Received: 14 April 2020 | Revised: 27 July 2020 | Accepted: 28 July 2020

DOI: 10.1002/emp2.12224

JACEP OPEN
JOURNAL OF THE AMERICAN COLLEGE OF EMERGENCY PHYSICIANS OPEN
A GLOBAL JOURNAL OF EMERGENCY MEDICINE
WILEY

REVIEW ARTICLE

Pediatrics

Emergency department treatment of asthma in children: A review

Moon O. Lee MD, MPH | **Shyam Sivasankar MD** | **Nicholas Pokrajac MD** | **Cherrelle Smith MD** | **Angela Lumba-Brown MD**

Department of Emergency Medicine, Stanford University School of Medicine, Stanford, California, USA

Correspondence
Moon O. Lee, MD, MPH, Department of Emergency Medicine, Stanford University School of Medicine, 900 Welch Road, Suite 350, Mailcode 5119, Stanford, CA 94304, USA.
Email: moonlee@stanford.edu

Funding and support: By *JACEP Open* policy, all authors are required to disclose any and all commercial, financial, and other relationships in any way related to the subject of this article as per ICMJE conflict of interest guidelines (see www.icmje.org). The authors have stated that no such relationships exist.

Abstract

Asthma is the most common chronic illness in children, with >700,000 emergency department (ED) visits each year. Asthma is a respiratory disease characterized by a combination of airway inflammation, bronchoconstriction, bronchial hyperresponsiveness, and variable outflow obstruction, with clinical presentations ranging from mild to life-threatening. Standardized ED treatment can improve patient outcomes, including fewer hospital admissions. Informed by the most recent guidelines, this review focuses on the optimal approach to diagnosis and treatment of children with acute asthma exacerbations who present to the ED.

KEYWORDS
asthma, child, emergency service hospital, emergency treatment, respiratory disorders

1 | INTRODUCTION

Asthma is the most common chronic illness in children, affecting >6 million children in the United States.[1] In 2014 to 2015, of all emergency department (ED) visits in the United States, 9.5% had an asthma diagnosis documented in the medical record; the largest proportion of these patients were children aged 5 to 17 years (13%).[2] Each year, asthma accounts for >700,000 ED visits by children and 2% to 5% of all pediatric hospitalizations.[3,4]

In this review, we provide an up-to-date overview of the ED diagnosis and treatment of children with acute asthma exacerbations. Optimal treatment in the ED can improve patient outcomes, including both reducing ED length of stay and hospital admission rates.[5–8] The review focuses on ED treatment and reflects the literature and guideline recommendations through 2019. We begin with a brief discussion of the clinical presentation of asthma in children, the differential diagnoses, and the scoring tools available to aid in the assessment of severity. We then review the ED treatment of asthma, with a focus on the most well-supported therapies. We conclude with general guidelines to determine patient disposition.

2 | CLINICAL PRESENTATION

2.1 | Definition

Asthma is a chronic lower respiratory tract disease characterized by a combination of airway inflammation, bronchoconstriction, bronchial hyperresponsiveness, and variable outflow obstruction. In patients with asthma, inflammatory cell activation leads to airway edema and hypersecretion of mucous, and, if sufficiently chronic and severe, may progress to airway remodeling and persistent narrowing.[9] An acute, severe asthma exacerbation unresponsive to repeated administration of inhaled β-agonists may be life-threatening if untreated.

Supervising Editor: Benjamin T. Kerrey, MD, MS.

2.2 | Diagnosis

Young children can be particularly difficult to diagnose with asthma because wheezing can be caused by a variety of etiologies, most commonly respiratory viral infections. Rather than formally diagnosing children with asthma, ED clinicians are tasked with determining when to initiate treatment for a possible or likely asthma exacerbation. ED clinicians must use a combination of history and physical exam findings to determine when to start treatment while looking for alternative diagnoses if patients are not improving.

2.2.1 | History

A hallmark of asthma is recurrent exacerbations. Characteristic features of exacerbations include wheezing, cough, shortness of breath, and chest tightness. Caretakers may report a worsening of symptoms at night and fatigue or poor sleep, especially in school-age children. Exacerbations are often triggered by respiratory infections, creating considerable overlap between symptoms of an isolated viral lower respiratory tract infections and asthma. Other common triggers include inhaled irritants such as tobacco smoke, exposure to environmental allergens, or changes in weather.[9]

2.2.2 | Physical exam

Wheezing is the most common physical finding associated with asthma and is included on almost all published scoring tools for the assessment of asthma in children.[10] Wheezing should not be used in isolation. Wheezing is notably absent in many severe exacerbations as a result of a lack of airflow.[11] Wheezing may also be caused or mimicked by other disease processes. Wheezing is a high-pitched, continuous noise that is most often expiratory. Wheezing should not be confused with stridor, which is also high pitched but primarily inspiratory, or stertor, which is more variable in pitch and respiratory phase and is primarily associated with nasal congestion and discharge. Wheezing has good reliability in controlled studies of children with asthma and pneumonia.[10,12,13] In clinical practice, however, the assessment of wheezing is likely to be less valid and reliable when using loudness and focality to determine severity.

Physical examination should focus on a child's mental status and respiratory effort. Particular attention should be paid to a child's mental status—a fussy, crying child is more reassuring than a quiet, listless child, which should alert the clinician to the possibility of more severe disease. On examination, children may have increased work of breathing which manifests as nasal flaring, facial pallor, grunting, head bobbing, and retractions. The expiratory phase may be notably prolonged and is usually associated with intercostal retractions and wheezing. In contrast, prolonged inspiration and sternal retractions are physical examination findings more consistent with an upper airway obstruction and should not be confused with a severe asthma exacerbation.

2.2.3 | Differential diagnosis

The differential diagnosis of acute asthma exacerbation is detailed in Table 1. Asthma may be challenging to diagnose in children and assessing the severity of an acute exacerbation may be particularly difficult. Any child with a reported asthma history unresponsive to asthma therapy requires reconsideration of the diagnosis.[14]

3 | SCORING TOOLS AND DISEASE SEVERITY

Despite being the gold standard to diagnose asthma, pulmonary function tests and incentive spirometry lack reproducibility in young children and are unreliable during acute illness. Pulmonary function tests are also impractical in the ED and should not be used during an acute exacerbation.[11,15,16] Clinical assessments of children with an asthma exacerbation may also be variable.[17]

To standardize clinical diagnosis and deliver timely ED care, several pediatric asthma scoring tools have been developed (Table 2). These scoring tools are intended to guide treatment decisions during acute asthma exacerbations by determining the level of ED treatment, repeating scores to determine changes in treatment, and patient disposition. Each scoring tool has several examination findings scored on a 3-point, 4-point, or 7-point scale; higher numbers on each scale represent more severe disease. Examples of common examination findings are the presence of wheezing, respiratory rate, work of breathing, oxygen saturation, expiratory phase, and dyspnea.

