Facilitators Guide

Author **Miriam Saey Al-Rifai**
(Edits by the DFTB Team)
fellows@dontforgetthebubbles.com

**Duration** | Up to 2 hrs
---|---
**Facilitator level** | Senior Trainee/ANP and above
**Learner level** | Junior trainee/Staff nurse and Senior trainee/ ANP
**Equipment required:** | None
OUTLINE

- Basics (10 mins)
- Main session: (2 x 15 minute) case discussions covering the key points and evidence
- Advanced session: (2 x 20 minutes) case discussions covering grey areas, diagnostic dilemmas; advanced management and escalation
- Sim scenario (30-60 mins)
- Quiz (10 mins)
- Infographic sharing (5 mins): 5 take home learning points

We also recommend printing/sharing a copy of your local guideline for sharing admission criteria.

PRE-READING FOR LEARNERS

Expectation is for the learners to have watched or read one of the basic pathophysiology links before the session.

Khan Academy
Openpediatrics.org, Video
Asthma Podcast
DFTB Asthma
PATHOPHYSIOLOGY (FROM OPENPAEDIATRICS/LITFL)

Asthma is a chronic inflammatory condition of the airways demonstrated by reversible airway obstruction.

It is manifested as a triad of:

**Bronchoconstriction** - Smooth muscle hypertrophy and hyperplasia, innervated by parasympathetic muscarinic M3 receptors.

**Hypersecretion** - Goblet cell and mucous gland hyperplasia with mucous hypersecretion.

**Inflammation** - Inflammatory cell infiltration and oedema in the submucosa, including eosinophils, activated helper T-cells, mast cells, and sometimes neutrophils.

**Risk Factors (patient.co.uk)**

Personal or family history of atopy, living in an urban environment, socio-economic deprivation, obesity, prematurity and low birth weight, viral infections in early childhood, smoking, maternal smoking, early exposure to broad spectrum antibiotics.

**Presentation**

Cough, wheeze, breathlessness, chest tightness.

**Diagnoses**

A diagnosis of asthma should be suspected in a person whose symptoms are worse at night and in the early morning, triggered by exercise, allergen exposure, cold air, after taking non-steroidal anti-inflammatory drugs, or beta-blockers and occur in the absence of an upper respiratory tract infection.

In addition they are likely to have a history of atopic disorder and present with widespread wheeze (bilateral, predominantly expiratory).
LEARNING POINTS - CASES

● Establishing the severity score of an acute asthma presentation
● Key features of asthma management
● How to plan for a safe discharge
● General approach to paediatric wheeze

CASE 1 (15 MINS)

Joseph, a 10yr old boy comes into the ED. He is a known asthmatic on treatment. He appears breathless with an audible wheeze. He is able to talk in complete sentences. He has a RR of 25, sats of 94%, pulse of 100 and his PEF is 60% of normal.

What is the severity score of this child’s asthma presentation?
What investigations and treatment options should you consider?
How do you decide when it is safe to discharge home?
The boy is 3 years old with the same presentation – his mum asks you if her son has asthma. What is your response?

Discussion points:
When seeing a child with an acute asthma attack, the initial assessment is key to establishing the severity of the attack as this influences ongoing management.

SIGN and BTS guideline
The following clinical signs should be recorded:

**Pulse rate** - Increasing tachycardia generally denotes worsening asthma; severe airway obstruction can result in pulsus paradoxus and a fall in heart rate in life-threatening asthma is a preterminal event. **Respiratory rate and degree of breathlessness** - Le too breathless to complete sentences in one breath or to feed. **Use of accessory muscles of respiration** - subcostal, intercostal recessions, tracheal tug. You can also assess by palpation of neck muscles. Also consider including a prolonged expiratory phase. **Amount of wheezing** - which might become biphasic or less apparent with increasing airways obstruction. Silent chest is an indicator of life threatening asthma. It is important to auscultate and document any improvement with treatment. **Degree of agitation and conscious level** - always give calm reassurance.

