ASTHMA

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OUTLINE

- Definition
- Pathology
- Epidemiology
- Aetiology
- Precipitating factors

- Mediators
- History
- Examination
- Investigations
- Management



Plinius the Younger A.D. 76

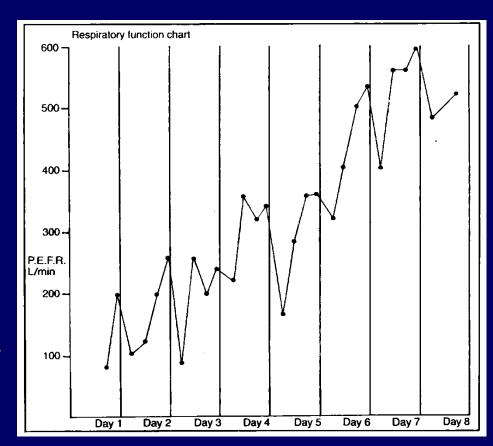
"Leaning on two servants, he brought himself upright and immediately collapsed again, I suppose because he was affected by the dense fog that obstructed his airways that were weak in nature, narrow and subject to inflammation"

DEFINITION (1/3) - CLINICAL

Clinical

partial airflow limitation

 Varies over short periods of time and with treatment



Peak flow chart with treatment

DEFINITION (3/3) - PATHOLOGY

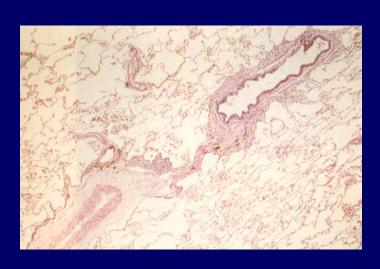
Site: smaller bronchi (& bronchiole)

Findings:

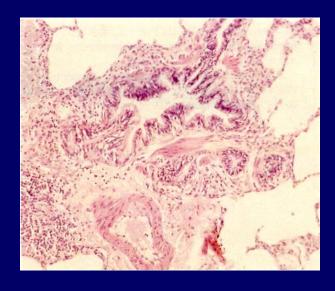
- smooth muscle hypertrophy
- submucous gland hypertrophy
- cellular infiltration (lympho + eosin's)

Asthma = eosinophilic bronchitis

PATHOPHYSIOLOGY OF ASTHMA



Normal airway



Asthmatic airway

- 1) Smooth muscle constriction
 - 2) Mucosal oedema
- 3) Chronic inflammatory cell infiltrate

KEY POINT

Asthma is an inflammatory condition, not just bronchoconstriction as a result of smooth muscle contraction

EPIDEMIOLOGY

- Common, prevalence 5%
- Peak incidence in second decade (10-15%)
- (Probably) increasing, especially in developing counties
 - ? pollution
 - -? dietary
 - ? improved standards of living

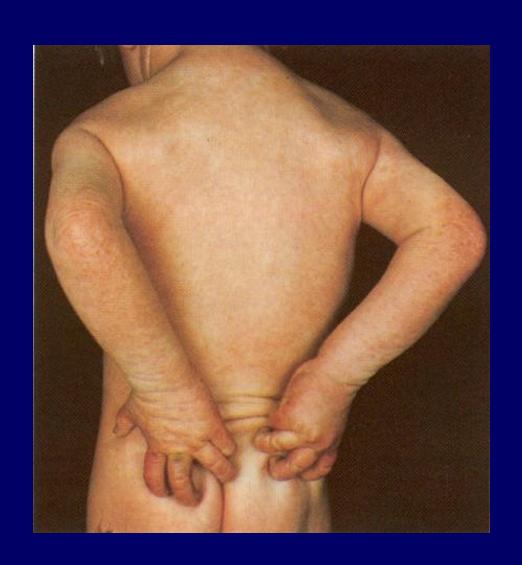
AETIOLOGY

2 major factors:

1) Atopy

 Type 1 IgE mediated inflammatory response to commonly encountered antigens, runs in families

CHILDHOOD ECZEMA



AETIOLOGY 2/2

- 2) Airway hyper-reactivity
- Early phase within 1 hr of exposure, smooth muscle contraction: use bronchodilators
- Late phase more prolonged, lasts 12 –24 hrs, inflammatory mucosal oedema: use corticosteroids
 - Bronchial provocation tests with methacholine

