PAEDIATRIC RESPIRATORY REVIEWS (2000) 1, 274–278
doi: 10.1054/prrv.2000.0059, available online at http://www.idealibrary.com on IDEAL®

Paediatric Respiratory Reviews

MANAGEMENT

Atelectasis: mechanisms, diagnosis and management

D. G. Peroni and A. L. Boner*

Clinica Pediatrica, University of Verona, Verona and Istituto Pio XII, Misurina, Italy

KEYWORDS
atelectasis, children, asthma, cystic fibrosis, mucus plugs

Summary The term atelectasis describes a state of collapsed and non-aerated region of the lung parenchyma, which is otherwise normal. This pathological condition is usually associated with several pulmonary and chest disorders and represents a manifestation of the underlying disease, not a disease *per se*. Atelectasis may occur in three ways: (i) airway obstruction; (ii) compression of parenchyma by extrathoracic, intrathoracic, chest wall processes; and (iii) increased surface tension in alveoli and bronchioli. Chest radiographs using both the anterior-posterior and lateral projections are mandatory to document the presence of atelectasis. Differentiation from lobar consolidation may be a clinical dilemma. The treatment of atelectasis varies depending on duration and severity of the causal disease from chest physiotherapy to postural drainage, bronchodilator and anti-inflammatory therapy. Persistent mucous plugs should be removed by bronchoscopy. © 2000 Harcourt Publishers Ltd

INTRODUCTION

The developing lung is particularly predisposed to atelectasis once airway obstruction develops: in early childhood the airways are smaller and more collapsable, the chest wall is more compliant, and the collateral ventilation through intra-alveolar and bronchiole-alveolar pores is not completely developed. Atelectasis is influenced by pulmonary surfactant deficiency, such as in hyaline membrane disease (HMD) of prematurity and by surfactant dysfunction, in near-drowning, and in the acute respiratory distress syndrome (ARDS). Surfactant can directly modify alveolar tension with the changes of lung volumes, preventing collapse of small alveoli at low lung volumes and facilitating collapse at high lung volumes. The surfactant components (phospholipids, lipids, four surfactant-associated proteins, and calcium) may explain the properties of surfactant which are required for the normal function of peripheral gas exchange apparatus.[1]

PATHOPHYSIOLOGY

Air trapping and hyperinflation are produced by partial airway obstruction, atelectasis by complete obstruction. The gases trapped by complete occlusion are absorbed by the blood perfusing that region of the lung. The rate of gas absorption depends on the gas solubility: respirable air nitrogen and helium are absorbed in 2–3 h, 100% oxygen within a few min. For these reasons atelectases are more common in HMD and in the postoperative period, when high concentrations of oxygen are administered to the patient. If alveoli are collapsed a greater effort is required to reinflate them than when they are filled with air; an even greater effort is required to expand lung tissue which has been collapsed for days rather than for hours. Atelectasis produces alveolar hypoxia and pulmonary vasoconstriction to prevent ventilation-perfusion mismatching and to minimize arterial hypoxia. The vascular response is less effective when a large part of the lung is collapsed; if the blood cannot be diverted, it flows through the atelectatic non-ventilated region and produces intrapulmonary shunting.[2,3]

CAUSES OF ATELECTASIS

The causes of atelectasis in childhood are summarized in Table 1. Atelectasis may occur mainly in three ways: (i) airway obstruction; (ii) compression of parenchyma by extrathoracic, intrathoracic, chest wall processes; and (iii) increased surface tension in alveoli and bronchioli due to surfactant deficiency or dysfunction.

*Address for correspondence: Prof. Attilio Boner, Clinica Pediatrica Universita' di Verona, Policlinico GB Rossi, Via Menegone 37134 Verona, Italy. Tel: 39 045 8074615, Fax: 39 045 8200993, E-mail: boner@borgoroma.univr.it

1526–0550/00/030274 + 05 $35.00/0

Table 1 Causes of atelectasis in childhood.

1. Airway obstruction
(a) Exogenous
Foreign body
Recurrent aspiration
Histoplasmosis
(b) Endogenous
Polyps
Papilloma
Adenomas
Granulomas
Mucus plugs in
cystic fibrosis
asthma
bronchopulmonary dysplasia
bronchiectasis
pneumonia
immobile cilia syndromes
2. Compression of parenchyma
(a) Extrinsic bronchial compression
Tumors
Metastasis,
Lymph nodes
Cardiomegaly
(b) Intrathoracic compression
Chilothorax
Hemothorax
Pneumothorax
(c) Chest wall defects and Neuromuscular diseases
Abnormalities of diaphragm
Spinal muscular atrophy
Werdnig–Hoffmann disease
Muscular dystrophies
Guillain–Barre syndrome
3. Surfactant deficiency or dysfunction
Hyaline membrane disease
Pneumonia
Pulmonary oedema
Near-drowning
Adult respiratory distress syndrome (ARDS)

Most of the causes of atelectasis in childhood are due to airway obstruction: the obstruction leads to a non-ventilation of the distal airways; the gas residing in that region is completely absorbed by pulmonary blood flowing through that area. Compression of the parenchyma and increased surface tension produce an extrusion of the gas out of the alveoli and reduce the capability of the involved parenchyma to reinflate.

