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Management of Children with Severe Asthma Exacerbation in the Emergency Department

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Contents

Abstract

Although acute asthma is a very common cause of emergency department visits in children, there is as yet insufficient evidence for the establishment of a standardized treatment protocol. The aim of this review is to describe updated information on the management of asthma exacerbations in the pediatric emergency department.

Oxygen is the first-line treatment of acute asthma exacerbations in the emergency department to control hypoxemia. It is accompanied by the administration of β_2-adrenoceptor agonists followed by corticosteroids. β_2-Adrenoceptor agonists have traditionally been administered by nebulization, although spacers have recently been introduced and proven, in many cases, to be as effective as nebulization.

Oral prednisolone, with its reliability, simplicity, convenience and low cost, should remain the treatment of choice for the most severe asthma exacerbations, when the lung airways are extremely contracted and filled with secretions. Recently, several studies have shown that high-dose inhaled corticosteroids are at least as effective as oral corticosteroids in controlling moderate to severe asthma attacks in children and therefore should be considered an alternative treatment to oral corticosteroids in moderate to severe asthma attacks.

Studies of other drugs have shown that ipratropium bromide may be given only in addition to β_2-adrenoceptor agonists; theophylline has no additional benefit, and magnesium sulfate has no clear advantage.

Comprehensive asthma management should also include asthma education, measures to prevent asthma triggers, and training in the use of inhalers and spacers. Proper management will avoid most asthma attacks and reduce admission and readmission to emergency departments.

Hospital admissions for childhood asthma have increased dramatically **((Author: worldwide or in certain countries?))** over the past 2 decades.[1] Burt and Knapp[2] reported an estimated 13.7 million ambulatory care visits for **((Author: childhood?))** asthma in 1993-94 in the US, 12% of them to the emergency department (ED). Furthermore, 10 to 17% of patients **((Author: children?))** discharged from the ED after initial treatment require additional interventions within 2 weeks. [3,4]

An acute asthma exacerbation is defined as the abrupt onset or worsening of symptoms of shortness of breath, wheezing, chest tightness and breathlessness, associated with respiratory distress and a decrease in expiratory airflow.[5] The severity of asthma attacks is difficult to define because asthma, by its nature, is a fluctuating disorder, with aggravations and improvements. The Global Initiative for Asthma[6] stipulated two measurable parameters of asthma exacerbation severity: (i) moderate exacerbation – peak expiratory flow rate (PEFR) 60 to 80% of predicted and/or oxygen saturation of 91 to 95%; and (ii) severe exacerbation – PEFR <60% and/or oxygen saturation <90%.

Oxygen, inhaled β_2-adrenoceptor agonists and corticosteroids remain the cornerstones of therapy for the child with severe exacerbation of asthma. The exact methods of administration of these drugs have not been fully established, though guidelines were formulated in 1991 by the National Asthma Education Program.[7]

The aim of this article is to review and discuss the most recently published data on the management of children with moderate and severe asthma exacerbation in the ED. Almost all the papers reviewed describe randomized, controlled studies. The few uncontrolled studies are so indicated in the text, and they are cited only when the relevant information was unavailable from a controlled study. A comprehensive Medline search was conducted, and all relevant Cochrane studies were reviewed. All information found up to the time of completion of this review **((Author: can you give the month and year?))** was included.

1. Oxygen

Oxygen has long been considered essential for the management of acute asthma. During severe exacerbations, some lung units are subjected to over- or under-inflation because of changes in airway resistance. These changes lead to ventilation/perfusion mismatch[8] and thereby to hypoxemia.

The administration of a nebulized β_2-adrenoceptor agonist can increase the blood flow in relatively poorly ventilated areas of the lung.[9] This may in turn increase the ventilation/perfusion mismatch[10] and worsen the hypoxemia. Therefore, in severe asthma attacks, β2-adrenoceptor agonists should always be given with oxygen.

Arterial oxygen desaturation after bronchodilator therapy is well recognized in acute asthma.[11] Typically, the maximum decrease in saturation occurs within 5 to 10 min of treatment and is self-limited within 15 min. The importance of continued oxygen treatment after mobilization has been emphasized.[12] Connett and Lenney[13]demonstrated pneumonic consolidation on chest radiography in children with acute asthma who showed a prolonged drop in oxygen saturation (up to 20 min) after treatment with albuterol (salbutamol). They suggested that the vasodilator effect of albuterol can overcome hypoxic pulmonary vasoconstriction, and this would cause increased perfusion of consolidated areas and, consequently, a decrease in saturation.