PRECIPITANTS

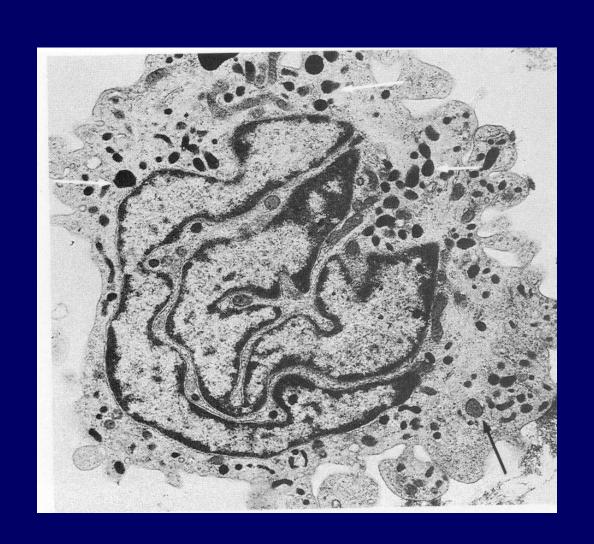
- Exercise / cold air
- Resp tract infection
- Inhaled allergens
 - Pollens
 - House dust mite
 - Pets

- Drugs
 - Beta-blockers
 - NSAID's
 - Histamine releasers
- Foods
- Menstruation & pregnancy

OCCUPATIONAL ASTHMA

- Materials encountered in the work place that cause or worsen asthma
- Allergens (over 200) from flour & grain, soldering fumes, paint spraying etc..
- 50% are cured if they are permanently removed from exposure
- Use face masks, respirators
- Drug control can be difficult

MAST CELL



MAST CELL DERIVED MEDIATORS

Preformed

- Histamine
- Eosinophil chemotactic factor
- Neutrophil chemotactic factor
- Neutral proteases
- Acid hydrolases
- Heparin proteoglycans

Membrane derived

- Leukotrienes
- Prostaglandins
 - Thromboxanes
 - Platelet activating factor

HISTORY

Known precipitating factors

- Nocturnal cough
- Shortness of breath
- Wheeze

KEY POINT

Nocturnal cough is the earliest feature of developing new asthma or deterioration of well-controlled of asthma

EXAMINATION (1/2)

Nil in between attacks

- No finger clubbing
- Abnormal chest wall shape (severe childhood asthma)
- Reduced chest wall movement
- Central mediastinum

EXAMINATION (2/2)

- Normal tactile vocal resonance
- Normal percussion note
- Normal vesicular breath sounds, prolonged expiratory phase
- Added breath sounds: bilateral, expiratory polyphonic wheeze
 - N.B. localized monophonic wheeze

INVESTIGATIONS (1/2)

Clinical diagnosis

- CXR: normal (or signs of hyperinflation)
- Eosinophils in blood or sputum

Lung function tests:

- Bronchial provocation tests
- Diurnal variation of peak flow

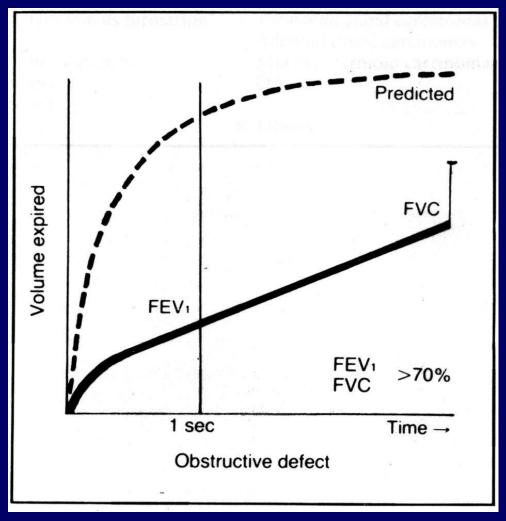
INVESTIGATIONS (2/2)

- Skin prick tests
- Spirometry
 - Obstructive ventilatory defect: FEV1/FVC
 - With >15% improvement in FEV1 and 200 ml
 absolute rise with bronchodilator
- Trial of corticosteroids (30mg prednisolone o.d. for 2 weeks)

SKIN PRICK TESTS



SPIROMETRY: CHRONIC AIRFLOW LIMITATION



KEY POINT

Asthma is a clinical diagnosis; there is no one diagnostic test to confirm clinical impression

MANAGEMENT

Aim: To allow the patient to live a normal active life

Requires patient motivation and education

- Non-drug treatment
- Drug treatment
 - Chronic outpatient asthma
 - Acute severe asthma

