Wheezeing in Children

Prof RJ Green
Department of Paediatrics
Adventitious Airway Sounds

- Snoring
- Stridor
- Wheezing
- Crepitations
BERNOULLI THEOREM - 1738

\[ E = P + \rho gh + \frac{1}{2} \rho v^2 \]

**TOTAL ENERGY**  **PRESSURE ENERGY**  **GRAVITY ENERGY**  **FLOW ENERGY**

**Air is light, gravity is ignored:**

\[ E = P + \frac{1}{2} \rho v^2 \]

**SIMPLY**

the **FASTER** the FLOW
the **LOWER** the PRESSURE
Airway Diameter

- Trachea 20 mm
- Conducing Zone
- Respiratory Zone

Airway Generation

Cross Section Area

3 cm²

500
Cause of Wheezing

- Not from obstruction of small airways – Surface area too large
- From increased intrathoracic pressure + decreased large airway pressure = vibration of airway wall in large airways (Generations 1-5)
Wheezing

- Sign of lower (intra-thoracic) airway obstruction
- Small airways
Air Trapping

- Hyperinflated chest
- Barrel shaped
- Loss of cardiac dullness
- Liver pushed down
- Hoover sign
Hoover Sign

- Normal diaphragm movement
- Hyperinflation = diaphragm flattened
- Diaphragm contraction = paradoxical inward movement of lower intercostal area during inspiration
Acute Wheezing

- Asthma
- Bronchiolitis
- Foreign body
Bronchiolitis
What Is Bronchiolitis?

- Bronchiolitis is acute inflammation of the airways, characterised by wheeze
- Bronchiolitis can result from a viral infection
- Respiratory Syncytial Virus (RSV) may be responsible for up to 90% of bronchiolitis cases in young children
RSV Is a Common Virus Causing Bronchiolitis in Children

In a clinical study in Argentina, RSV was the most common virus isolated from a sample of children aged <5 years with acute lower respiratory infection.

Chronic Wheezing

- Thriving child – Happy wheezer
- Child failing to thrive - Causes
Exclude other conditions

- Structural problems: bronchoscopy
- URTD: Polysomnography
- Esophageal disease: Barium swallow, pH probes, scopes and gram
- Primary ciliary dyskinesia: nasal ciliary motility, Exhaled NO, EM, saccharine test
- TB: mantoux, induced sputum/ gastric lavage/ BAL = Culture, microscopy & PCR
- Bronchiectasis: HRCT scan, BAL
- CF: sweat test, nasal potentials, genotypes
- Systemic immune deficiency: Ig subtypes, lymphocytes & neutrophil function, HIV
- Cardiovascular disease: echo, angiography
WHEEZING PHENOTYPES

12 Longitudinal birth cohorts
Original Tucson Group (Taussig L et al 1985)

Persistent

Atopic

Non Atopic

Transient
TRANSIENT WHEEZERS

Commonest form of wheeze
Decrease lung function at birth
No airway hyper-responsiveness
Non Atopic
No immune responses to viruses
Resolves by 3 years
- Wheeze in first year – better outcome
- Wheeze 2-3 year – worse outcome due to maturity of immune system

Affected by:
- Teenage pregnancy & smoking
- Male gender
- Day care- infections
STRUCTURAL CONSIDERATIONS

Lung Growth: Fetal 8 years

Affected by:
- Temperature & O2 tension
- Nutrition & Smoking
- Functional disorders eg CDH
- Prematurity
- Growth factors-Gene repair
- Drugs (B₂ agonist/ C/S)

Risk factors for COPD
- Mx: antioxidant, retinoids, MMPI
PERSISTENT NON ATOPIC WHEEZER

- Lung function abnormal at birth and reduced in later life
- Non Atopic
- Airway hyper-responsiveness
- Peak flow variability
- RSV induced wheeze due to alteration in airway tone

BETTER OUTCOME THAN ATOPIC PERSISTENT WHEEZERS
Immunology associated with RSV

- Unknown - natural infection ≠ total immunity
- Re-infection by same strains by 6 weeks
- Recurrent disease - all infected by 3 yrs
- Infancy - immature immune system
- Maternal antibodies
- Incomplete protection, worse in premature
Prevalence of RSV Infection

n = 125 children followed from birth to 12m & 92 children followed from age 24-60m, virtually all were infected with RSV by 24 m