To prevent hypoxemia, which can be an early sign of airway obstruction, oxygen saturation above 92% needs to be maintained.[8] In young infants, an initial oxygen saturation of <91% in room air is an indication for hospitalization.[11] Patients with initial saturation of 95% or more rarely relapse after discharge.[14]

2. Bronchodilators

2.1 Nebulization of β_2-Adrenoceptor Agonists

In acute exacerbations of asthma, lung airways become narrowed as a result of mucosal edema, hypersecretion and bronchospasm. Depending on the severity of the attack, treatment with bronchodilators is always required, in addition to oxygen and corticosteroids.

β_2-Adrenoceptor agonists are the most effective bronchodilators, owing to their rapid onset of action and the magnitude of bronchodilation achieved;[15,16] they yield the greatest benefit when delivered to the peripheral airways. However, this is difficult in acute asthma because of the narrowed airways and faster respiratory rate, which could lead to an increase in drug deposition in the throat and large airways.

Many clinicians recommend wet nebulization for delivery of β_2-adrenoceptor agonists in the ED.[17] A mist of β_2-adrenoceptor agonist diluted in saline is created, and the patient inhales the mist by tidal breathing through a mask. Nebulization should always be with oxygen. However, if the attack is not severe enough for oxygen, spacers rather than nebulizers should be used.

2.2 β_2-Adrenoceptor Agonists via Holding Chambers (Spacers)

Nebulization is performed with supplemental oxygen and requires a supply of compressed gas or a power source. Recently, metered-dose inhalers (MDI) with a holding chamber (spacer)

have been introduced for the treatment of acute asthma in children. The β_2-adrenoceptor agonist is delivered into the chamber, which is then emptied actively by the patient using a mouthpiece or mask. Studies have found this method to be as effective as nebulization.[18-21]

A recent meta-analysis found that outcomes such as hospitalization, lung function tests, respiratory rate and tremor were equally good with nebulizers and holding chambers.[20] However, children using holding chambers had a significantly shorter stay in the ED, a smaller increase in pulse rate and better oxygenation. The study authors suggested that on the basis of available evidence, in most cases, holding chambers could be substituted for nebulizers in the ED. Chou et al.,[22] in a randomized study, compared asthma treatment in the ED via spacers and nebulizers in 152 children. They found that the spacer group had a significantly shorter stay in the ED (66 *vs* 103 min, $p < 0.001$), a significantly smaller increase in pulse rate (5*vs* 15%, $p < 0.001$) and a significantly lower rate of episodes of vomiting (9*vs* 20%, $p < 0.04$). Parkin(**(Author: spelling differs in ref. list; please resolve)**) et al.,[21] in a randomized trial of 60 children aged 1 to 5 years with moderate asthma exacerbation, showed that after stabilization in the ED, treatment with MDI and spacer [albuterol 4 to 6 puffs (400 to 600μg) + ipratropium bromide 2 puffs (400μg)] was as effective as treatment with nebulization (albuterol 0/15 m/kg **((Author: do you mean 0.15 mg/kg?))** + ipratropium bromide 125μg). In this study, nurses preferred the nebulization route. Starting children on the use of MDI and spacer in the ED or during hospitalization makes it easier to continue at-home management and/or long-term prophylaxis; it is also less expensive. Boweton et al.[23] reported a 25% reduction in monthly costs after nebulizers were replaced by MDIs in a large adult tertiary center.

Kamps et al.,[24] in a study of 66 children aged 1 to 4 years, observed a poor inhalation technique more frequently in the children using a dry-powder inhaler with holding chamber than in the children using an MDI with holding chamber. Furthermore, the proper inhalation technique was practiced by only 39% of the children given a single instruction session compared with 93% of those who underwent comprehensive instruction with repeated checks.

