma Severity (Youths ≥ 12 years of age and adults)			
		Intermittent	Persistent		
			Mild	Moderate	Severe
Impairment Normal FEV <sub>1</sub> :FVC: 8–19 yr 85% 20–39 yr 80% 40–59 yr 75% 60–80 yr 70%	Symptoms	≤ 2 days/week	> 2 days/week but not daily	Daily	Throughout the day
	Nighttime awakenings	≤ 2x/month	3–4x/month	> 1x/week but not nightly	Often 7x/week
	Short-acting β <sub>2</sub> -agonist use for symptom control (not prevention of EIB)	≤ 2 days/week	>2 days/week but not daily	Daily	Several times per day
	Interference with normal activity	None	Minor limitation	Some limitation	Extremely limited
	Lung function	<ul style="list-style-type: none"> <li>• Normal FEV<sub>1</sub> between exacerbations</li> <li>• FEV<sub>1</sub> &gt; 80% predicted</li> <li>• FEV<sub>1</sub>:FVC normal</li> </ul>	<ul style="list-style-type: none"> <li>• FEV<sub>1</sub> &lt; 80% predicted</li> <li>• FEV<sub>1</sub>:FVC normal</li> </ul>	<ul style="list-style-type: none"> <li>• FEV<sub>1</sub> &gt; 60% but &lt; 80% predicted</li> <li>• FEV<sub>1</sub>:FVC reduced 5%</li> </ul>	<ul style="list-style-type: none"> <li>• FEV<sub>1</sub> &lt; 60% predicted</li> <li>• FEV<sub>1</sub>:FVC reduced &gt; 5%</li> </ul>
Risk	Exacerbations (consider frequency and severity)	0–2/year      >2/year      ←————→ ←————→ Frequency and severity may fluctuate over time for patients in any severity category Relative annual risk of exacerbations may be related to FEV <sub>1</sub> .			

From National Asthma Education and Prevention Program. *Expert Panel Report 3: Guidelines for the Diagnosis and Management of Asthma (EPR3)*. Bethesda, MD: National Heart, Lung, and Blood Institute; 2007.

**PEF** measurement as part of a combined assessment of severity and response to treatment.

Normal values for PEF depend on sex, age, and height- day-to-day variability

Reductions in **PEFR to <33%** of normal is considered as a life-threatening situation.

Respiratory alkalosis is the most common abnormality found during asthma exacerbations but as **PEFR and FEV1 drop to <30%** of predicted, **hypercarbia** and respiratory acidosis develop.

A peak flow **>80** percent of predicted or the FEV<sub>1</sub> is **>80** percent predicted suggests that asthma is well controlled. However, there is wide variability in PEFR; a one-time measurement may not be useful for evaluating airway obstruction. In general, a PEFR  $\leq$ 80 percent of predicted should be evaluated further with spirometry

## Clinical Evaluation upon Admission to the ICU

**Status A.** is a medical emergency-requires **urgent** assessment

The most **worrisome patients** will often be sitting upright, tachypneic, wheezing, and have sternocleidomastoid contraction with respiration, hypotensive because of dehydration and marked lung hyperinflation with impaired cardiac filling

**Paradoxical breathing** signifies impending respiratory **arrest** from total exhaustion and should prompt immediate endotracheal intubation and mechanical ventilation, **not** non-invasive mask ventilation (NIV) or continuous positive airway pressure ventilation (CPAP).

Patients with signs of respiratory failure—a decreased level of consciousness, shallow respirations, central cyanosis, or other signs of profound fatigue—should be **endotracheally** intubated **urgently**.

Safe endotracheal intubation in these patients is often a **challenge**.



## TREATMENT GOALS

- Rapid reversal of **airflow obstruction**
- Correction of **hypoxemia and/or severe hypercapnia**- hypoxemia as a result of ventilation-perfusion (V/Q) mismatch-
- worsen this mismatch by causing **pulmonary vasodilation** in areas of the lung that are poorly ventilated
- **Adjuncts to mechanical ventilation**, including heliox, general anesthesia, and extracorporeal carbon dioxide removal, can be used as life-saving measures in extreme cases.

## Initial ICU Management

**Supplemental oxygen** should be administered to improve the hypoxia caused by (V/Q) mismatch- FiO<sub>2</sub> levels of **30–50%** will correct the hypoxemia- **Failure** to do so should prompt an investigation for pulmonary parenchymal or vascular disease.