RSV-Induced Bronchiolitis May Consist of Several Phases

Phase I: Viral infection
- Days

Phase II: Acute phase
- Weeks

Phase III: Persistent wheezing
- Months

Long term: Wheezing and asthma

(Not to scale)
Wheezing Often Persists Post Bronchiolitis


83 children <2 years hospitalised with bronchiolitis, a large proportion had subsequent wheezing

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Children with wheezing (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-2 (n=83)</td>
<td>58%</td>
</tr>
<tr>
<td>2-3 (n=76)</td>
<td>76%</td>
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</tbody>
</table>

83 children <2 years hospitalised with bronchiolitis, a large proportion had subsequent wheezing.
RSV-Induced Bronchiolitis: Association With Asthma

n= 140, the incidence of asthma at 7.5 yrs was higher in children who had been infected with RSV compared with controls.

Children with asthma at age 7.5 years (%)

30%

RSV (n=47)

3%

Control (n=93)

TIME COURSE OF RECURRENT LOWER RESPIRATORY SYMPTOMS

Henry et al. 1985 Arch Dis Child
Webb et al. 1985 Arch Dis Child
Hall et al. 1984 J Pediatr
Therapeutic Options: viral induced wheeze
Options

- Humidified oxygen: Beneficial
- ?? Antibiotics -associated infection
- ??Efficacy of Bronchodilators
  - Inhaled & oral B2 agonists
  - Inhaled ipratropium bromide
  - theophyllines
- ??Use of corticosteroids
- ?Use on leukotriene antagonists
- ?Efficacy of immunoglobulin
Effect of Montelukast on RSV-Induced Bronchiolitis

- A RDBPC trial studied the effects of the LTRA montelukast on the post-infectious course of RSV-induced bronchiolitis
- 130 infants aged 3-36 months were randomized to receive montelukast or placebo
- Study treatment was montelukast 5 mg chewable tablets or matching placebo taken in the evening for 28 days
- Symptoms were recorded by the caretakers on diary cards

Montelukast Improved the Symptoms of RSV-Induced Bronchiolitis

Montelukast significantly improved symptom-free days & nights (daily median)

- Montelukast (n=61)
- Placebo (n=55)

Median symptom-free days and nights (%)

Days

p=0.015

Missing data were considered to be symptomatic days.

Montelukast: Reduced Exacerbations - Post RSV Bronchiolitis

Patients with exacerbations (%)

- Montelukast: 6.6%
- Placebo: 18.2%

* Withdrawal due to symptom severity, or attending emergency department or hospitalisation due to lung symptoms

PERSISTENT ATOPIC WHEEZER

- Lung function normal at birth but deteriorates with recurrent symptoms
- Increased symptoms with increasing age
- Airway liability
- Atopic (increase IgE at 6-9m; increase cytokines)
- Abnormal immune responses to viruses
PREDICTORS + RISK FACTORS FOR PERSISTENT WHEEZE

- Family history of Atopy
- Viral infections
- Allergens
- Environmental factors
  - Household chemicals (OR 2.3 CI 1.2-4.39)
- Genetics
- Multiple factors in combination
RISKS OF FAMILY HISTORY OF ATOPY

- No family history : 16%
- Single parent atopy : 22%
  Maternal Atopy : 32%
- Both parents atopic : 50%

(Aberdeen Study 1994)
MULTIPLE FACTORS

Triad of interactions
- Genetic variability
- Infections & environment
- Complex receptor interaction

- Single factor  PPV < 50%
- Combination factors  PPV  80%

(German MAS 1990)
% OF INFANTS SUBSEQUENTLY DEVELOPING ASTHMA

- Infant wheeze: 20%
- Infant wheeze with atopic parent(s): 40%
- Infant wheeze with atopic eczema and/or other food allergies: 50%
- Infant wheeze with atopic parent + positive skin prick test + raised sIL-2R: 90%
OUTCOME OF INFANT WHEEZING

- Low birth weight
- Pregnancy smoking
- Male Sex

- Affluence
- Atopy
- Low maternal age (first born)

Infant wheeze

- With viral infection alone:
  - Remission in 80%

- With various precipitants:
  - Persistent asthma (with or without evidence of atopy) in 50-60%
  - ?? COPD in adults
Points on examination

- LOW, FTT – systemic disease
- UAO: Tonsils, Adenoids, Polyps, Rhinitis
- Fixed Monophonic/asymmetric wheeze: foreign body
- Chest deformity: chronic lung disease
- Clubbing & Halitosis: chronic suppurative lung disease -bronchiectasis
- Stridor – bronchomalacia, vascular ring mediastinal syndrome
- Signs of cardiac or systemic disease