**Initial Investigations**

**Observations**
- Include general observations

**CXR**
- NOT routinely advised. A chest X-ray should be performed if there are persisting unilateral signs suggesting pneumothorax, inhaled foreign body, lobar collapse or consolidation and/or life-threatening asthma not responding to treatment.

**Blood Gas**
- Only indicated if not responding to treatment or needing further escalation of care

**Initial Management**

**Oxygen**
- If any life threatening features or sats <94%. Aim for sats 94-98%

**Inhaled β2 agonist**
- Salbutamol up to 10 puffs via spacer (1 puff = 5 breaths) assess after 15 mins and repeat if necessary. If sats <94% use, or patient refusal/poor inhaler technique use salbutamol nebulisers (2.5 - 5mg).
- Continuous nebulisation may be better, as intermittent may result in rebound bronchoconstriction. Be aware of ventolin toxicity.

**Ipratropium Bromide**
- If symptoms are refractory to initial β2 agonist treatment, add ipratropium bromide (250 micrograms/dose mixed with the nebulised β2 agonist solution) every 20-30 mins for the first two hours in severe asthma attacks. This should then be tapered to 4–6hrly before being discontinued. However, there are no clinical trials supporting ipratropium use beyond the first hour or first 3 doses in children (EMCases).
- In a systematic review and meta-analysis comparing the use of beta-agonists plus anticholinergics with beta-agonists alone, combination therapy was associated with significantly lower hospitalisation rates and improvements in asthma scores and pulmonary function test results (EMCases).
Oral steroids

- Give oral steroids in the management of acute asthma attacks.

Dexamethasone is starting to be used more, as a once only dose, but there is no evidence for benefit over Prednisolone, so not recommended yet.

Nebulised Magnesium

- Nebulised magnesium sulphate is not recommended for children with mild to moderate asthma attacks.

The RCT entitled MAGNETIC trial in 2013 of about 500 children showed that MgSO4 nebulisers added to the salbutamol and ipratropium bromide nebuliser in the first hour, for kids with acute severe asthma, significantly improved asthma severity scores without any increase in adverse events.

Antibiotics

- Insufficient evidence to refute or recommend.

Burst therapy

Salbutamol 100 mcgs x 10 puffs via inhaler & spacer every 20 mins for 1 hour. Add ipratropium bromide 20 mcgs (x 4 puffs < 5 years, x 8 puffs > 5 years) together with salbutamol as above for severe cases.

Video on how to use a spacer

When is it safe to discharge home

BTS/SIGN - Children can be discharged home once requiring no more than 3-4 hourly inhalers (based on a randomised controlled study in 1999), PEF >75% and sats >94%.

Safe follow up

- Reducing regime of salbutamol inhaler therapy with a clear plan as to when to come back to hospital (ie. requiring >10 puffs in 4 hours)

- Ensure good inhaler technique/ correct fitting spacer mask. Advise to use the B-agonist BEFORE the inhaled steroid and to wash the mouth out after the steroid inhaler to prevent thrush.

- If the parent/carer of the child smokes, advise them to stop.

- Address potentially preventable contributors to the exacerbation, such as exposure to trigger factors

- Ensure the patient is discharged home with 3-5 days oral steroids. Some trusts are now given single dose Dexamethasone, although prednisolone is still in the national guideline (https://www.stemlynsblog.org/dexamethasone-asthma-children/).

- Primary care follow-up in 24-48hrs

- If 2nd attack in 12 months refer to a secondary care asthma clinic.

The boy is 3 years old with the same presentation – his mum asks you if her son has asthma.

What is your response?

Wheeze is a common presentation in the ED and its diagnoses and management differs depending on the age of the child and the detail in the history (Snelson et al, 2019).
Bronchiolitis
● Slow onset of symptoms. 3-4 day period of worsening cough, poor feeding, wheeze and respiratory distress due to inflammation of the airways.