**Bronchodilators** are the cornerstone of acute asthma management. Delivery of the short-acting beta-agonist albuterol is the most common therapy for acute asthma.

**Anticholinergics** or antagonists of relevant muscarinic receptor subtypes in the tracheobronchial tree are emerging as an important bronchodilator therapy for persistent asthma.

**Systemic Corticosteroids:** There is typically a 6- to 24-h **delay** in clinical response to corticosteroids in SA and NFA, but they have been shown to reduce fatal asthma.

- **Methylxanthines**

The methylxanthines, theophylline and aminophylline, are **less specific** bronchodilators and some evidence showing that they improved diaphragmatic contractility may be of utility in a failing patient.

Nonetheless, given the higher risk of adverse cardiac events, narrow therapeutic dosing windows, and lack of proven additional efficacy, these are **not** commonly used.

- **Magnesium**: inhibiting calcium channels in bronchial smooth muscle and blocking parasympathetic tone in the tracheobronchial tree.

## Adjunctive-Nonstandard therapies

Inhaled **heparin**: immune-modulating adjunctive therapies

Inhaled **furosemide**: immune-modulating adjunctive therapies

They show a decrease in hyper-responsiveness.

**Helium** admixtures: 20% O<sub>2</sub>, 80% He or 30% O<sub>2</sub>, 70% He mixture, increased laminar flow and a reduction in airway resistance in areas of greatest turbulent flow. This can result in a reduction in the work of breathing.

No evidence yet exists that helium-oxygen (heliox) admixtures can prevent the need for endotracheal **intubation**. However, heliox has been shown to improve PEF and reduce the degree of pulsus paradoxus in acute asthma attacks.

## Adjunctive-Nonstandard therapies

- **Nitroglycerin** – While rarely used for the treatment of asthma, IV nitroglycerin has been shown to be effective in the treatment of acute bronchospasm by direct smooth-muscle relaxation. Nitroglycerin causes hypotension.
- **Extracorporeal life support** — Oxygenation and carbon dioxide removal through an artificial membrane may be beneficial as a temporizing measure in patients with severe asthma **refractory** to usual care and mechanical ventilation.

## Initial ICU Management cont.

### Non-invasive Ventilation

NIV decreases morbidity and mortality in COPD, but data are **limited** to asthma, but has been shown to decrease the need for mechanical ventilation in some asthmatics with acute respiratory acidosis.

NIV should **not** be used in patients in respiratory distress, patients with altered levels of consciousness, or patients with hemodynamic instability and impending cardiorespiratory arrest.

# The decision to endotracheally intubate and mechanically ventilate

1-who are failing to respond to treatment and are fatiguing, 2-to provide adequate supplemental oxygen to prevent anoxia and organ ischemia, 3-reduce the work of breathing, 4-and prevent severe acidemia, respiratory, or metabolic, 5-or allow permissive hypercapnia to prevent ventilator-induced lung injury, including barotraumas, 6-and any patient with progressive lethargy, somnolence, near exhaustion, 7-unresponsiveness, apnea, near apnea or 8- cardiopulmonary arrest should be intubated.

**An elevated PaCO<sub>2</sub> level on admission**, although shown to be associated with increased mortality, may not necessarily warrant immediate endotracheal intubation. Any patient with a progressive rise in PaCO<sub>2</sub> despite therapy and increasing fatigue most likely will require intubation

NFA patients may worsen in the hours immediately after intubation-because manipulation of the airway can cause increased airflow obstruction.

## **The decision to endotracheally intubate and mechanically ventilate** **complications**

Hypotension (20–40% of cases), arrhythmias, barotrauma, laryngospasm, worsening bronchospasm, aspiration, and seizures will be encountered in the peri-intubation period in near-fatal asthma patients.

**Hypotension** often results from a combination of sedative and neuromuscular blocking agents, in the setting of high intrathoracic pressures. Intravascular volume supplementation prior to and immediately after intubation can prevent this expected complication.

**Massive auto-PEEP** while on mechanical ventilation can go unrecognized immediately after intubation during the early minutes of mechanical ventilation when the patient is either still paralyzed or heavily sedated.