Most scoring tools have demonstrated good interrater reliability, but studies comparing tools are limited.[12,16,18–25] Asthma severity scoring tools have been associated with more timely administration of medications, reductions in ED length of stay, and reductions in admissions without a corresponding increase in return ED visits.[8,26,27] Some tools can also predict the need for admission and level of inpatient care.[25,28–31] Several institutions have developed asthma treatment pathways based on scoring tools to improve care through standardization.[32,33]

4 | TREATMENT

For patients with mild asthma, first-line treatment is albuterol hydrofluroalkane-pressurized (HFA) and an oral steroid, for example, dexamethasone, and may also include inhaled ipratropium. Patients with moderate to severe asthma exacerbations will typically receive inhaled albuterol and ipratropium, an oral or systemic steroid, and if symptoms are not improving intravenous magnesium. Patients with severe or life-threatening exacerbations will receive all of these treatments and one or more adjunctive treatments, which may include systemic epinephrine. Patients with severe asthma exacerbation that does not respond to treatment may also require non-invasive positive pressure ventilation (NIPPV) and monitoring for deteriorating mental status and ventilatory failure. Medication dosages and administration routes are detailed in Table 3.

TABLE 1 Differential diagnoses for acute asthma exacerbation in a child

Diagnosis	Chronicity	Clinical features
Anaphylaxis	Acute	Urticaria, facial/oral edema, emesis, abdominal pain
Bronchiolitis	Acute	Fever, nasal congestion, rhinorrhea, coarse rales on auscultation
Foreign body (lung or esophagus)	Acute	History of choking, unilateral wheezing
Pneumonia	Acute	Fever, focal wheezing, cough, fatigue
Aspiration syndromes	Chronic	Coughing or choking with feeding, recurrent pneumonia, cough
Anatomic abnormalities (eg, malacias, external compression)	Chronic	Recurrent pneumonia, fixed wheezing
Bronchopulmonary dysplasia	Chronic	History of prematurity, history of oxygen requirement
Cystic fibrosis	Chronic	Frequent pneumonia, failure to thrive, persistent cough
Primary ciliary dyskinesia	Chronic	Frequent pneumonia, recurrent sinusitis and otitis, cough
Asthma	Acute and/or chronic	Diminished air flow, prolonged expiratory phase, cough
Heart disease	Acute and/or chronic	Poor feeding or sweating with feeds, failure to thrive, cyanosis, tachycardia, hepatomegaly

TABLE 2 Severity scoring tools for pediatric asthma

Asthma score (study)	Ages	Interrater reliability	Components (point range)	Score interpretation and when to use
PASS: Pediatric Asthma Severity Score[a] (Gorelick et al[21])	1–18 years	Good to excellent	Degree of wheezing (0–2) Work of breathing (0–2) Prolonged expiration (0–2)	Score >2 predicts length of stay >6 hours or hospital admission Use during initial assessment
PRAM: Pediatric Respiratory Assessment Measure[a] (Chalut et al[22])	Original 3–6 years; subsequent validation from 18 months to 18 years	Good	Pulse oximetry value (0–2) Intensity of air entry (0–3) Degree of wheezing (0–3) Suprasternal retractions (0–2) Scalene retractions (0–2)	Mild: 0–1 Moderate: 4–7 Severe: 8–12 Use during initial assessment and to assess response to treatment
AAIRS: Acute Asthma Intensity Research Score (Arnold et al[23])	5–18 years	Good	Retractions (0–6) Characteristics of air entry (0–3) Degree of wheezing (0–3) Pulse oximetry value (0–3) Prolonged expiration (0–3)	Mild: 1–6 Moderate: 7–11 Severe: 12–16 Use during initial assessment to determine pediatric ICU admission and to assess response to treatment
PAS: Pediatric Asthma Score (Kelly et al[24])	2–18 years	Not reported	Respiratory rate (1–3) Oxygen requirements (1–3) Auscultation (1–3) Retractions (1–3) Dyspnea (1–3)	Mild: 5–7 Moderate: 8–11 Severe: 12–15 Use during initial assessment and to guide management

[a]Score developed for the emergency department.

4.1 | Albuterol

Albuterol is one of the two mainstays of acute asthma treatment. Albuterol is a long-acting β2 receptor agonist, producing bronchodilation through smooth muscle relaxation. Albuterol's onset of action is <5 minutes, and duration of action is 3 to 6 hours. The most common side effects with albuterol are vomiting, tremor, and tachycardia attributed primarily to peripheral vasodilatation.[34] Albuterol is most commonly administered via HFA metered dose devices. Most young children cannot create a seal around the mouthpiece of the HFA and will need a mask and spacer to improve medication delivery. Albuterol can also be administered via nebulization, but albuterol HFA with a spacer delivers an equivocal dose compared with nebulizers.[35] Albuterol HFA also results in shorter ED length of stay, fewer hospital admissions, and no differences in oxygen saturations.[36,37] Albuterol HFA is also the preferred mode of administration for patients under airborne precautions, such as for severe acute respiratory syndrome coronavirus 2.

In severe asthma exacerbations, the use of continuous, nebulized albuterol is recommended.[35] A recent study of continuous albuterol in pediatric patients found no optimum weight-based dose for decreasing the length of hospital stay. This study suggests that lower doses of

TABLE 3 Medications for the emergency department management of asthma exacerbations

Medication name	Route	Typical dose	Typical maximum dose
Primary medications			
Albuterol sulfate	HFA	4–8 puffs	
	Nebulized	2.5–5 mg	
	Continuous	5–20 mg/hour	
Ipratropium bromide	HFA	4–8 puffs	
	Nebulized	0.25–0.5 mg	1.5 mg/hour
Dexamethasone	PO, IV, IM	0.6 mg/kg	16 mg
Prednisone	PO	2 mg/kg	60 mg
Prednisolone	PO	2 mg/kg	60 mg
Secondary medications			
Magnesium sulfate	IV	25–75 mg/kg	2 g
Epinephrine	IV, IM	0.01 mg/kg	1 mg
Terbutaline	SC	<12 years 10 mcg/kg/dose every 15 minutes for 2 doses >12 years 0.25 mg/dose every 15 minutes for 2 doses	250 mcg /dose
	IV	2–10 mcg/kg loading dose followed by infusion 0.1–0.4 mcg/kg/min	3 mcg/kg/min
Ketamine	IV	2 mg/kg loading dose followed by 20–60 mcg/kg/min	

HFA, albuterol hydrofluroalkane-pressurized; IM, intramuscular; IV, intravenous; PO, orally; SC, subcutaneous.

continuous albuterol may be just as effective and potentially decrease cost and unwanted side effects.[38]

4.2 | Ipratropium

Ipratropium, a muscarinic receptor antagonist, also produces bronchodilation as well as decreasing mucous production. Ipratropium's onset of action is 15 to 30 minutes and the duration of action is 3 to 5 hours.[39] Common side effects of ipratropium are dry mouth, headache, and nasal congestion.