Viral Wheeze
● Rapid onset of wheeze and respiratory distress over hours due to bronchospasm.

Asthma
● Described above.

The age based approach to wheeze can be explained by the changes in a child's immune system:
● At birth and in the first few months, immunity is largely provided by maternal antibodies. These antibodies offer protection from most simple viral infections. Acute atopic IgE mediated reactions are very rare. If infections do occur it is likely to be serious bacterial infections. In addition the baby's own immune system is not yet fully turned on and the response to infection is therefore muted, making the recognition of sepsis difficult in this age group.

● Preschool age children no longer rely on maternal antibodies. However, their own immune system is still not fully developed. They compensate for this by having heightened and indiscriminate responses to infections. They produce lots of white blood cells, but do not yet have circulating antibodies. You are more likely to see associated problems of viral infections in this age group like transient synovitis. Atopy is becoming more common now. Sepsis is also difficult to recognize in this age group due to the extreme reaction to often uncomplicated viral infections. These children present with viral wheeze. It is worth knowing that there are wheezy presentations in this age group that can look a lot like viral wheeze. These include bronchomalacia, acute allergy, and cardiac failure due to e.g. acute myocarditis.

● Older children have a more mature immune system and response to infection is like that of an adult. As the response to infection is less vigorous and indiscriminate than the pre-schoolers, some specific infections like Varicella can cause severe reactions in these children. These children are more likely to
have asthma. True asthma is rarely seen under the age of 5 as it requires a fully matured immune system to develop.

Management

By looking at the history we can direct our inhaled beta agonist treatment to one that matches a story consistent with bronchospasm. This would include children with likely viral wheeze and asthma.

The best evidence for the use of oral steroids for viral wheeze between the ages of 1 and 5 would suggest that the following group are most likely to have a small benefit (http://gppaedstips.blogspot.com/search/label/Asthma):

- Children with a diagnosis of asthma
- Children who have required substantial amounts of inhaled beta-agonist prior to presentation
- Children whose severity and lack of response to treatment with beta-agonists requires admission to hospital

Case 1 Summary

- Joseph has presented with features of a moderate asthma attack
- This can be managed with beta 2 agonist therapy and oral steroids
- Once Joseph does not need beta 2 agonist bronchodilation for more than 4hrs and obs remain stable he can be safely discharged home with safe follow up.
- The 3 year old presenting with the same symptoms of sudden onset wheeze and breathlessness, likely has a diagnosis of viral wheeze. They would benefit from beta 2 agonist bronchodilation. They are too young to be diagnosed with asthma, but risk factors for developing asthma could be explored in the history.

CASE 2 (15 MINS)

Leila, a 13yr old female, known asthmatic on treatment, presented to ED breathless and finding it hard to speak in full sentences. Her oxygen saturations are 92%, HR 130 and RR35.

1-What is the severity score of this child's asthma presentation?
2-What investigations and treatment options should you consider?
3-When do you need to re-assess response to treatment to decide on discharge vs escalation?

Case Summary

- In this instance, Leila falls into the acute severe asthma presentation.
- As her sats <94% oxygen needs to be given via a facemask.
• Beta 2 agonist bronchodilator via a nebuliser (preferably oxygen driven) to be given due to sats <94% and she falls into the severe category.
• Oral steroids must be given. This can be given as IV Dexamethasone if too unwell/vomiting.
• Re-assess response to treatment after 15 minutes.
• If no improvement after 15 minutes give a further two beta 2 agonist nebulisers and add Ipratropium Bromide nebuliser.
• A consideration of nebulised Mg can be given in acute severe asthma.
• Plan for admission, escalate and refer to paediatrics for consideration of second line treatment.

LEARNING POINTS - ADVANCED CASES

• How to Manage life threatening asthma
• IV bronchodilators
• The role of heliox, ketamine and NIV
• Cardio-respiratory arrest in asthma and modifications to the ALS algorithm

ADVANCED DISCUSSION (20 MINS PER CASE)

This is an opportunity to cover grey areas, diagnostic dilemmas and advanced management and escalation if there are more experienced trainees or senior registrars in your group.