Most of the studies so far have used repeated doses of β_2-adrenoceptor agonists at short intervals (e.g. one respule by nebulizer or four actuations of an MDI with holding chamber every 10 to 15 min).[20] To overcome the uncertainty of dosage delivery from different devices, the number of treatments is adjusted according to the individual patient response.[20] The recommended dose for MDI with holding chamber[7] is 4 to 8 puffs every 20 min for the first 3h, and then every 1 to 4h on an as-needed basis. Nebulization should be performed every 20 min for the first 3h at a dose of 0.15 mg/kg, provided that the maximum dose does not exceed 10mg every 1 to 4h.[7]

2.3 Subcutaneous β_2-Adrenoceptor Agonists

Subcutaneous epinephrine or terbutaline has no advantage over inhaled β_2-adrenoceptor agonists, except in uncooperative patients.[25]In a prospective, randomized study of 154 adult patients with acute asthma, subcutaneous epinephrine or orciprenaline (metaproterenol) was found to be as effective as nebulized orciprenaline.[26] Subcutaneous epinephrine or terbutaline should be administered at a dose of 0.01 mg/kg (maximum 0.3mg).[7]

2.4 Continuous Nebulization of β_2-Adrenoceptor Agonists

Browne et al.,[27] in a prospective, randomized study of 17 children with impending respiratory failure due to status asthmaticus who were treated in the pediatric intensive care unit, showed that continuous nebulization of albuterol (0.3 mg/kg/h) was safe and was found to induce more rapid clinical improvement than intermittent nebulization (0.3 mg/kg/h over 20 min every hour). However, Porthoy et al.[28] reported that continuous nebulization of albuterol was associated with adverse events such as muscle cramps, hypokalemia and hyperglycemia. It should therefore be considered only in the hospital setting and only in the presence of a risk of significant obstruction, before institution of parenteral β_2-adrenoceptor agonists or mechanical ventilation.

2.5 Parenteral β_2-Adrenoceptor Agonists

Parenteral β_2-adrenoceptor agonists may be considered in patients who have failed to respond to inhaled or subcutaneous therapy. Browne et al.[27] conducted a double-blind, randomized, controlled study in 29 children aged 1 to 12 years with acute asthma in the ED who were refractory to nebulized albuterol (2.5mg for age ≤2 years and 5.0mg for age >2 years). They found that adding intravenous albuterol 15 μg/kg (over 10 min) to the standard treatment of nebulized albuterol, continuous oxygen, and intravenous hydrocortisone (5 mg/kg) yielded a significantly more rapid clinical improvement than adding saline (control group) [$p = 0.03$]. Children in the study group were also less dependent than controls on oxygen after 2h, and they were ready for discharge from the ED 9.7h earlier ($p < 0.05$). By contrast, however, Barnes[25] found that side effects such as tachycardia, hypokalemia and cardiac arrhythmias were more frequent with parenteral than with inhaled therapy. Further studies are still needed to resolve this issue.

3. Corticosteroids

3.1 Oral Corticosteroids

The airway edema and secretions associated with acute asthma are best treated with anti-inflammatory agents. The timing, route, dose and target population may vary markedly. In a meta-analysis of 12 randomized, controlled trials, Rowe et al.[29] reported that early (within 1h of presentation) use of corticosteroids for acute asthma in the ED significantly reduced admission rates. Benefits were greatest in patients with more severe asthma and those not currently receiving corticosteroids.[29]Oral therapy was particularly effective in children. Adverse effects were not significantly different from those with placebo.

Other studies have shown that oral prednisolone, **((Author: given?))** within 4h, significantly reduced the need for hospitalization, especially in children with more severe attacks[30] and higher relapse rates.[8] Storr et al.[31] achieved good results with early administration of a single dose of oral prednisolone (30mg for children <5 years of age, otherwise 60mg) in addition to β_2-adrenoceptor agonists. Lapin and Cloutier[32] also noted a significant decrease in the number of relapses and in β_2-adrenoceptor agonist use with a short course of 1 to 2 mg/kg corticosteroids.

3.2 Intramuscular and Intravenous Corticosteroids

Early administration of a single dose of 4 mg/kg intramuscular methylprednisolone in addition to **((Author: a?))** β_2-adrenoceptor agonist in children with an acute asthma attack in the ED significantly reduced the hospital admission rate.[33] At discharge from the ED, intramuscular or oral corticosteroid treatment can prevent relapse and reduce β-adrenoceptor agonist use.[34] A comparison of intravenous corticosteroids (an initial bolus of 2 mg/kg methylprednisolone followed by 1 mg/kg every 6h) with placebo[35] demonstrated a greater rate of improvement in clinical score index, more rapid recovery from peripheral airway obstruction, and longer incidence of relapse within 4 weeks of discharge in the study group.[35]

3.3 Inhaled Corticosteroids

Inhaled corticosteroids have been shown to be effective alternatives to oral corticosteroids in long-term asthma therapy, reducing or even eliminating oral corticosteroid requirements.[36-44] Inhaled corticosteroids have a potential benefit also in the acute setting because they are delivered directly to the airways, induce a reduction in airway reactivity and have fewer systemic adverse effects.[37] According to current data, their use in relatively high doses should be confined to moderate to severe asthma exacerbations. They are not applicable for the most severe exacerbations in which lung airways are extremely contracted and filled with secretions,[38-45] where oral corticosteroids should be given.