Intrabronchial obstruction may be exogenous (foreign body, histoplasmosis, etc.) or endogenous (polyps, papillomas, adenomas, granulomas, mucous plugs due to asthma, cystic fibrosis (CF), bronchopulmonary dysplasia, or bronchiectasis). The poor clearance of inflammatory debris, smooth muscle constriction and oedema of the bronchial wall in asthma and CF produce atelectasis by occluding the lumen of the airways; bronchiectasis causes an inadequate bronchial wall integrity, which may lead to obstruction.

Extrinsic bronchial compression may cause a compression of the adjacent bronchi: the more common process involved is enlargement of the hilar or mediastinal lymphnodes by a disease such as tubercolosis, sarcoidosis, immunodeficiency syndrome, lymphomas, or metastases in, for example, Wilms tumour, neuroblastoma or retinoblastoma. Cardiomegaly in congenital heart disease could produce atelectasis, especially of the left upper bronchus, left main bronchus and middle bronchus.[3]

Different disorders which occupy space in the mediastinum, such as chylothorax, haemothorax, and pneumothorax may cause compression of normal lung. Congenital abnormalities of the diaphragm (hernia, eventration, etc.) may produce atelectasis by occluding bronchi by the displaced viscera.

Neuromuscular diseases such as spinal muscular atrophy or Werdnig–Hoffmann disease, the muscular dystrophies and the Guillain-Barre syndrome, characterized by weakness of the diaphragm, are more rare causes of atelectasis.

Surfactant deficiency or dysfunction causes increased alveolar surface tension with consequent diffuse atelectasis; the most common cause is HMD, but pneumonia or pulmonary oedema may affect surfactant dysfunction. Surfactant insufficiency or inactivation are typical causes of atelectasis in ARDS. ARDS in childhood may be precipitated by shock, sepsis, trauma, aspiration or inhalation injury; these all produce surfactant dysfunction, which leads to an increase in surface tension.

Presentation of atelectasis

The signs and symptoms of atelectasis are often non-specific: the disease, which leads to the obstruction, may cause fever, cough, tachypnoea, wheezing, ronchi, and chest pain. Furthermore, in the case of infections such as bronchiolitis, bronchitis, pneumonia and tuberculosis and with tumours, asthma and CF, the presence of atelectasis does not change the clinical picture unless the obstructed area is large. Suggestive signs of atelectasis are localized reduced breath sounds or a constant wheeze. There may also be reduced chest wall expansion. If the obstruction involves a main bronchus, wheeze can be heard and cyanosis and asphyxia may be present, with mediastinal and cardiac displacement and elevation of the diaphragm.

Aspiration of a foreign body in a previously well child is an acute respiratory emergency. If a large bronchus is present immediate intervention is mandatory. If the foreign body is lodged in a smaller airway there may be a symptom-free period, followed by infection in the area of atelectasis causing fever, malaise and cough.

A full medical history and specific clinical characteristics may help to identify the aetiology of the atelectasis. The patient with bronchopulmonary dysplasia exhibits a

neonatal history of HMD and mechanical ventilation;[4] the patient with CF may present with failure to and steatorrhea, but persistent wheezing may give a clue to the presence of atelectasis.[5] Frequent vomiting or cough during and immediately after feeding, especially in young babies, suggests recurrent aspiration or gastroesophageal reflux.[6]

In asthma the presence of bronchial inflammation producing cellular debris and mucus plugs may cause airway obstruction, which is particularly frequent in the right middle lobe, leading to the so-called "right middle lobe syndrome"; this characterizes most cases of persistent atelectasis in childhood.[7] The right middle lobe bronchus is longer than other bronchi, starting at the bronchus intermedius after a sharp angle, leading to retention of intrabronchial secretions.

The occurrence of migrating or rotating atelectasis may be an early manifestation of neuromuscular diseases such as Werdnig–Hoffman disease.[8] Another rare cause of intrabronchial obstruction with ensuing persistent atelectasis, often accompanied by pneumonitis resistant to therapy (with wheeze, and haemoptysis), is a bronchial carcinoid tumour.[9]

Diagnosis of atelectasis

Once the diagnosis of atelectasis is suspected, chest radiographs using both the anterior-posterior and lateral projections are mandatory to document the presence, extent, and distribution of the atelectasis (Fig. 1). Radiographically, there can be problems differentiating atelectasis from simple lobar consolidation (Table 2).[10] With consolidations the alveoli are full of exudates and there is no significant loss of lung volume. However, as the pneumonia clears, atelectasis may develop because of mucus plugs or surfactant dysfunction. A more marked loss of volume in one lobe and compensatory emphysema in an accompanying lobe indicates the presence of atelectasis. The difficulty in diagnosis is best exemplified by atelectasis of the right middle lobe. In this case, the loss of volume is so small it may appear as a dense band, suggesting pleural thickening rather than an atelectatic lobe.[10] In atelectasis associated with greater loss of volume, other radiographic findings may be useful (Table 2). With loss of volume, the mediastinum shifts towards the involved lung: with volume gain, such as a pleural effusion or a large unilateral chest mass, it moves away from the involved lung. The presence of a unilateral elevated diaphragm may also suggest atelectasis, whereas it is unaffected in lobar pneumonia. With a pleural effusion the diaphragm may be displaced downwards and flattened on the side of the effusion. Ultrasonography may be useful in determining whether the opacity represents fluid or collapsed lung.[10]