Ipratropium is typically added to albuterol in moderate or severe exacerbations. The addition of ipratropium to albuterol has been associated with reduced hospital admission in pediatric patients with moderate and severe asthma exacerbations.[40] Early initiation of ipratropium in the ED has been shown to prevent hospital admission, but little benefit has been reported in continued use in the critical care or floor setting after admission.[41]

4.3 | Systemic steroids

Systemic steroids are the other mainstay of acute asthma therapy. Multiple studies have shown early steroid administration in the ED reduces hospitalization.[6,42] Recent literature supports dexamethasone as the preferred systemic steroid. Dexamethasone is a long-acting steroid that can be given orally or intramuscularly. Dexamethasone's onset of action when given orally is 1 to 2 hours and 30 to 120 minutes after intramuscularly administration. The half-life is 2 to 9 hours, and commonly reported side effects include nausea, vomiting, restlessness, and fatigue.[43] A 2016 study reported that 1 dose of dexamethasone was not inferior compared with 3 days of prednisone in terms of hospital admission rate or unscheduled return visits to healthcare providers.[44] In a study comparing a 2-dose regimen of oral dexamethasone to a traditional 5-day course of prednisone, similar efficacy with increased compliance was reported.[40,45] In a separate study, children with acute asthma treated with 1 dose of dexamethasone had a 36% relative risk reduction in 72-hour ED return rate compared with children treated with a 3-5-day course of oral prednisone.[46] Dexamethasone is also more cost-effective and tastes better, and patients are more likely to complete treatment when compared with prednisolone.[47,48]

The literature comparing single-dose dexamethasone to multiple doses of dexamethasone is limited. Most studies suggest single-dose versus a daily dose for 2 days.[49] More research is needed to determine if providing a second oral dose will aid in pediatric exacerbation relapses as well as more investigation of the effect of this regimen in the hospitalized pediatric patient.[47]

Prospective randomized trials show no clinical difference between oral and intramuscular steroid administration.[50–52] Intramuscular administration may be used in patients who are unable to tolerate oral steroids. Parents reported a preference for a single dose of intramuscular dexamethasone compared with 5 days of oral prednisone.[51]

4.4 | Magnesium

Magnesium is a second-line therapy in children presenting with moderate to severe asthma exacerbation. Magnesium is administered intravenously, competing for calcium channels with resultant smooth muscle relaxation and bronchodilation. Magnesium also decreases histamine release from mast cells and produces an anti-inflammatory effect by decreasing super-oxide production by neutrophils.[53] Magnesium's onset of action is immediate and given over 15 to 60 minutes.[54] Magnesium is well tolerated and has few side effects. Hypotension, dry mouth, nausea, flushing, hyporeflexia, and respiratory depression are reported but rare.

Magnesium should be considered in patients insufficiently responsive to first-line treatment or for those with more severe presentations on arrival. In a 2016 Cochrane review of 5 randomized, placebo-controlled studies, children treated with intravenous magnesium were 68% less likely to be admitted to the hospital. There were insufficient data to evaluate the effects of magnesium administration on secondary outcomes, including ED treatment durations, ICU admission rates, hospital lengths of stay, and adverse events.[55] There is no conclusive evidence on the impact of inhaled versus intravenous magnesium.[56,57]

4.5 | Epinephrine and terbutaline

Epinephrine and terbutaline are adjunctive therapies for patients with severe exacerbations who fail to improve after first-line treatment and magnesium. These patients will also typically be candidates for admission to the pediatric ICU, and concern for respiratory failure may be high.

Epinephrine is both an α-agonist and β-agonist and induces smooth muscle relaxation as a result of systemic β-agonist effects and can be administered intramuscularly, subcutaneously, intravenously, or through nebulization. Epinephrine is typically administered intramuscularly in the ED because of more rapid availability and ease of administration, although the literature is mixed on improved efficacy of intravenous β-agonists.[58] Onset of action is 5 to 10 minutes if subcutaneous and immediate when given intravenously. The duration of action is <5 minutes intravenously. Most common side effects are anxiety, tremors, agitation, and sweating.

Terbutaline is pure β-agonist and is an option for hypertensive patients who may not tolerate epinephrine. Subcutaneous dosing is only for children older than 12 years of age. Side effects of terbutaline include tachycardia, arrhythmias, hypokalemia, and rarely myocardial ischemia.[59]

4.6 | Ketamine

Ketamine is an N-methyl-D-aspartate receptor antagonist that has dissociative, amnestic, and analgesic properties. Ketamine has been shown to promote bronchodilation and prevent bronchospasm.[60,61] Children with severe asthma in the ED who received a 2-hour ketamine infusion did not have improvement in respiratory rate, oxygen saturation, hospital admission rate, or need for mechanical ventilation when compared with normal saline.[62] However, ketamine has been shown to increase pulmonary compliance in mechanically ventilated patients.[63]

4.7 | Heliox

Helium-oxygen mixture (heliox) is a potential adjunct for severe asthma exacerbations refractory to first-line and other adjuncts, in particular in patients at risk for respiratory failure. Heliox's potential benefit is indirect—it enhances the delivery of other inhaled treatments through its lower gas density by decreasing flow resistance, lowering viscosity, and increasing laminar flow. Heliox is available in concentrations of 80% helium/20% oxygen and 70% helium/30% oxygen. If the patient requires >40% oxygen, the potential beneficial effect is diminished. Ideally heliox should be started early in the patient's presentation.[64–66] Heliox has limited use past the first 24 hours of disease and in patients with hypoxia.[67,68]

The literature on heliox in children is limited to case reports or case studies and has shown conflicting results. One study demonstrated no significant improvement in the recovery of pulmonary function in children treated with heliox.[69] However, a 2014 meta-analysis examining 113 children found that the use of heliox in delivering continuous albuterol therapy was associated with a significant improvement in peak expiratory flow.[70]