## What is the best approach when a patient requires intubation?

It should be performed by the **most** experienced person available

rapid-sequence technique(**RSI**) using neuromuscular blockade with succinylcholine or rocuronium

Because of its intrinsic sympathomimetic and bronchodilating properties, **ketamine** is an agent of choice

Barbiturates such as **thiopental** should not be used because of their association with histamine release

**Propofol** is the intravenous (IV) induction agent of choice for the hemodynamically stable asthmatic patient and has been shown to attenuate the bronchospastic response to intubation in both asthmatic and nonasthmatic patients. However, rare allergic reactions with significant bronchospasm have occurred with propofol administration, possibly due to the sodium metabisulfite used as a **preservative** in some preparations

**lidocaine** 1 to 1.5 mg/ Kg is typically administered during induction to suppress the cough reflex during laryngoscopy and intubation. Although lidocaine may mildly increase **airway tone**, it reduces airway responsiveness to noxious stimuli and to drugs that cause bronchospasm, thereby potentially preventing bronchospasm.

**Inhaled lidocaine** should be avoided since it is an airway irritant and may cause bronchospasm

## Setting the Ventilator

- **Errors** occur because physicians attempt to correct the hypercarbia and acidemia too quickly and fail to recognize the extent of the dynamic hyperinflation. The key goal at this time is to maximize the time for expiration and target a low-minute ventilation strategy.
- **low-minute ventilation** strategy (less than 115 mL/kg/min) aims to permit time for expiration, decrease air trapping, and reduce PEEPi. The ICU physician should accept a moderate degree of hypercapnia with this strategy and PaCO<sub>2</sub> levels <100 mmHg are usually well tolerated on the first day
- **One exception** to this is in patients who have suffered a cardiorespiratory arrest at the time of presentation. In these patients, PaCO<sub>2</sub> levels should be normalized, if possible, to prevent cerebral vasodilatation and cerebral edema
- Lung-protective ventilation refers to the use of **low tidal volumes** (6 to 8 mL/kg predicted body weight), positive end-expiratory pressure (**PEEP**) 6 to 8 cmH<sub>2</sub>O.

## Setting the Ventilator cont

- require **deep sedation** initially in order to breathe synchronously with the controlled modes
- to decrease the variability of peak pressures, set the flow in a **decelerated waveform**
- Failure of the flow (L/min) ventilator graphic to return to baseline should prompt lengthening of the expiratory time or a **decrease in the I:E ratio** and/or add extrinsic PEEP to reduce the patient's work of breathing to exhale trapped air.
- low-minute ventilation of **8 to 10 l/min**
- inspiratory flow rates of **80-90 l/min** allow for a prolonged expiratory phase
- **Plateau pressures** should remain <35 cmH<sub>2</sub>O to prevent barotrauma and the set PEEP should be 0 to 5 cmH<sub>2</sub>O initially.
- **FiO<sub>2</sub>** should be decreased from a level of 1.0 over the first several hours.
- If near-fatal asthmatics continue to **require FiO<sub>2</sub> > 0.55**, the intensivist should search for a concomitant process, such as disorder worsening V/Q mismatching or an intrapulmonary shunt from atelectasis, such as pneumonia, pulmonary embolism, or pulmonary edema.

## Non-bronchospastic causes

- **Endobronchial intubation** – Decreased breath sounds on one side (usually left) and a deep endotracheal tube (ETT).
- **Pneumothorax** – Decreased breath sounds on one side; asymmetric chest rise, particularly if high peak inspiratory pressures have been used or in a clinical scenario where pneumothorax is likely (eg, trauma, diaphragm injury).
- **Pulmonary edema** – Frothy secretions in the ETT; crackles on the pulmonary exam.
- **Kinked or obstructed ETT** – Difficulty passing a suction catheter and/or removal of secretions upon suctioning.

## Permissive hypercapnia

- **Permissive hypercapnia** — The strategies of decreasing the respiratory rate and tidal volume to prevent barotrauma can lead to an increase in the arterial tension of carbon dioxide ( $\text{PaCO}_2$ ). Permissive hypercapnia refers to the acceptance of the elevated  $\text{PaCO}_2$  and associated respiratory acidosis. The effects of respiratory acidosis are generally better tolerated than the consequences of barotrauma, such as pneumothorax. The indications, contraindications, and technique of permissive hypercapnia are discussed separately.
- **Rate of rise of arterial carbon dioxide tension** —  $\text{PaCO}_2$  levels should rise gradually during mechanical ventilation rather than rapidly, preferably at a rate of <10 mmHg per hour. The rise should be even slower if the  $\text{PaCO}_2$  exceeds 80 mmHg.
- **Upper limit of arterial carbon dioxide tension** — There is no absolute level above which  $\text{PaCO}_2$  should not rise, although levels are practically limited by the associated degree of acidosis, and levels higher than 100 mmHg are generally not required.
- **Lower limit of pH and correction of acidosis** — There is no consensus regarding an acceptable lower limit for pH, whether the acidosis should be corrected or at what level correction should be considered. However, most physicians agree that the pH should not need to be normalized

