# ASTHMA

## Asthma – Adult and Child

### Definition

*A reversible airway obstruction*

### Epidemiology

- 300 million people worldwide
- Childhood: M>F
- Post puberty: F>M

### Cause

**Atopy and allergy:**
- Individuals readily develop IgE.

**Combination:** Genetic predisposition and environmental influences.

**Other possibilities:**
- Hygiene hypothesis: ↓ infections in early life bias immune system towards allergic phenotype – Th2. T lymphocytes may differentiate into Th1 (fights virus/bacteria) or Th2 (allergic response).
- Warm, humid, centrally heated homes = multiplication of dust mites.
- Obesity – Asthma due to mechanical mechanisms e.g. GORD.

### Pathophysiology

<table>
<thead>
<tr>
<th>Environmental factors</th>
<th>Genetic predisposition</th>
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- Bronchial inflammation
- Bronchial hyperreactivity and trigger factors

- Oedema
  - Bronchoconstriction
  - ↑ Mucus production
  - Smooth muscle hypertrophy

- Airways narrowing and obstruction

**Symptoms:** Cough, Wheeze, Breathlessness, Tight chest

### Extrinsic or Intrinsic Asthma?

**Extrinsic:**
Sensitive to external allergens e.g. pollen, animal, dust – Begins in childhood – Often atopy.

**Intrinsic:**
Reaction to internal, nonallergenic factors e.g. post severe RTI, emotional stress, fatigue, temp changes – especially in adults.

*Note: You can have both!*
## Clinical Features

- Diurnal pattern (symptoms/PEF worse early in morning).
- Nocturnal asthma – cough and wheeze disturbing sleep.
- ‘Cough-variant asthma’ – cough dominant symptoms
- Medication related – β-blockers (may induce bronchospasm), aspirin (associated with rhinosinusitis, nasal polyps), other NSAIDS.
- Occupational asthma – Considered if working age and symptoms improve during time away from work - ↑
  - risk: atopy and smokers.

### Other symptoms/ presentations:
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- ‘Cough-variant asthma’ – cough dominant symptoms
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### Mild:
Asymptomatic between exacerbations which occur during viral RTI or after exposure to allergens.

### Persistent:
Pattern of chronic wheeze and breathlessness. Long standing obstruction causes *pectus carinatum* (pigeon chest) and/or Harrison’s sulcus.

## Investigations

### Diagnosis:
Clinical history with demonstration of variable airflow obstruction.

#### i) Pulmonary function tests
- Record PEFR after rising in morning and before retiring in evening.
- **Diagnostic:** >20% diurnal variation on ≥3 days in a week for 2 weeks on PEF diary – amount of variability is some indication of disease severity.
- **Spirometry:** Detects signs of obstructive airway disease – almost normal/ ↓VC, ↑total lung and residual capacities.
- **Trial corticosteroids** – useful to see improvement in PEFR. **Diagnostic:** FEV₁ ≥ 15% ↑ following administration of a bronchodilator/ trial of corticosteroids.

#### ii) Bronchial challenge test (AHR)
- Demonstrates airway hyper-reactivity – due to bronchoconstriction - ↑ concentrations of histamine/ methacholine causes a ↓ in FEV₁ if asthmatic.
- Note: Has a high –ve predictive value but +ve results may be seen in other conditions e.g COPD, CF.

#### iii) Exercise test
- For patients whose symptoms are related to exercise

### Questions to ask in children:
- a. How frequent are the symptoms?
- b. How is it affecting the child’s life?
- c. How much school has been missed in the last 6 months?
- d. Can he play sport normally?
- e. How often is sleep disturbed?
- f. What is the longest symptoms-free period?
- If asthmatic exercise should cause ↓ PEF/ FEV₁.
- **Diagnostic:** FEV₁ ≥ 15% ↓ after 6 mins of exercise.

**iv) Radiological**
- Generally unhelpful but may show alternative diagnosis.
- **Acute asthma signs:** Hyperinflation and ± lobar collapse.

**v) Measurement of allergic status**
- Skin-prick tests- Measurement of IgE to confirm sensitivity to specific agent.
- Atopic asthma: ↑ sputum or peripheral blood eosinophil count and ↑ serum total IgE.

**vi) ABGs**

**vii) Pulse oximetry**
- May show ↓ SaO₂ level.

**Occupational asthma** – Diagnosis difficult. Do two-hourly PEF recordings including time away from work. **Bronchial provocation tests** with suspected agent may be required.

### Management

<table>
<thead>
<tr>
<th>Step 1</th>
<th>Step 2</th>
<th>Step 3</th>
<th>Step 4</th>
<th>Step 5</th>
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<tbody>
<tr>
<td><strong>B₂ bronchodilator</strong></td>
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<tr>
<td>Low dose inhaled steroids</td>
<td>Moderate dose inhaled steroids</td>
<td>Long acting broncho-dilator ± leukotriene</td>
<td>High dose inhaled steroids</td>
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</tr>
<tr>
<td>Long acting broncho-dilator</td>
<td>± leukotriene</td>
<td>± theophyllines or ipatropium</td>
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<td>± theophyllines or ipatropium ± regular oral corticosteroids</td>
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**Goals:**
- Achieve and maintain control of symptoms.
- Prevent asthma exacerbations.
- Maintain pulmonary function as close to normal as pos.
- Avoid adverse effects from meds.
- Prevent development of irreversible airflow limitation.
- Prevent asthma mortality.