4.8 | High-flow nasal cannula

High-flow nasal cannula (HFNC) provides heated and humidified gas at a rate greater than typical inspiratory flow and is well tolerated by most patients. In addition, HFNC reduces anatomic dead space and potentially provides some degree of positive end-expiratory pressure.[71] The degree of positive end-expiratory pressure provided by HFNC is not as consistent as that provided by NIPPV.[72]

HFNC is primarily used to treat respiratory distress/failure with bronchiolitis, and the literature on HFNC in asthma is limited. One retrospective, single-center study of HFNC in 73 children with status asthmaticus demonstrated HFNC was feasible and likely safe, with only 2 instances of treatment failure—pneumothorax in 1 patient and another requiring escalation of therapy to NIPPV.[71]

4.9 | Non-invasive positive pressure ventilation (NIPPV)

Asthma, as a result of bronchiolar narrowing/closure, results in air-trapping within the alveoli, leading to dynamic hyperinflation. The patient must generate a higher negative inspiratory force to overcome the gradient to initiate inspiratory flow. If the patient cannot generate sufficient negative force to overcome the gradient, then

hypercarbia may ensue, leading to altered mental status, severe acidosis, and respiratory depression. This sequence of events is the most feared result of severe asthma and puts the patient at risk of cardiac arrest.

NIPPV refers to either bilevel positive airway pressure (BiPAP) or continuous positive airway pressure devices. Continuous positive airway pressure delivers a single degree of pressure support throughout the respiratory cycle, whereas BiPAP provides pressure support during both inspiration (inhalation positive airway pressure [IPAP]) and expiration (exhalation positive airway pressure). IPAP reduces the work by respiratory musculature.[73] Exhalation positive airway pressure allows for pressure equilibration between the mouth and alveoli, reducing the amount of force needed to generate inspiratory flow.

NIPPV is primarily indicated with severe asthma either insufficiently responsive to initial treatment or with extreme respiratory distress/ventilatory failure on arrival. The goal of NIPPV is to provide the patient with pressure support, ease the work of breathing/patient distress, and forestall or treat ventilatory failure. In a child, BiPAP and continuous positive airway pressure can be provided through a nasal mask or a full-face mask, although use may be limited by patient size and equipment availability. Typical settings for BiPAP are an IPAP of 10 cm H_2O and an exhalation positive airway pressure of 5 cm H_2O, with or without a backup ventilation rate.[59] Because these supports require NPO status, intravenous administration of glucose-containing fluids should be initiated in infants and considered in all patients. Children may require anxiolysis and other supportive measures to tolerate NIPPV. Allowing the child to sit on a caregiver's lap and involvement of a child-life specialist can ease the transition to respiratory support.

A 2016 Cochrane review evaluated 2 randomized controlled trials that investigated the efficacy of BiPAP in children presenting with asthma exacerbations. Both studies report that BiPAP improves respiratory rate, accessory muscle use, and dyspnea as compared with standard therapy, but the effect on overall outcomes are unclear.[74] Despite NIPPV use, 7% of patients will progress to require mechanical ventilation.[75]

4.10 | Endotracheal intubation

Endotracheal intubation and mechanical ventilation may be indicated rarely for severe asthma, but the decision should not be taken lightly. Avoiding intubation is the primary goal of the other treatments for severe asthma. Patients who are candidates for intubation are already at risk for complications, including respiratory and cardiac arrest, and intubation only increases that risk. The apnea associated with medication-assisted approaches to intubation inevitably worsens hypercarbia and respiratory acidosis. The reported rate of complications of endotracheal intubation is as high as 26%, including hypotension, pneumothorax, subcutaneous emphysema, and cardiac arrest.[76,77] Reported mortality after intubation is as high as 8%.[78] Worsening hypercarbia after the initiation of treatment is ominous, but hypercarbia in itself does not necessitate intubation. With aggressive therapy, intubation can be avoided.[79]

Criteria used to consider endotracheal intubation include poor response to maximum therapy, hypercarbia ($pCO2 > 50$ mmHg), severe hypoxemia ($PO2 < 60$ mmHg), worse mental status, impending respiratory arrest indicated by respiratory depression or bradycardia, worse metabolic acidosis, and cardiopulmonary arrest.[76]

Before the start of intubation attempts, asthma therapy should be maximized and a clear plan in place for cardiac arrest, with code doses of epinephrine and the defibrillator prepared. Ketamine is associated with bronchodilating properties and is a good choice for induction. As noted, the decision to use a paralytic medication specifically should be made with extreme caution and likely in consultation with critical care. As patients with severe asthma exacerbations will be intolerant of the resultant apnea; respiratory acidosis will worsen, maybe critically and irreversibly. If a paralytic medication is given, succinylcholine and rocuronium have rapid onset and are effective.[79] The patient should be oxygenated as well as possible before intubation. Cardiac arrest will often be preceded by worsening of hypoxemia and bradycardia, and loss of pulses may be subtle in a patient already in extremis. The care environment should therefore be as quiet as possible, with clear roles and responsibilities and anticipation of deterioration.

Immediately, post intubation, it is paramount to use adequate deep sedation in these patients as it can prevent patient–ventilator asynchrony. Deep sedation should be achieved with propofol or ketamine. Once intubated, general ventilation parameter goals include an oxygen saturation >91%, permissive hypercarbia, and a $pH > 7.2$.[76] Permissive hypercarbia is achieved through controlled hypoventilation and lower tidal volumes to prevent hyperinflation and minimize barotrauma while providing adequate ventilation and oxygenation. Despite the inclination to remedy the patient's hypercarbia, it is vital to remember that permissive hypercarbia is preferable to excessive pressures that can lead to pneumothoraces and breath stacking, which can lead to circulatory collapse.

Mechanical ventilation can also be very challenging in severe asthma, and coordination of care with a pediatric intensivist is strongly recommended. A pressure support approach is preferred, allowing the patient to set their own rate and tidal volume, although studies have looked at other common modes such as pressure support and volume control.[80] Ventilatory settings should use low tidal volumes (5–10 mL/kg), lower respiratory rates for age, inspiratory-to-expiratory time rates of 1:4 to 1:5, and plateau pressures <35 cm H_2O.[79,81] The goal of prolonged expiratory times is to avoid breath stacking.

If there is hemodynamic instability after endotracheal intubation, in particular bradycardia and/or hypotension, there is a step-by-step approach to troubleshoot the issue. First, disconnect the patient from the ventilator to allow his or her chest to naturally recoil; blood pressure should improve immediately. This hypotension is attributed to the decrease of systemic venous return (preload) as a result of worsening hyperinflation brought on by mechanical ventilation. If this maneuver is successful, the tidal volume and respiratory rate are too high and mechanical ventilation can be restarted at lower settings. Confirm endotracheal tube placement and ensure there is no dislodgement and suction the endotracheal tube to guarantee there is no obstruction. Consider use of a bedside ultrasound or chest X-ray to rule out the

presence of a pneumothorax, and lastly, verify there are no equipment issues.