## Setting the Ventilator cont

- The use of PEEP for ventilation in asthmatics is **controversial**.
- Extrinsic PEEP may actually worsen air trapping and exacerbate hyperinflation. Conversely, PEEP may prevent airway collapse by stenting airways open, thereby decreasing air trapping. When PEEP is used in asthmatic patients, it should be used cautiously, monitoring for signs of hyperinflation.
- Extrinsic positive end-expiratory pressure (extrinsic PEEP, also known as applied PEEP) less than 80 percent of the intrinsic PEEP, or 5 cm H<sub>2</sub>O if intrinsic PEEP is <10 cm H<sub>2</sub>O
- Ideally, the intrinsic PEEP should be <10 cm H<sub>2</sub>O.
- **Additional methods** to decrease intrinsic PEEP include prolonging the expiratory phase, reducing minute ventilation, and administering medications to promote bronchodilation.

- **Reasonable initial ventilator settings** include a respiratory rate from 10 to 12 breaths/min, a tidal volume of 6 to 8 mL/kg, a minute ventilation less than 115 mL/kg, and an inspiratory flow up to 80-90 L/min. Positive end-expiratory pressure (PEEP) is usually initiated at 5 cm H<sub>2</sub>O.
- Increasing the amount of extrinsic PEEP (eg, up to 80 percent of the intrinsic PEEP) can offset the effects of intrinsic PEEP and reduce the effort necessary to trigger inspiration during patient-initiated breaths.

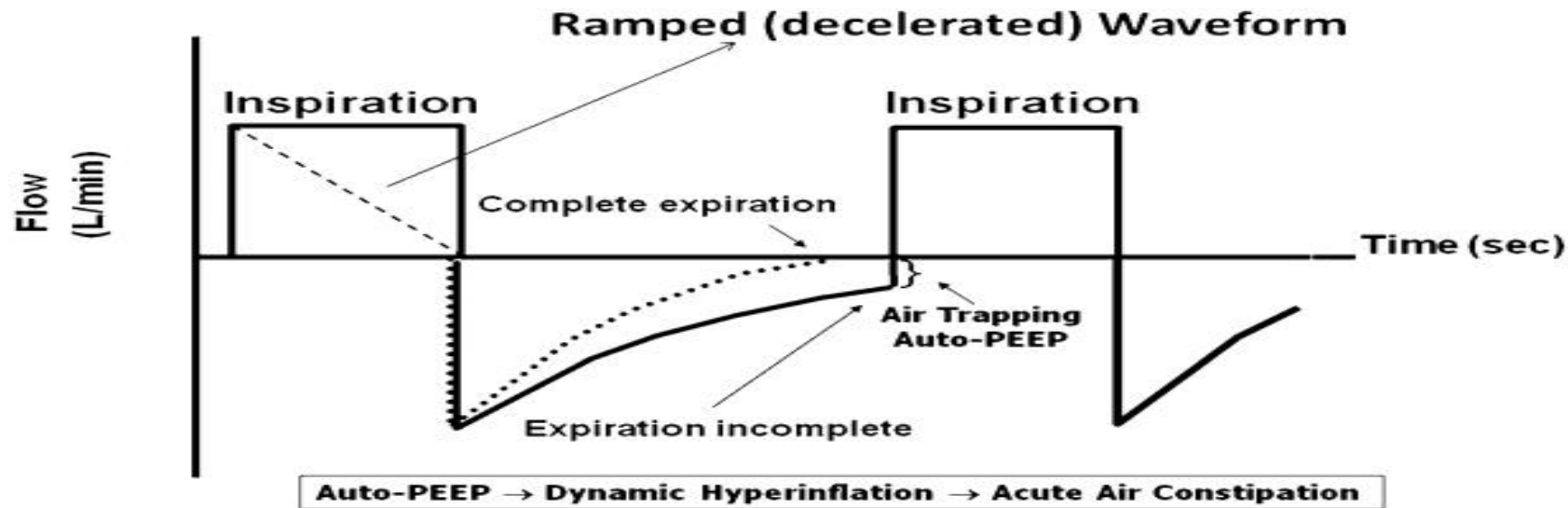
**Table 3** Suggested initial ventilator settings