Recently, five randomized, double-blind, controlled studies,[39-42,44] including a total of 580 children aged 3 to 17 years, compared treatment with oral corticosteroids to a relatively high (initial) dose of inhaled corticosteroids. The schedules were as follows: nebulized budesonide 800μg in three doses at 30-min intervals;[41] nebulized budesonide 2000μg every 8h;[39] budesonide 1600μg via Turbohaler®**1**[42]nebulized dexamethasone 1.5 mg/kg;[40] and nebulized fluticasone propionate 1000μg twice daily.[44] Inhaled corticosteroids were consistently found to be at least as effective as oral corticosteroids (2 mg/kg) in controlling moderate to severe asthma attacks. In one of these studies,[41] which included 80 children aged 3 to 12 years, the patients given budesonide showed a significantly greater improvement than those given oral prednisolone in oxygen saturation, respiratory rate, pulmonary index, and respiratory distress score ($p < 0.01$). A higher proportion of the **((Author: inhaled corticosteroid?))** group was fit for discharge at 2h after the third dose of nebulization **((Author: compared with the oral corticosteroid group?))** ($p < 0.001$). Treatment with inhaled corticosteroids was not associated with suppression of corticosteroid secretion, as observed during treatment with oral corticosteroids **((Author: please confirm that rewording is ok))**.[42]These findings were supported by the systematic review of Edmonds et al.[38] Analysis of the pooled results of the children and adults (n-**((Author: should this be 'n ='?))**352) showed that inhaled corticosteroid therapy reduced hospital admissions by 55% compared with placebo.

Contrary findings were reported in a recent double-blind, randomized trial of 100 children aged 5 to 17 years, which found a hospitalization rate of 10% for the prednisolone group compared with 30% for the fluticasone propionate (2000μg) group using MDI and spacer ($p = 0.0001$).[45] However, these children had very severe asthma attacks, with a mean forced expiratory volume in 1 second (FEV_1) of 45% predicted on entry to the trial.

In a placebo-controlled trial of 60 children aged 3 to 12 years admitted to the ED with moderately severe asthma exacerbation (mean PEFR 54% of predicted), admission of budesonide 400μg by MDI and spacer at 30-min intervals for 3h led to a significantly quicker recovery than placebo.[43] The children in the budesonide group showed significantly greater improvement in respiratory distress score ($p < 0.01$), less use of systemic corticosteroid ther-

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apy ($p < 0.05$), lower likelihood of oxygen need for more than 2h ($p < 0.05$) and fewer admissions on reassessment at 4h ($p < 0.05$).

In all these studies,[39-44,46] the authors used at least 5-fold higher doses of inhaled corticosteroids than the maintenance dose. Simply doubling the dose of inhaled corticosteroids during acute exacerbation was found to be insufficient,[47] as the drug still fails to reach the small airways.[45] Indeed, the β_2-adrenoceptor agonist dose is usually increased by a factor of 19 during asthma exacerbation.

Recently, two uncontrolled studies dealt with the use of high-dose inhaled corticosteroids for the treatment of children with acute asthma at home.[48,49] The first study, performed in 60 children aged 1 to 14 years, found that a daily dose of budesonide 1600μg (for 3 to 5 days) induced a similar increase in lung function and a statistically significantly greater drop in pulmonary index score (2.6 versus 1.9; $p = 0.04$) compared with the combination of a daily dose of methylprednisolone 1 mg/kg and budesonide 800μg.[48] In the other study of 150 children aged 1 to 14 years, an initial daily dose of **((Author: budesonide?))** 800 to 1600μg (for 4 to 8 days) was able to control 94% of the total 1061 episodes of acute asthma.[49] In both studies, treatment with inhaled budesonide was preceded by inhaled terbutaline (2000μg daily).