4.11 | Other therapies

Methylxanthines, such as aminophylline and theophylline, were historically used for acute asthma exacerbations. In recent years, the National Asthma Education and Prevention Program Expert Panel recommended against the use of methylxanthines for treatment of hospitalized children with status asthmaticus.[35] However, there has been some literature to suggest that there may be an indication for theophylline in the hospitalized pediatric ICU asthmatic patient.[82] Leukotriene-modifying agents, such as montelukast, are not indicated in the treatment of an acute asthma exacerbation in the ED, although they are often used as adjunct therapies for maintenance in the outpatient setting in addition to an inhaled corticosteroid.[83] The efficacy of inhaled nitric oxide in severe asthma exacerbations is unknown.

5 | DISPOSITION

The ED disposition of a child with an asthma exacerbation is dependent on his or her projected clinical stability and response to treatment. The aforementioned asthma severity scores may aid in prognostication of the need for hospital admission (Table 2). Some EDs offer observation units for prolonged monitoring before discharge.[21,84] Recent internally validated asthma prediction models found several risk factors were strongly associated with hospital admission: oxygen saturation <94%, respiratory rate >31/min, history of pneumonia, and past ED visit for asthma in the prior 12 months.[21] Hospitalization or observation should be strongly considered for any child with (1) persistent hypoxemia despite treatment and observation, (2) mental status/inability to drink well worsening, (3) respiratory distress requiring bronchodilators every 2 hours or more, or (4) social or environmental factors that could limit a caregiver's ability to provide treatment. Children requiring ED escalation of care beyond nebulized short-acting β-agonist treatments every 2 hours may require ICU admission.[84]

Children with improving asthma symptoms may be discharged safely from the ED if they have (1) demonstrated manageable respiratory distress with short-acting β-agonist bronchodilators every 3 to 4 hours or more during their ED course, (2) do not pose imminent risk of clinical decompensation, and (3) have feasible asthma action plans in place with reliable caregivers.[85]

Discharge medications for outpatient prescription include appropriately dosed inhaled bronchodilators with spacer as well as counseling to continue the child's pertinent daily home medications including allergy medications and controller therapy with inhaled steroids. Children with little controller therapy are at higher risk for admission.[86]

In 2018, Parikh et al examined the value of the following 4 general discharge practices: (1) asthma education; (2) medications provided at discharge, including spacer, β-agonist, controller medication, and oral steroids for current or future exacerbation; (3) primary doctor contact by phone and with scheduled follow-up to manage longer term asthma management plans; (4) other post-discharge components including follow-up phone call to caregivers, home visit referrals, and environmental mitigation program referrals.[87] When evaluated individually, providing comprehensive education resulted in a 6% to 7% reduction in readmission rates at 3 months. Bundling of measures further reduced readmission rates. Similar studies report mixed results, suggesting that a follow-up phone call may reduce ED revisits after hospitalization.[88,89] In studies of caregivers of children with asthma, 90% believed that instruction regarding follow-up appointments, medications, reasons to seek medical care, and education in general were important aspects of the discharge process, preferably with live demonstration.[89,90] Factors associated with an increased risk of return ED visits within the year include younger age, lower socioeconomic and educational status, and chronic obstructive pulmonary disease.[91] In addition, previous critical care admission for asthma increased a child's risk for readmission for an asthma exacerbation.[92]

Consideration must be given to social determinants of health, as these may play a critical role in both effective treatment of individual patients and in eliminating disparities in care. Interventions supporting asthma education and counseling should address a spectrum of contextual and psychosocial factors that may not be included in historical or more traditional materials.[93,94] Examples include inner-city dwelling, exposure to air pollution, presence of pests, stress in the home, low income and other limitations affecting access to care should be considered prior to discharge. Physician counseling, coordination with primary care from the ED, and social work consult from the ED visit may support a transition to outpatient care. Perhaps most important, effectively addressing the social determinants of health will almost certainly require multidisciplinary, institution-level commitments to initiatives that partner with community members and both government and nongovernmental groups.

6 | CONCLUSION

Asthma exacerbation in children is a common presentation in the ED often requiring escalation of care and hospital admission. Clinical scoring tools are important ED adjuncts to guide treatment, prognosticate disposition, and inform admission. Albuterol, ipratropium, and dexamethasone are the mainstays of acute treatment of severe acute asthma exacerbations in children. All 3 agents have been shown to prevent hospitalization and improve other short-term outcomes. More severe asthma exacerbations should be treated with magnesium. Endotracheal intubation should be approached with extreme caution given the high mortality rate and complications.

CONFLICT OF INTERESTS

The authors have no conflicts of interest to disclose.

AUTHOR CONTRIBUTIONS

Moon O. Lee, Cherrelle Smith, Nicholas Pokrajac, Shyam Sivasankar, and Angela Lumba-Brown have made substantial contributions to

conception and design, acquisition of data, or analysis and interpretation of data and have been involved in drafting the manuscript and revising it critically for important intellectual content, giving final approval of the version to be published.