NON-DRUG TREATMENT

- Education and reassurance
- Rapid identification of allergen
- Avoidance is difficult e.g. house dust mite
 - Reduce soft furnishings
 - Synthetic fillings for pillows and mattresses
 - Regular dusting

MECHANISMS FOR ASTHMA DRUG THERAPY

- Preventing mediator formation
- Preventing mediator release
- Preventing mediators reaching targets
- Physiological antagonism of mediators

DRUGS

Relievers

- SALBUTAMOL
- terbutaline
- salmeterol
- IPATROPIUM
- THEOPHYLLINE

<u>Preventers</u>

- GLUCOCORTICOIDS
 - beclomethasone
 - budesonide
 - fluticasone
- Na cromoglycate
- Montelukast
 - Zafirlukast
 - •Zileuton

RELIEVERS

Drugs that reverse acute bronchoconstriction:

B₂ agonists

Methylxanthines

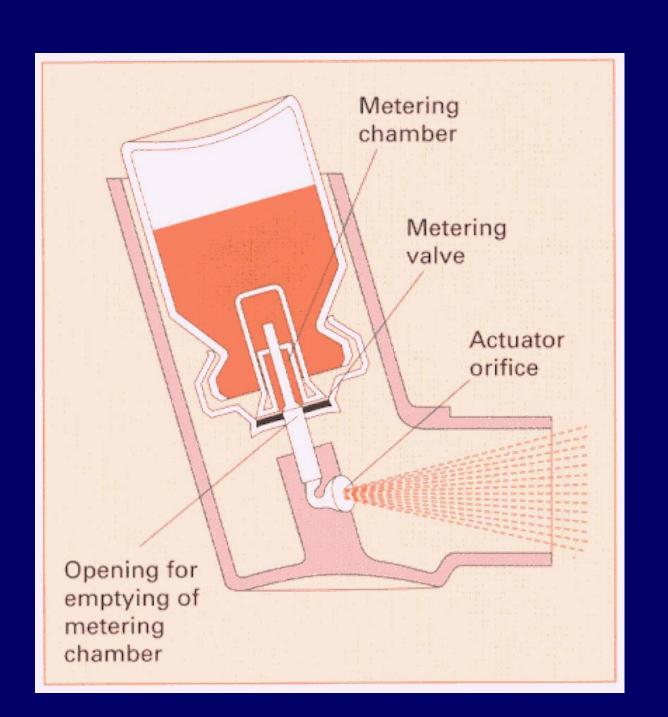
Anti-muscarinics

Anti-leukotriene agents

PREVENTERS

Do not cause acute bronchodilatation: no use for acute treatment

- 'Anti-inflammatory action'
- reduce bronchial hyper-reactivity
 - reduce entry of inflammatory cells
 - inhibit release of mediators from cells
 - reduce formation of mucosal oedema by mediators



INHALER TECHNIQUE

- Shake canister
- Exhale to FRC i.e. end of tidal breathing, not RV
- Simultaneously activate inhaler and inhale to TLC
- Hold breathe for 10 seconds
- Maximally 15% reaches bronchial tree

SPACER DEVICES



MANAGEMENT OF OUTPATIENT ASTHMA

Principles of Management

- Educate patients on deteriorating control
- Aim to gain control of symptoms rapidly
 - Use short courses of oral steroids as required
- Monitor compliance & inhaler technique
- Once well controlled, reduce doses progressively until symptoms reappear