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Modes	Synchronized, intermittent mandatory ventilation Volume control with decelerated waveform Pressure control ventilation
Rate	8–12 breaths/min
Tidal volume	6–8 cc/kg ideal body weight
Minute ventilation	8–10 l/min
<i>I:E</i>	1:3–1:4
Plateau pressure	<30 cmH <sub>2</sub> O
PEEP	<5 cmH <sub>2</sub> O
FiO <sub>2</sub>	100%

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**Fig. 4** Ventilator graphic illustrating air trapping and auto-PEEP during volume control mechanical ventilation. It is important to increase the expiratory time, reduce the  $I:E$  ratio to 1:3 or 1:4 and/or add external PEEP to permit the patient to completely exhale before the next breath. A ramped waveform is also diagrammed and may be used with certain volume control modes to minimize high peak pressures

- The majority of intubated near-fatal asthmatics will be liberated from mechanical ventilation with a mean time to **extubation of 3.5 days.**
- Patients require close observation immediately post-extubation for worsening bronchospasm and can usually be transferred out of the ICU **after 24 h.**

## **ICU TREATMENT & MANAGEMENT**

- Activate Critical Care ICU Team
- Oxygen to keep SpO<sub>2</sub> > 92%
- Continue albuterol and corticosteroids.
- Add ipratropium & consider other treatments
- Consider noninvasive ventilation (NIV)  
e. g. BiPAP 12/5 if patient able to protect airway and LOC not impaired
- Monitor clinical response every 15 minutes with ABGs if possible for the next 1 hour
- Rapid sequence intubation with No. 8 mm endotracheal tube if ARF ensues or if evidence of pneumonia on CXR
- Mechanical ventilation SIMV with FiO<sub>2</sub> 1.0, TV 6 to 8 ml/kg, I:E ratio 1:3 or 1:4, PEEP<sub>e</sub> 5 cm H<sub>2</sub>O. Check for auto-PEEP (PEEP<sub>i</sub>)
- Monitor PIP and Pplat. Keep Pplat < 30 cm H<sub>2</sub>O
- If pH < 7.25 and falling and/or PaCO<sub>2</sub> > 80 mm Hg and rising after 2 to 4 hours, consider permissive hypercapnia or either IV (ketamine, propofol, dexmedetomidine) or general anesthesia (isoflurane, sevoflurane)
- Check ABGs every hour. Keep pH > 7.20
- Monitor for barotraumas, e. g. pneumothorax
- If respiratory acidosis and hypoxemia persist or worsen after 4 to 6 hours, activate ECCOR and ECMO Team

**TABLE 2.2****Most Clinically Useful Spirometric Tests of Lung Function**

**Forced expiratory volume in 1 sec ( $FEV_1$ ):** The volume of air that can be forcefully exhaled in 1 sec. Values between 80% and 120% of the predicted value are considered normal.

**Forced vital capacity (FVC):** The volume of air that can be exhaled with maximum effort after a deep inhalation. Normal values are  $\approx 3.7$  L in females and  $\approx 4.8$  L in males.

**Ratio of  $FEV_1$  to FVC:** This ratio in healthy adults is 75%–80%.

**Forced expiratory flow at 25%–75% of vital capacity ( $FEF_{25\%–75\%}$ ):** A measurement of airflow through the midpoint of a forced exhalation.

**Maximum voluntary ventilation (MVV):** The maximum amount of air that can be inhaled and exhaled within 1 min. For patient comfort, the volume is measured over a 15-sec time period and results are extrapolated to obtain a value for 1 min expressed as liters per minute. Average values for males and females are 140–180 and 80–120 L/min, respectively.

**Diffusing capacity ( $D_{LCO}$ ):** The volume of a substance (carbon monoxide [CO]) transferred across the alveoli into blood per minute per unit of alveolar partial pressure. CO is rapidly taken up by hemoglobin. Its transfer is therefore limited mainly by diffusion. A single breath of 0.3% CO and 10% helium is held for 20 sec. Expired partial pressure of CO is measured. Normal value is 17–25 mL/min/mm Hg.