ADVANCED CASE 1

Leila has not improved despite the treatment given in ED as outlined in case 2. Her sats are now 89%, she appears cyanosed and has a poor respiratory effort. On auscultation her chest is quiet. What are the next steps that need to be taken.

How would you rate the severity score of this presentation?
What investigations or treatment needs to be considered?
Which IV medications if any should be used?
Which important differentials need to be considered?
What escalation plans need to be put in place?

Children with continuing severe asthma despite optimal first-line treatments, frequent nebulised β2 agonists and ipratropium bromide plus oral steroids, and those with life-threatening features, need urgent review by a specialist with a view to management in an appropriate high-dependency area or transfer to a paediatric intensive care unit to receive second-line intravenous therapies.
It is important to do a blood gas prior to starting bronchodilators to measure the pCO2 and also to monitor side effects of IV salbutamol (decreasing potassium and lactic acidosis).

**IV bronchodilators**

PERUKI (Paediatric Emergency Research in the UK and Ireland network) is a research collaborative of paediatric-specific and mixed adult and paediatric emergency departments (EDs). In 2015 PERUKI carried out a study looking at the variation in practise of the use of IV bronchodilators as a second line treatment in the management of acute asthma in children. There was a large discrepancy between what clinicians felt was the appropriate management and what they actually administered. A survey of 183 clinicians in 30 EDs revealed that when escalating to intravenous bronchodilators, 99 (54%) preferred salbutamol first line, 52 (28%) magnesium sulfate (MgSO4) and 27 (15%) aminophylline. 87 (48%) administered intravenous bronchodilators sequentially and 30 (16%) concurrently, with others basing approach on case severity. 146 (80%) continued inhaled therapy after commencing intravenous bronchodilators.

Of 170 who used intravenous salbutamol, 146 (86%) gave rapid boluses, 21 (12%) a longer loading dose and 164 (97%) an ongoing infusion, each with a range of doses and durations. Of 173 who used intravenous MgSO4, all used a bolus only. What this demonstrates is the considerable variability in practise and opinion. So what is the evidence? (Cochrane review)

**IV salbutamol**
- There have not been enough trials to form a robust evaluation of its benefits.

**IV MgSO4**
- Appears to be safe and beneficial in severe asthma

**IV Aminophylline**
- Improves lung function within 6hrs. However, there is no apparent reduction in symptoms, number of nebulised treatments or length of hospital stay. We do not know the impact on oxygenation, PICU admissions or need for NIV.

**IV Ketamine**
- There has only been 1 study conducted, which reveals no known benefit in non intubated children.

In one RCT comparing IV aminophylline, salbutamol and magnesium in 100 children, a bolus of magnesium sulphate was shown to reduce clinical symptoms faster than the
other treatments. There were no significant side effects documented in the magnesium sulphate group. A systematic review of four paediatric trials comparing IV salbutamol with IV aminophylline demonstrated equivalence.

**BTS/SIGN guidance for IV bronchodilators**

In children who respond poorly to first-line treatments, consider the addition of intravenous magnesium sulphate as first-line intravenous treatment (40 mg/kg/day).

Consider early addition of a single bolus dose of intravenous salbutamol (15 micrograms/kg over 10 minutes) in a severe asthma attack where the child has not responded to initial inhaled therapy. It is not clear whether IV bolus vs infusion is more beneficial. Prior to IV salbutamol administration insure blood potassium is checked and on cardiac monitor. If using an IV infusion monitor lactate to check for toxicity.

Consider aminophylline for children with severe or life-threatening asthma unresponsive to maximal doses of bronchodilators and steroids. Some of the side effects include abdominal pain, anxiety, headache, nausea, palpitations and seizures. Toxicity can occur with aminophylline. This presents as vomiting (which may be severe and intractable), agitation, restlessness, dilated pupils, sinus tachycardia, and hyperglycaemia. More serious effects are haematemesis, convulsions, and supraventricular and ventricular arrhythmias. Severe hypokalaemia may develop rapidly especially in combination with salbutamol.