5 STEPS OF ASTHMA MANAGEMENT

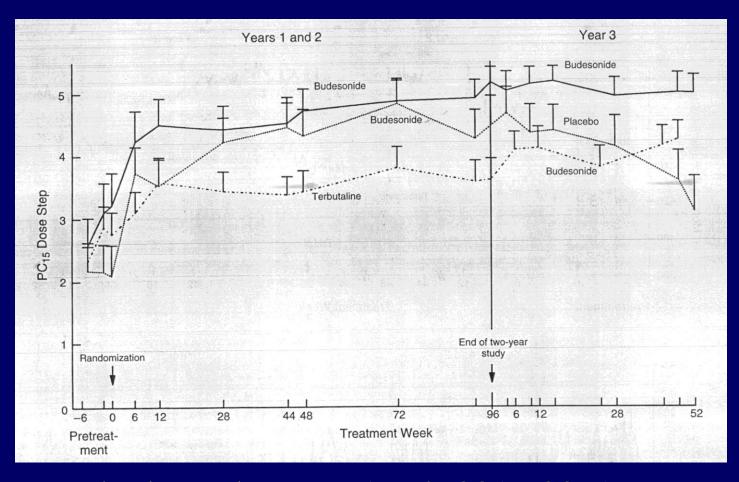
 $SA \beta_2$ agonist

Step 1 + Low dose ICS

Step 1

Step 2

VALUE OF ICS AT STEP 2



Haahtela et al NEJM 1994; 331:700-5

5 STEPS OF ASTHMA MANAGEMENT

 $SA \beta_2$ agonist

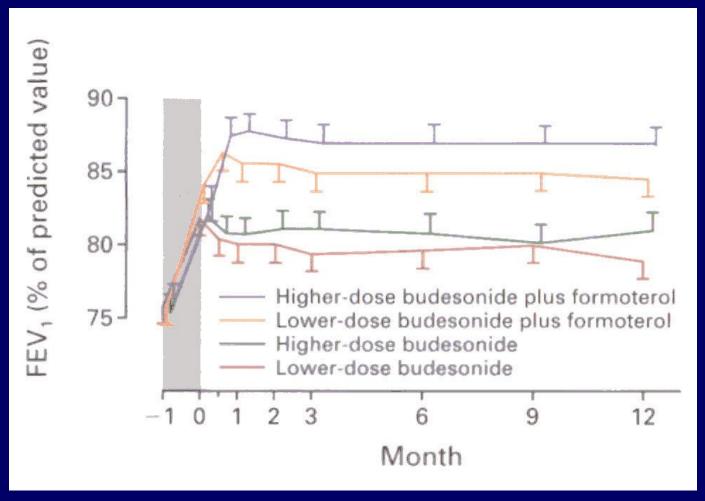
Step 1 + Low dose ICS Step 2 + either High dose ICS, or LA β_2 agonist

Step 1 S

Step 2

Step 3

USE OF LAB₂ AGONISTS AT STEP 3



Pauwels et al NEJM (1997); 337:1405-11

5 STEPS OF ASTHMA MANAGEMENT

Low dose $SA \beta_2$ agonist

Step 1

Step 2

Step 1 +

ICS

Step 3

Step 4

regular oral steroids

Step 4 +

Step 2 + either High dose ICS, or LA B₂ agonist

Step 3 +

High dose

ICS +

LA β_2 , anti-

cholinergic,

theophylline,

or oral B₂

Step 5

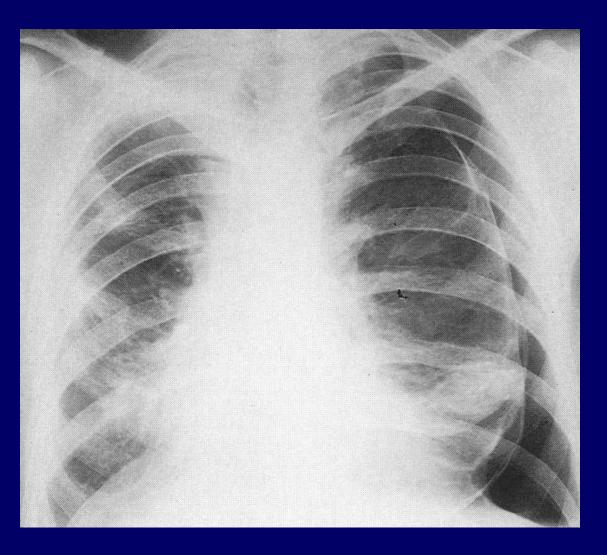
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Nocturnal cough is the earliest feature of developing new asthma or deterioration of well-controlled of asthma

COMPLICATIONS

- Pneumothorax
- Allergic bronchopulmonary aspergillosis and bronchiectasis
- Chronicity: irreversible airway obstruction
- Cor Pulmonale

PNEUMOTHORAX



CHRONICITY

Distinguishing Asthma from COPD

- Cigarette exposure
- Symptoms in earlier life (?atopy)
- Variability of symptoms

Why bother?

SUMMARY

- Definition
- Pathology
- Epidemiology
- Aetiology
- Precipitating factors

- Clinical features
- Investigations
- Management
 - Non drug
 - Drug

GUIDELINES

ON THE

MANAGEMENT OF ASTHMA



SRI LANKA MEDICAL ASSOCIATION

COLOMBO

2000