### Step-up Step-down approach to asthma treatment

Consider step down if symptom control good for 3+ months.

Only withdraw anti-inflammatory treatment if patient well for 6+ months.
### Management continued

<table>
<thead>
<tr>
<th>Patient education</th>
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<tbody>
<tr>
<td>- Inhaler technique.</td>
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<tr>
<td>- Medication – when to use, what each does, frequency, dosage.</td>
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<tr>
<td>- Use of PEF.</td>
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<tr>
<td>- Relationship between symptoms and inflammation and important key symptoms e.g. nocturnal waking</td>
</tr>
<tr>
<td>- What to do if acute asthma attack.</td>
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<tr>
<td>- Avoidance of aggravating factors.</td>
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<tr>
<td>- Do not smoke.</td>
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### Management of Acute Asthma

#### Clinical Features

<table>
<thead>
<tr>
<th>Moderate asthma exacerbations</th>
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<tr>
<td>- ↑ symptoms</td>
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<tr>
<td>- PEFR 50-75% of predicted or best</td>
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<tr>
<td>- Speech normal</td>
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<tr>
<td>- RR&gt;30bpm</td>
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<tr>
<td>- Pulse&gt;120bpm</td>
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</table>

**Treat at home or surgery**

<table>
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<tr>
<th>Severe asthma attack</th>
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<tr>
<td>- Can’t complete sentences/ too breathless to speak or feed</td>
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<tr>
<td>- Pulse: Adults ≥110bpm. Children ≥120bpm. Under5’s ≥130bpm.</td>
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<tr>
<td>- PEFR 33-50% predicted or best.</td>
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**Normally hospital admission**

<table>
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<tr>
<th>Life-threatening asthma attack</th>
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<tr>
<td>- PEFR ≤33% of predicted or best</td>
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<tr>
<td>- SpO₂ &lt; 92%</td>
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<tr>
<td>- PaO₂ &lt; 8kPa (normal PaCO₂)</td>
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<tr>
<td>- Silent chest</td>
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<tr>
<td>- Cyanosis</td>
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<tr>
<td>- Fatigue or exhaustion</td>
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<tr>
<td>- Bradycardia</td>
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<tr>
<td>- Hypotension</td>
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<tr>
<td>- Confusion</td>
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<td>- Coma</td>
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**Immediate hospital admission**

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<th>Near fatal asthma attack</th>
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<td>- ↑ PaCO₂ and/ or requiring mechanical ventilation with raised inflation pressure.</td>
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#### Initial assessment

- Ability to speak
- Pulse
- RR
- BP
- SaO₂
- PEF
- ABG
- Chest xray (if pneumothorax suspected)

#### Good signs to look for in children:

- Contraction of sternomastoids
- Chest recession
- Pulse rate

#### Management

| a. Oxygen – High flow – 40-60%. |
| b. Nebulised salbutamol (+ ipatropium bromide if acute severe) |
of life-threatening attack).

c. Systemic corticosteroids – Oral prednisolone or iv hydrocortisone (if unable to swallow).

d. IV fluids – Also K⁺ supplements may be necessary as repeat salbutamol ↓ serum K⁺.

e. Aminophylline or iv magnesium if 1. Life threatening attack or 2. Poor response to Nebulised bronchodilators.

<table>
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<th>Indications for assisted ventilation in acute severe asthma:</th>
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<tbody>
<tr>
<td>- Coma.</td>
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<td>- Respiratory arrest.</td>
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<td>- Deterioration of ABG (↓ PaO₂ ± ↑ PaCO₂ ± ph ↓).</td>
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<tr>
<td>- Exhaustion, confusion, drowsiness.</td>
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**Discharge**
- Should be stable on discharge meds (nebulised therapy discontinued for 24 hours).
- PEF 75% of predicted or best.
- Appointment with GP/asthma nurse in 2 working days and follow-up at specialist hospital clinic in a month.

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### Atopic Disorders

**Asthma**

**Eczema**

**Allergic rhinitis** – Recurrent or persistent obstruction of the nostrils with sniffing, sneezing and nasal discharge.

**Allergic conjunctivitis** – Redness and swelling of the eyes.

**Urticaria and angioedema**

**Food and drug allergies**

**Investigations:**
- +ve skin test to common allergens, eosinophilia and ↑ serum level of IgE.

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**Important Note**

These notes were written by Harriet Wood, as a medical student in 2009. They are presented in good faith and every effort has been taken to ensure their accuracy. Nevertheless, medical practice changes over time and it is always important to check the information with your clinical teachers and with other reliable sources. Disclaimer: no responsibility can be taken by either the author or publisher for any loss, damage or injury occasioned to any person acting or refraining from action as a result of this information. Please report any inaccuracies to: support@askdoctorclarke.com