**What are your next steps?**

Ensuring you have appropriately assessed and optimised their condition

**Reassess the patient?**
- Consider revisiting history, respiratory examination and consider adjuncts to assessment such as a capillary or venous blood gas.

**Have you exhausted medical management?**
- ? adrenaline ? ketamine ? heliox

**What could be missing?**
- Consider your confidence of whether you have the right diagnosis or if there is a need to assess for a secondary pathology such as pneumonia, foreign body, anatomic airway anomalies, airway compression by masses/lymph nodes, cardiac disease? Some can be excluded with a good history.

Do you need to further investigate with bloods, CXR? Do you
need to append your management and provide antibiotic coverage? Do you need to assess for a complication from treatment e.g. pneumothorax.

**Escalation options**
- Have you sought a senior review/notified the admitting paediatrician?
- Do you need an ICU consult, NETS consult or retrieval to a tertiary centre?
- How long are you comfortable to wait to see if there is a response to IV bronchodilation?

**Non invasive ventilation**
- Is there any evidence in acute asthma attack?
- What settings/mode would you use?

Does this child need to be intubated?
- How would you determine this?
- Who should be involved in the conversation? Who should perform the intubation?
- What sedation would you use?
- What equipment would you use?
- What settings would you use?

**Adrenaline**
Give for severe or life threatening asthma - if the diagnosis is in doubt. Asthma and atopy often co-exist - and in these patients death from anaphylaxis is more likely. So if a patient fails to respond to initial therapy, the diagnosis of anaphylaxis needs to be considered. In addition nebulised Adrenaline causes bronchodilation.

**Heliox**
May improve respiratory score, but it probably won’t reduce the risk of admission. Nor should you use it in routine asthma to stave off intubation (PEMBlog). Can be considered in the ICU setting with maximum oxygen therapy has failed.

**Ketamine**
Limited number of trials with mixed outcomes on the benefits of Ketamine. However, it is safe at dissociative dosages, and is a reasonable option when all others measures have failed.

**NIV**
A few case reports and observational studies of the use of BiPAP in pediatric asthma show some promise. The one RCT of only 20 patients does show a benefit in clinical asthma scores, respiratory rate, and supplemental oxygen need. There is no evidence that it prevents the need for intubation (Basnet S et al, 2012).

Critical care input is the next step for children with severe asthma not responding to treatment or with any life threatening features. There are a number of ongoing trials on the use of ketamine, sevofluorane and NIV, but the evidence is currently lacking so they’re not recommended by BTS/SIGN.
ADVANCED CASE 2

A 15yr old male has been brought into resus with features of life threatening asthma. Pre hospital the paramedics gave continuous salbutamol nebulisers, 500mcg Ipratropium nebulisers and 0.5mg IM Adrenaline. The attending medical team in resus administered 2g IV Mg over 20 mins and a bolus of 250mcg IV Salbutamol. The patient then became unresponsive with no respiratory effort.

**What are the next steps that need to be taken?**

**What is the ‘deadly triad’ in asthma?**

**What are the key ALS modifications in asthma arrest?**

In the pre-hospital setting, paramedics usually give IM Adrenaline to cover for the possibility of a diagnosis of anaphylaxis.

In this case the patient has arrested. As soon as this has been identified, CPR needs to be initiated as per the ALS guidelines.

The cause of cardiac arrest in asthma is a result of the ‘deadly triad’:

**Arrest in Asthma**

**The Deadly Triad**

1. Respiratory exhaustion

2. Respiratory acidosis

3. Impaired venous return

@RCEMLearning
Important modifications and considerations in managing cardio respiratory arrest in asthma
RCEMLearning - www.rcemlearning.co.uk/foamed/arrest-asthma/

1. **Intubate Early**
   Due to the need for high inflation pressures, an endotracheal tube (ETT) is needed. In addition this protects the airway from the increased risk of regurgitation and aspiration.