REFERENCES

1. Moorman JE, Akinbami LJ, Bailey CM, et al. National surveillance of asthma: United States, 2001-2010. *Vital Health Stat* 2012;3(35):1-58.
2. QuickStats: Percentage of all emergency department (ED) visits made by patients with asthma, by sex and age group — National hospital ambulatory medical care survey, United States 2014–2015. *MMWR Morb Mortal Wkly Rep* 2018;67:167.
3. Akinbami LJ, Moorman JE, Garbe PL, Sondik EJ. Status of childhood asthma in the United States, 1980–2007. *Pediatrics*. 2009;123(suppl 3):S131-S145.
4. Nath JB, Hsia RY. Children's emergency department use for asthma, 2001–2010. *Acad Pediatr*. 2015;15(2):225-230.
5. Walls TA, Hughes NT, Mullan PC, Chamberlain JM, Brown K. Improving pediatric asthma outcomes in a community emergency department. *Pediatrics*. 2017;139(1):e20160088.
6. Bhogal SK, McGillivray D, Bourbeau J, Benedetti A, Bartlett S, Ducharme FM. Early administration of systemic corticosteroids reduces hospital admission rates for children with moderate and severe asthma exacerbation. *Ann Emerg Med*. 2012;60(1):84-91.e3.
7. Bekmezian A, Fee C, Weber E. Clinical pathway improves pediatrics asthma management in the emergency department and reduces admissions. *J Asthma*. 2015;52(8):806-814.
8. Rutman L, Migita R, Spencer S, Kaplan R, Klein EJ. Standardized Asthma Admission Criteria Reduce Length of Stay in a Pediatric Emergency Department. Walthall J, ed. *Acad Emerg Med*. 2016;23(3):289-296. https://doi.org/10.1111/acem.12890
9. Papadopoulos NG, Arakawa H, Carlsen K-H, et al. International consensus on (ICON) pediatric asthma. *Allergy*. 2012;67(8):976-997.
10. Bekhof J, Reimink R, Brand PLP. Systematic review: Insufficient validation of clinical scores for the assessment of acute dyspnoea in wheezing children. *Paediatr Respir Rev*. 2014;15(1):98-112.
11. Arnold DH, Gebretsadik T, Abramo TJ, Hartert TV. Noninvasive testing of lung function and inflammation in pediatric patients with acute asthma exacerbations. *J Asthma*. 2012;49(1):29-35.
12. Eggink H, Brand P, Reimink R, Bekhof J. Clinical scores for dyspnoea severity in children: a prospective validation study. Latzin P, ed. *PLOS ONE*. 2016;11(7):e0157724.
13. Florin TA, Ambroggio L, Brokamp C, et al. Reliability of examination findings in suspected community-acquired pneumonia. *Pediatrics*. 2017;140(3):e20170310.
14. Ullmann N, Mirra V, Di Marco A, et al. Asthma: differential diagnosis and comorbidities. *Front Pediatr*. 2018;6:276.
15. Schneider WV, Bulloch B, Wilkinson M, Garcia-Filion P, Keahey L, Hostetler M. Utility of portable spirometry in a pediatric emergency department in children with acute exacerbation of asthma. *J Asthma*. 2011;48(3):248-252
16. Arnold DH, Gebretsadik T, Hartert TV. Spirometry and PRAM severity score changes during pediatric acute asthma exacerbation treatment in a pediatric emergency department. *J Asthma*. 2013;50(2):204-208.
17. Bekhof J, Reimink R, Bartels I-M, Eggink H, Brand PLP. Large observer variation of clinical assessment of dyspnoeic wheezing children. *Arch Dis Child*. 2015;100(7):649-653.
18. Johnson MD, Nkoy FL, Sheng X, Greene T, Stone BL, Garvin J. Direct concurrent comparison of multiple pediatric acute asthma scoring instruments. *J Asthma*. 2017;54(7):741-753.
19. Gorelick MH, Stevens MW, Schultz TR, Scribano PV. Performance of a novel clinical score, the Pediatric Asthma Severity Score (PASS), in the evaluation of acute asthma. *Acad Emerg Med*. 2004;11(1):10-18.
20. Chalut DS, Ducharme FM, Davis GM. The Preschool Respiratory Assessment Measure (PRAM): A responsive index of acute asthma severity. *J Pediatr*. 2000;137(6):762-768.
21. Arnold DH, Gebretsadik T, Moons KGM, Harrell FE, Hartert TV. Development and internal validation of a pediatric acute asthma prediction rule for hospitalization. *J Allergy Clin Immunol Pract*. 2015;3(2):228-235.
22. Kelly CS, Andersen CL, Pestian JP, et al. Improved outcomes for hospitalized asthmatic children using a clinical pathway. *Ann Allergy Asthma Immunol*. 2000;84(5):509-516.
23. Parkin PC, Macarthur C, Saunders NR, Diamond SA, Winders PM. Development of a clinical asthma score for use in hospitalized children between 1 and 5 years of age. *J Clin Epidemiol*. 1996;49(8):821-825.
24. Gouin S, Robidas I, Gravel J, Guimont C, Chalut D, Amre D. Prospective evaluation of two clinical scores for acute asthma in children 18 Months to 7 years of age: two pediatric asthma clinical scores. *Acad Emerg Med*. 2010;17(6):598-603.
25. Alnaji F, Zemek R, Barrowman N, Plint A. PRAM score as predictor of pediatric asthma hospitalization. Walthall J, ed. *Acad Emerg Med*. 2014;21(8):872-878.
26. Gray MP, Keeney GE, Grahl MJ, Gorelick MH, Spahr CD. Improving guideline-based care of acute asthma in a pediatric emergency department. *Pediatrics*. 2016;138(5):e20153339.
27. Johnson DP, Arnold DH, Gay JC, et al. Implementation and improvement of pediatric asthma guideline improves hospital-based care. *Pediatrics*. 2018;141(2):e20171630.
28. Alherbish M, Mobaireek K, Alangari A. Admission predictability of children with acute asthma. *Ann Thorac Med*. 2018;13(1):36.
29. Paniagua N, Elosegi A, Duo I, et al. Initial asthma severity assessment tools as predictors of hospitalization. *J Emerg Med*. 2017;53(1):10-17.
30. Ryan KS, Son S, Roddy M, et al. Pediatric asthma severity scores distinguish suitable inpatient level of care for children admitted for status asthmaticus. *J Asthma*. 2019:1-9. https://doi.org/10.1080/02770903.2019.1680998
31. Maue DK, Krupp N, Rowan CM. Pediatric asthma severity score is associated with critical care interventions. *World J Clin Pediatr*. 2017;6(1):34.
32. https://www.seattlechildrens.org/globalassets/documents/healthcare-professionals/clinical-standard-work/asthma_pathway.pdf. Accessed June 29, 2020.
33. Asthma Clinical Pathway—Emergency Children's Hospital of Philadelphia. https://www.chop.edu/clinical-pathway/asthma-emergent-care-clinical-pathway. Accessed June 29, 2020.
34. Lexicomp. http://online.lexi.com.laneproxy.stanford.edu/lco/action/doc/retrieve/docid/stanu_f/119018?searchUrl=%2Flco%2Faction%2Fsearch%3Bjsessionid%3DBBC74EE0BA9900D24228006F35D20BA6%3Forigin%3Dapi%26t%3Dglobalid%26q%3D5577%26nq%3Dtrue. Accessed June 25, 2020.
35. *Expert Panel Report 3: Guidelines for the Diagnosis and Management of Asthma*. Bethesda, MD: National Heart, Lung, and Blood Institute; 2007.
36. Cates CJ, Welsh EJ, Rowe BH. Holding chambers (spacers) versus nebulisers for beta-agonist treatment of acute asthma. *Cochrane Database Syst Rev*. 2013;9:CD000052. https://doi.org/10.1002/14651858.CD000052.pub3
37. Castro-Rodriguez JA, Rodrigo GJ. β-agonists through metered-dose inhaler with valved holding chamber versus nebulizer for acute exacerbation of wheezing or asthma in children under 5 years of age: a systematic review with meta-analysis. *J Pediatr*. 2004;145(2):172-177.