Current evidence indicates that oral prednisolone, owing to its reliability, simplicity, convenience and low cost, should be the treatment of choice for the most severe asthma exacerbations (i.e., PEFR <45% of predicted and/or oxygen saturation <90%), when lung airways are extremely contracted and filled with secretion. High-dose inhaled corticosteroids, by nebulization[39-41,44] with MDI and spacer[43] or via Turbuhaler®/Turbohaler®,[42] may be considered as an alternative treatment to oral corticosteroids in cases of acute moderate to severe asthma exacerbations, especially in children familiar with these modes of inhalation.[48,49] The most suitable place for administration of short-term, high-dose inhaled corticosteroids for acute asthma exacerbations is at home,[48-50] before reaching the ED.

4. Other Treatments

4.1 Ipratropium Bromide

Anticholinergic agents such as ipratropium bromide have a slower onset of action and weaker bronchodilating effect than β_2-adrenoceptor agonists. However, they may have a place in the specific relief of cholinergic bronchomotor tone with mucosal edema and secretions.[51-53] A single dose of an anticholinergic agent is not effective for the treatment of severe asthma exacerbations,[54] but multiple doses combined with β_2-adrenoceptor agonists may improve lung function and avoid hospital admission in a small percentage (1 of 12%) of school-aged children. This regimen has also been attempted for the treatment of infants with airway obstruction with associated wheeze, but findings are controversial.

In a recent review of six trials[55] (321 infants), including two studies undertaken in the ED[56,57] (130 infants), no difference was found between treatment with β_2-adrenoceptor agonists alone or in combination with ipratropium bromide in improving respiratory rate or oxygen saturation in the ED, or decreasing hospital stay. Although the combined regimen reduced the need for additional treatment, the reviewers concluded that there is not enough evidence to support the uncritical use of anticholinergic therapy for wheezing infants.[51] By contrast, Calvo et al.[58] found the combination of albuterol and ipratropium bromide significantly more effective in the treatment of acute asthma exacerbations than each medication alone with regard to both clinical scores and PEFR. Osmond and Klassen[59] reported that the addition of ipratropium bromide bromide to β_2-adrenoceptor agonists yielded a significant improvement in percent-predicted FEV_1 but not in clinical symptoms. Several other studies reported no benefit at all of adjunctive ipratropium bromide.[52,55,60] For example, in a randomized, blinded, controlled trial of 298 children aged 3 to 17 years, Ducharme and Davis[60] noted that the addition of ipratropium bromide (two puffs every 6 to 8h by spacer or 125 to 500μg every 6 to 8h by nebulization) to frequent doses of β_2-adrenoceptor agonists failed to improve bronchodilation compared with β_2-adrenoceptor agonists alone.

Based on current evidence, combination therapy with ipratropium bromide and β_2-adrenoceptor agonists should be considered only in patients who do not respond to inhaled β_2-adrenoceptor agonists.

4.2 Theophylline

Theophylline has been used traditionally for acute bronchodilation, but it provides less benefit than inhaled β_2-adrenoceptor agonists and adds little when combined with β_2-adrenoceptor agonists and corticosteroid therapy.[61] It is also associated with adverse effects and the risk of toxicity.[62] Furthermore, a meta-analysis of aminophylline (another phosphodiesterase inhibitor) performed a decade ago indicated no benefit in acute asthma **((Author: please confirm that rewording is ok))**.[61]

4.3 Magnesium Sulfate

Hypomagnesemia causes contraction of the smooth muscle cells, and hypermagnesemia causes their relaxation. There is

some evidence that magnesium infusion can provide additional bronchodilation in asthmatic patients.[63,64]

Magnesium sulfate appears to be beneficial and has a good safety profile in patients who present with severe acute asthma **((Author: please confirm that rewording is ok))**,[64]but it is not in routine use in the ED. Pooling of studies yielded a statistically significant heterogeneity: improvement in lung function tests was more pronounced in patients with severe asthma than those with mild to moderate asthma.[65]

5. Monitoring the Child with Acute Asthma Attack

The increase in hospital admissions of children with acute asthma attacks in the last 2 decades cannot be explained by an increase in morbidity alone. Morgan et al.[66] claimed that it is apparently influenced by the extent to which hospital services are organized and provided. Improved assessment strategies and immediate management in the ED can reduce hospital admission rates. Connet**((Author: spelling differs from that in ref. list; please resolve))** et al.[67] compared rates of discharge from the ED between a registrar who based the decision to discharge on the children's response to nebulized albuterol and the ability of their parents to continue treatment at home, and a senior officer of the same hospital. The registrar was able to send 53 of 158 children (33.5%) home, and the senior house officer only 6 of 39 children (15.4%).