2. **Ventilate with caution**
   The European Resuscitation Council recommends 8-10 breaths per minute with the lowest tidal volume required to see the rise and fall of the chest, to avoid dynamic hyperinflation. Tachypnoea must be avoided as this reduces expiratory time, thus increasing the residual volume in the alveoli. This auto PEEP increases intrathoracic pressure which reduces venous return, impeding CPR.

3. **Manual chest deflation**
   If the patient has a hyperinflated chest/poor excursions of the chest wall, disconnect the ETT and apply manual pressure to the patient’s chest to expel the trapped air.

4. **Consider tension pneumothorax**
   If ETT disconnection does not improve ventilation, consider performing a bilateral thoracostomy.

5. **Rehydrate**
   Dehydration and reduced intravascular volume compromises effective CPR. It also causes mucus to be thicker which can plug small airways. So ensure you give IV fluids.

6. **GIVE ADRENALINE!** - Utilise its bronchodilator effect.
In an acute asthma exacerbation in children, monitoring the oxygen sats is important because:

A. Hypoxaemia is an early sign of clinical deterioration
B. Sats <95% may suggest the need for prolonged bronchodilator therapy
C. Hypoxaemia occurs in the presence of life threatening asthma. Children may have normal sats for some time before critical desaturation occurs.
D. Sats >96% supports the decision to safely discharge home

Explanation:
- In an acute asthma attack hypoxic vasoconstriction occurs. This is coupled with decreased blood flow to the under ventilated lung (matching pulmonary perfusion with alveolar ventilation).
- In the hospital setting SaO2<91% may be a helpful predictor of prolonged frequent bronchodilator therapy more than 4 hours and SaO2 of <89% is associated with a need for bronchodilator therapy over 12 hours.
- Hypoxaemia and hypocarbia only occur in the presence of life threatening asthma. Children may have normal sats for some time before critical desaturation occurs. Whilst low oxygen saturations mean that a patient is unwell it should be clinically obvious at this point. Low oxygen saturations may also represent a degree of mucus plugging that may be helped with repositioning.
- Hyperoxia can lead to absorption atelectasis as well as intra-pulmonary shunting with subsequent reduction in cardiac output. In addition concerns have been raised that oxygen administration may lead to potential delay in recognising clinical deterioration.
Question 2.

What is an appropriate length of time to stretch children in the ED prior to discharge

A. After two sets of 3-4hrly inhaler/nebulisers
B. After they reach the first 3-4hrs post last inhaler/nebuliser
C. After two sets of 3hrly
D. After 1 hour, if obs are completely normal and has had a consultant review

BUT this is based on a randomised control trial in 1999. The most recent study in 2018 suggests that there is no benefit to 4hours vs 3hrs, and infact 3hrs post inhalation resulted in a reduction in length of stay. A recent retrospective analysis study in Australia looked at discharging children after 1 hour. They suggested that children that were clinically ‘well’ at 1 hour were likely to go home and if they were showing any moderate symptoms at one hour would likely need to be admitted. There is no strong evidence or recent studies, which is why there is such variation in practice.

Question 3.

Under what circumstances would you choose to administer a beta agonist via nebuliser as opposed to a pMDI with a spacer?

A. When the child has become more tachycardic with worsening salbutamol induced tremor
B. In severe or life threatening asthma or when under the age of 1yrs old/learning difficulties
C. If the pMDI is ineffective
D. Some departments prefer nebulisers as it is cheaper than inhaled preparations

Cochrane review 2013 - “Metered-dose inhalers with a spacer can perform at least as well as nebulisation in delivering beta-agonists in children with acute asthma”

Salbutamol has systemic side effects – tremor and increased pulse rate were more common when using nebulisers. SIGN/BTS guidelines state to give nebulisers in severe or life threatening asthma. Nebulisers are also preferential in very young children, or those with learning difficulties, as coordinating breathing with an inhaler can be difficult. Cost savings can be made with inhaled preparations.
Question 4.