38. Parlar-Chun R, Arnold K. Association of various weight-based doses of continuous albuterol on hospital length of stay. *J Asthma*. 2020:1-6. https://doi.org/10.1080/02770903.2020.1723622
39. Lexicomp. http://online.lexi.com.laneproxy.stanford.edu/lco/action/doc/retrieve/docid/stanu_f/2910515?cesid=aEvu4iypveh&searchUrl=%2Flco%2Faction%2Fsearch%3Fq%3Dipratriopium&t%3Dname&va%3Dipratriopium#parentdoc-tab-content. Accessed June 25, 2020.
40. Qureshi F, Zaritsky A, Poirier MP. Comparative efficacy of oral dexamethasone versus oral prednisone in acute pediatric asthma. *J Pediatr*. 2001;139(1):20-26.
41. Rehder KJ. Adjunct therapies for refractory status asthmaticus in children. *Respir Care*. 2017;62(6):849-865.
42. Rowe BH, Spooner C, Ducharme F, Bretzlaff J, Bota G. Early emergency department treatment of acute asthma with systemic corticosteroids. *Cochrane Database Syst Rev*. 2001;1:CD002178. https://doi.org/10.1002/14651858.CD002178
43. Lexicomp. http://online.lexi.com.laneproxy.stanford.edu/lco/action/doc/retrieve/docid/pdh_f/2838039?searchUrl=%2Flco%2Faction%2Fsearch%3Forigin%3Dapi&t%3Dglobalid&q%3D388152&nq%3Dtrue#admp. Accessed July 13, 2020.
44. Cronin JJ, McCoy S, Kennedy U, et al. A randomized trial of single-dose oral dexamethasone versus multidose prednisolone for acute exacerbations of asthma in children who attend the emergency department. *Ann Emerg Med*. 2016;67(5):593-601.e3.
45. Kravitz J, Dominici P, Ufberg J, Fisher J, Giraldo P. Two days of dexamethasone versus 5 days of prednisone in the treatment of acute asthma: a randomized controlled trial. *Ann Emerg Med*. 2011;58(2):200-204.
46. Watnick CS, Fabbri D, Arnold DH. Single-dose oral dexamethasone is effective in preventing relapse after acute asthma exacerbations. *Ann Allergy Asthma Immunol*. 2016;116(2):171-172.
47. Meyer JS, Riese J, Biondi E. Is dexamethasone an effective alternative to oral prednisone in the treatment of pediatric asthma exacerbations? *Hosp Pediatr*. 2014;4(3):172-180.
48. Andrews AL, Wong KA, Heine D, Scott Russell W. A cost-effectiveness analysis of dexamethasone versus prednisone in pediatric acute asthma exacerbations: a cost-effectiveness analysis of dexamethasone for acute asthma. *Acad Emerg Med*. 2012;19(8):943-948.
49. Keeney GE, Gray MP, Morrison AK, et al. Dexamethasone for acute asthma exacerbations in children: a meta-analysis. *Pediatrics*. 2014;133(3):493-499.
50. Gordon S, Tompkins T, Dayan PS. Randomized trial of single-dose intramuscular dexamethasone compared with prednisolone for children with acute asthma. *Pediatr Emerg Care*. 2007;23(8):521-527.
51. Gries DM, Moffitt DR, Pulos E, Carter ER. A single dose of intramuscularly administered dexamethasone acetate is as effective as oral prednisone to treat asthma exacerbations in young children. *J Pediatr*. 2000;136(3):298-303.
52. Klig JE, Hodge D, Rutherford MW. Symptomatic improvement following emergency department management of asthma: a pilot study of intramuscular dexamethasone versus oral prednisone. *J Asthma*. 1997;34(5):419-425.
53. Gürkan F, Haspolat K, Boşnak M, Dikici B, Derman O, Ece A. Intravenous magnesium sulphate in the management of moderate to severe acute asthmatic children nonresponding to conventional therapy. *Eur J Emerg Med*. 1999;6(3):201-205.
54. Lexicomp. http://online.lexi.com.laneproxy.stanford.edu/lco/action/doc/retrieve/docid/patch_f/5925339?cesid=ae6QtI2wuRM&searchUrl=%2Flco%2Faction%2Fsearch%3Fq%3Depinephrine&t%3Dname&va%3Depinephrine#adr. Accessed June 26, 2020.
55. Griffiths B, Kew KM. Intravenous magnesium sulfate for treating children with acute asthma in the emergency department. *Cochrane Database Syst Rev*. 2016;4(4):CD011050.
56. Ciarallo L, Sauer AH, Shannon MW. Intravenous magnesium therapy for moderate to severe pediatric asthma: Results of a randomized, placebo-controlled trial. *J Pediatr*. 1996;129(6):809-814.
57. Ciarallo L, Brousseau D, Reinert S. Higher-dose intravenous magnesium therapy for children with moderate to severe acute asthma. *Arch Pediatr Adolesc Med*. 2000;154(10):979.
58. Travers AH, Milan SJ, Jones AP, Camargo CA Jr, Rowe BH. Addition of intravenous beta $_2$ -agonists to inhaled beta $_2$-agonists for acute asthma. *Cochrane Database Syst Rev*. 2012. https://doi.org/10.1002/14651858.CD010179
59. Nievas IFF, Anand KJS. Severe acute asthma exacerbation in children: a stepwise approach for escalating therapy in a pediatric intensive care unit. *J Pediatr Pharmacol Ther*. 2013;18(2):88-104.
60. Gateau O, Bourgain J-L, Gaudy J-H, Benveniste J. Effects of ketamine on isolated human bronchial preparations. *Br J Anaesth*. 1989;63(6):692-695.
61. Cook DJ, Carton EG, Housmans PR. Mechanism of the positive inotropic effect of ketamine in isolated ferret ventricular papillary muscle. *Anesthesiology*. 1991;74(5):880-888.
62. Allen JY, Macias CG. The efficacy of ketamine in pediatric emergency department patients who present with acute severe asthma. *Ann Emerg Med*. 2005;46(1):43-50.
63. Youssef-Ahmed MZ, Silver P, Nimkoff L, Sagy M. Continuous infusion of ketamine in mechanically ventilated children with refractory bronchospasm. *Intensive Care Med*. 1996;22(9):972-976.
64. Rivera ML, Kim TY, Stewart GM, Minasyan L, Brown L. Albuterol nebulized in heliox in the initial ED treatment of pediatric asthma: a blinded, randomized controlled trial. *Am J Emerg Med*. 2006;24(1):38-42.
65. Kim IK. Helium/oxygen-driven albuterol nebulization in the treatment of children with moderate to severe asthma exacerbations: a randomized, controlled trial. *Pediatrics*. 2005;116(5):1127-1133.
66. Rodrigo GJ, Pollack CV, Rodrigo C, Rowe BH. Heliox for non-intubated acute asthma patients. *Cochrane Database Syst Rev*. 2006;4:CD002884. https://doi.org/10.1002/14651858.CD002884.pub2
67. Gupta VK, Cheifetz IM. Heliox administration in the pediatric intensive care unit: an evidence-based review: *Pediatr Crit Care Med*. 2005;6(2):204-211.
68. Wade A, Chang C. Evaluation and treatment of critical asthma syndrome in children. *Clin Rev Allergy Immunol*. 2015;48(1):66-83.
69. Rodrigo GJ, Rodrigo C, Pollack CV, Rowe B. Use of helium-oxygen mixtures in the treatment of acute asthmaa. *Chest*. 2003;123(3):891-896.[CrossRef]
70. Rodrigo GJ, Castro-Rodriguez JA. Heliox-driven β2-agonists nebulization for children and adults with acute asthma: a systematic review with meta-analysis. *Ann Allergy Asthma Immunol*. 2014;112(1):29-34.
71. Baudin F, Buisson A, Vanel B, Massenavette B, Pouyau R, Javouhey E. Nasal high flow in management of children with status asthmaticus: a retrospective observational study. *Ann Intensive Care*. 2017;7(1):55.
72. Craig SS, Dalziel SR, Powell CV, Graudins A, Babl FE, Lunny C. Interventions for escalation of therapy for acute exacerbations of asthma in children: an overview of Cochrane Reviews. *Cochrane Database Syst Rev*. 2018. https://doi.org/10.1002/14651858.CD012977
73. Najaf-Zadeh A, Leclerc F. Noninvasive positive pressure ventilation for acute respiratory failure in children: a concise review. *Ann Intensive Care*. 2011;1(1):15.
74. Korang SK, Feinberg J, Wetterslev J, Jakobsen JC. Non-invasive positive pressure ventilation for acute asthma in children. *Cochrane Database Syst Rev*. 2016. https://doi.org/10.1002/14651858.CD012067.pub2
75. Mayordomo-Colunga J, Medina A, Rey C, et al. Non-invasive ventilation in pediatric status asthmaticus: A prospective observational study. *Pediatr Pulmonol*. 2011;46(10):949-955.
76. Jones BP, Paul A. Management of acute asthma in the pediatric patient: an evidence-based review. *Pediatr Emerg Med Pract*. 2013;10(5):1-23; quiz 23-24.