Another reason for the increase in overall hospital admissions for asthma is the high readmission rate, which can reach 25% or more within **((Author: 1?))** year.[68] The factors associated with asthma relapse are a history of numerous ED visits over the previous year, history of urgent clinic visits over the previous year, use of a home nebulizer, and multiple asthma triggers.[4] Patient age, as well as use of bronchodilators and exposure to cigarette smoke, may also play a role.[3] Most of these factors are preventable with appropriate patient education. In the study by Connet**((Author: spelling?))** et al.,[67] the children were discharged after oral instruction on the use of a spacer device and receipt of written instructions regarding treatment and follow-up. The introduction of a special training program for senior house officers in the treatment of an acute asthma attack in the ED and the manner of instructing parents and children in home management resulted in a significant increase in the number of children sent home during the intervention year (299 of 687, 43.5%) compared with the previous year (12.3%). The clinical scores of the children sent home were no worse than those for the hospitalized children. **((Author: ref. 69 has not been cited; should it be referred to in this paragraph?))**

Comprehensive, nurse-led patient education programs administered during hospitalization of children[69,70] for acute asthma attack or at discharge[71] significantly reduce subsequent admission to hospital for asthma.[69,70] They were also found to be cost-effective, saving $**((Author: US?))**87 000 by a 79% decrease in ED readmissions and an 86% decrease in hospitalizations.[71]

Health education interventions in the community can help as well.[72] A group of 310 asthmatic children from low-income urban areas with one or more hospitalizations during the previous year attended an education program to improve asthma management at home. A significant decrease was noted in their use of emergency services compared with that of a randomized control group ($p > 0.05$).**((Author: do you mean p <0.05?))** The program saved $**((Author: US?))**11.22 for every $**((Author: US?))**1.00 spent on the program.[72]

6. Conclusions

This manuscript reviews the most recent data on ED management of moderate and severe asthma exacerbation in children.

Oxygen supplementation to maintain oxygen saturation above 92% remains the first and most important means of treatment for severe exacerbations.[8]

β_2-Adrenoceptor agonist administration by MDI with a holding chamber is as effective as nebulization and may be preferable to nebulization, being easier to use, less costly, and associated with fewer adverse effects.[19,20,22] When an asthma attack occurs, a combination of β_2-adrenoceptor agonists and inhaled corticosteroids should be used.

Inhaled corticosteroids are delivered directly into the lung, providing them with greater anti-inflammatory and antiasthmatic potency and fewer systemic effects than oral corticosteroids,[73,74] which makes them excellent candidate agents for controlling acute moderate to severe asthma exacerbations.[27,33,42,43,45] Most recent studies[27,39,42,43,45] except one (in patients with very severe attacks)[45] found inhaled corticosteroids to be at least as effective as oral corticosteroids in controlling acute asthma attacks in children in the ED. They can be administered by nebulization,[39-41,44] with MDI via spacer[43] or by Turbuhaler®/Turbohaler®.[42] For very severe asthma exacerbation (PEFR <45% of predicted and/or oxygen saturation <90%), in which airways are too narrow to benefit from the inhaled drug, oral corticosteroids are the treatment of choice. They are reliable, convenient and easy to use. Inhaled corticosteroids may also be used at home,[49] though further randomized, double-blind, placebo-controlled studies are still needed to clarify this issue.

To ensure compliance, and proper use of inhalers and spacers, comprehensive patient and parental education should accompany treatment in the ED with practice using inhalers/spacers. Powell et al.[72] successfully introduced the use of MDI and spacers instead of nebulization in their ED in Australia. They attributed the success of the program to good planning and use of a proven structured strategy.

Health education interventions have been found to be effective in reducing readmissions to the ED and hospitalizations.[75] Especially cost effective are nurse-led programs conducted at discharge from ED and geared to the whole family.[69,70]

Since prevention is still the best medicine, comprehensive asthma management should always include asthma education, measures of prevention against asthma triggers, appropriate anti-asthma drugs, and training in the correct use of inhalers and spacers. Good management avoids most severe asthma attacks.

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