When is intubation indicated in paediatric asthma presentations?

A
When the HR > 160 OR the RR > 60

B
When you have given all first line and second line treatment and trialled NIPPV and the patient has still not improved.

C
The child looks exhausted with worsening hypercapnia and changes in mental status.

D
When the child has a history of fast deterioration and need for intubation.

Up to 26% of children intubated due to asthma suffer complications including pneumothorax, impaired venous return, and cardiovascular collapse because of increased intrathoracic pressure. Mechanical ventilation during an asthma exacerbation is associated with an increased risk of death and should therefore be a last resort. The decision to intubate should be based on clinical judgement as opposed to any one observation or blood result. Some variables to consider for intubation are worsening hypercapnia, patient exhaustion and changes in mental status (EMCases).

Question 5.

You have a 4yr old, with two days of wheeze, coryzal symptoms and one day of increased work of breathing symptoms. You suspect that this may be viral induced wheeze. How do you manage this child?

A
Burst therapy with salbutamol.

B
6-10 puffs of salbutamol and reassess. If severe symptoms give oral steroids.

C
Humidified air nebuliser and antipyretics for fever.

D
6-10 puffs of salbutamol and Ipratropium bromide nebuliser. If severe symptoms give oral steroids.

At what age would be appropriate to consider a trial of ventolin for potential viral induced wheeze?

- (Note - This is a good opportunity to survey your team and colleagues to see what the practice is at your local department).
- Regarding this grey area question, in Australian practice, some clinicians will trial
salbutamol for potential viral induced wheeze if the child is 12 months or older. Other doctors may wish to trial if the child is slightly younger (e.g. from 10 months) if they have a strong family history of asthma and atopy or if they have had previous ventolin use reported by their family with good effect. The younger the child is, the less likely that the story and case is to fit viral induced wheeze.

If you have decided to trial ventolin - would you give only an initial 10-6 puffs and reassess or would you give a burst (x puffs x 20 minutely x 3) ?

- It would be prudent to give 10-6 puffs (or do you use another number?) and reassess following to see if there is any improvement or change.

**Steroids in VIW**

If you are not sure if the child is presenting with asthma or viral induced wheeze, but they are displaying severe symptoms - it is advisable to give steroids. But be cautious in giving too many courses of steroids if presenting frequently to the ED.
Finish-infographic of the take home tips (5 minutes)

1. Age (immune system development) and clinical history are key to determining the cause of wheeze in children. <1yrs old is likely bronchiolitis, 1-5 is likely viral wheeze and >5yrs may indicate asthma.

2. The initial assessment is key to establishing the severity of the asthma attack which guides initial management.

3. Always reassess response to treatment after 15 mins.

4. Always give oral steroids.

5. Know criteria for discharge and ensure safe follow up is in place.

6. Ensure critical care referrals are made early for children with severe asthma not responding to treatment or with any life threatening features.

7. Consider modifications to ALS protocol in asthma cardiorespiratory arrest.

REFERENCES


DFTB - Managing acute asthma, Simon Craig (2017)
DFTB - Asthma for ambos (2016)
DFTB - Are nebulisers or spacers better for managing acute asthma (2013)
DFTB - The curious incident of the wheeze in the night
PEMBLOG - Heliox in the emergency department (2017)
EMCases - Management of acute paediatric exacerbations (2016)
BTS/SIGN British guideline on the management of asthma in children (2019)
PaediatricFOAMed - ventilation strategies for the critically ill asthmatic (2019)
RCEMLearning - Arrest in asthma
gppaedstips.blogspot.com - The NYCE guideline for viral induced wheeze - Let's clear a few things up (2019)

fellows@dontforgetthebubbles.com