77. Krishnan V, Diette GB, Rand CS, et al. Mortality in patients hospitalized for asthma exacerbations in the United States. *Am J Respir Crit Care Med*. 2006;174(6):633-638.
78. McFadden ER. Acute severe asthma. *Am J Respir Crit Care Med*. 2003;168(7):740-759.
79. Sabato K, Hanson JH. Mechanical ventilation for children with status asthmaticus. *Respir Care Clin N Am*. 2000;6(1):171-188.
80. Cox RG, Barker GA, Bohn DJ. Efficacy, results, and complications of mechanical ventilation in children with status asthmaticus. *Pediatr Pulmonol*. 1991;11(2):120-126.
81. Sarnaik AP, Daphtary KM, Meert KL, Lieh-Lai MW, Heidemann SM. Pressure-controlled ventilation in children with severe status asthmaticus. *Pediatr Crit Care Med*. 2004;5(2):133-138.
82. Montserrat JM, Barberà JA, Viegas C, Roca J, Rodriguez-Roisin R. Gas exchange response to intravenous aminophylline in patients with a severe exacerbation of asthma. *Eur Respir J*. 1995;8(1):28-33.
83. Romanet S, Stremler-Lebel N, Magnan A, Dubus JC. Role of leukotriene inhibitors in the treatment of childhood asthma. Archives de Pediatrie: Organe Officiel de la Societe Francaise de Pediatrie 7.9. 2020:969-975.
84. Jean T, Yang S-J, Crawford WW, Takahashi SH, Sheikh J. Development of a pediatric asthma predictive index for hospitalization. *Ann Allergy Asthma Immunol*. 2019;122(3):283-288.
85. Lo H, Messer A, Loveless J, et al. Discharging asthma patients on 3-hour β-agonist treatments: a quality improvement project. *Hosp Pediatr*. 2018;8(12):733-739.
86. Belhassen M, Langlois C, Laforest L, et al. Level of asthma controller therapy before admission to the hospital. *J Allergy Clin Immunol Pract*. 2016;4(5):877-883.
87. Parikh K, Hall M, Kenyon CC, et al. Impact of discharge components on readmission rates for children hospitalized with asthma. *J Pediatr*. 2018;195:175-181.e2.
88. Teufel RJ, Shuler AB, Ebeling MD, Morella K, Andrews AL. Enhancing postdischarge asthma care by using pharmacy claims and telephone follow-up. *Hosp Pediatr*. 2018;8(5):251-259.
89. Vepraskas SH, O'Day P, Zhang L, Simpson P, Gage S. Parents support teach-back, demonstration, and a postdischarge phone call to augment discharge education. *Hosp Pediatr*. 2018;8(12):778-784.
90. Samuels-Kalow ME, Stack AM, Porter SC. Effective discharge communication in the emergency department. *Ann Emerg Med*. 2012;60(2):152-159.
91. To T, Zhu J, Ryckman K, Gershon A. Risk factors for return to the emergency department for asthma: a population-based study. *J Allergy Clin Immunol Pract*. 2018;6(6):1907-1913.e4.
92. Tse SM, Samson C. Time to asthma-related readmission in children admitted to the ICU for asthma. *Pediatr Crit Care Med*. 2017;18(12):1099-1105.
93. Williams DR, Sternthal M, Wright RJ. Social determinants: taking the social context of asthma seriously. *Pediatrics*. 2009;123(suppl 3):S174-S184.
94. Fisher EB. Community organization to reduce the need for acute care for asthma among African American children in low-income neighborhoods: the neighborhood asthma coalition. *Pediatrics*. 2004;114(1):116-123.

How to cite this article: Lee MO, Sivasankar S, Pokrajac N, Smith C, Lumba-Brown A. Emergency department treatment of asthma in children: A review. *JACEP Open*. 2020;1:1552–1561. https://doi.org/10.1002/emp2.12224