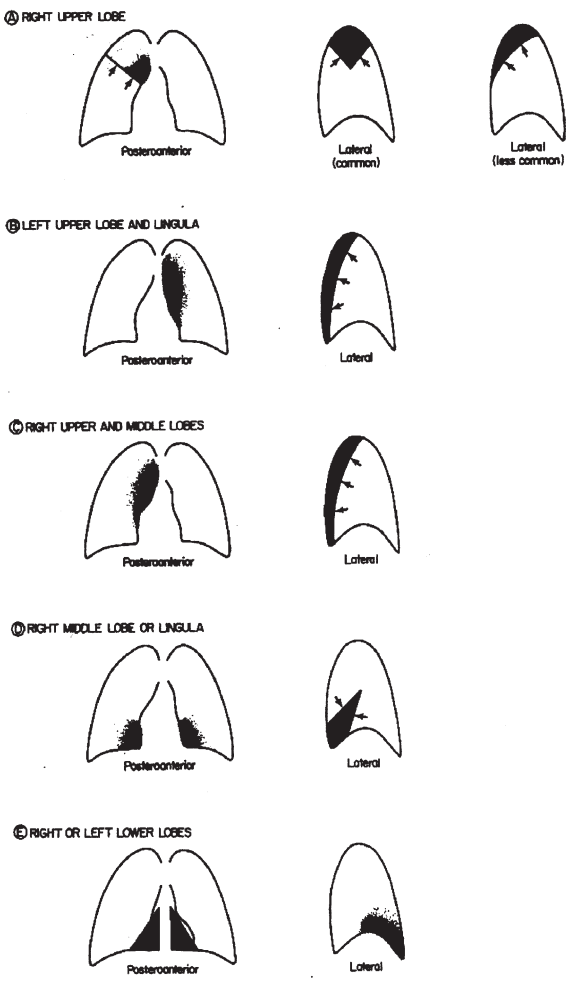

Figure 1 Chest X-rays using the antero-posterior and lateral projections document the presence and distribution of atelectasis.

Differentiating atelectasis from lobar consolidation is a common dilemma in the assessment of respiratory infections in children (Table 3). Viral infections commonly cause lower respiratory tract infections in childhood, whilst bronchial asthma causes atelectasis secondary to mucous plugging; in some patients radiographic differentiation of atelectasis and pneumonia is very difficult, and in

Table 2 Radiographic differences between atelectasis versus consolidation.

	Atelectasis	Consolidation
Lung volume loss	Marked	Not significant
Compensatory emphysema	++	–
Mediastinum shift	Toward lesion	–
Diaphragm position	Unilateral elevated	Not significant

Table 3 Radiological differentiation of atelectasis versus consolidation: the pulmonary infiltrate patterns.

Type of infiltrate	Atelectasis	Consolidation
Virus	+++	–
Tuberculosis	+++	+
Fungi (actinomycosis)	+	+
Pneumocystis carinii	–	+/–
Mycoplasma	+++	+++
Pertussis	+	+/–
Pneumococcus	–	+++
Staphylococcus	–	++
Haemophilus	–	+++
Aspiration pneumonia	+++	++
Pulmonary haemorrhage	–	+
Asthma	+++	+
Cystic fibrosis	+++	+

such cases a clinical history is helpful. Coryza, nasal congestion and mild fever generally accompany viral infections; in asthma the consolidation is almost always due to atelectasis, since bacterial pneumonia seldom precipitate an acute asthma attack. If confusion remains, chest radiographic follow-up may be useful to demonstrate rapid resolution, which is more rapid with atelectasis.

Peripheral atelectasis may be mistaken for a tumour mass, in which case a CT scan is helpful.

Atelectasis may occur in any lobe or segment of the lung, but the right and left lower lobes are the most frequently involved. The right middle lobe is vulnerable from enlargement of the hilar lymph nodes and is also the most commonly affected in asthma (Fig. 2).

In recurrent aspiration syndrome (usually caused by milk aspiration in infants), the most frequently affected lobes are the posterior areas of the upper and lower lobes, because the infants lie supine for much of the day. In toddlers or older children, who spend more time vertical, the lower lobes the lingula and the right middle lobe are more frequently affected.[6]

Treatment of atelectasis

The treatment of atelectasis varies depending on the cause, the duration and the severity. Respiratory infections are common causes of atelectasis in childhood, but are generally transient (except TB) with resolution within 3 months following antimicrobial therapy.[3] If the atelectasis does not resolve, bronchodilator inhalation and physiotherapy are performed.[3] Aerosol therapy by inhalation of jet-nebulized saline increases sputum volume and clearance compared with physiotherapy alone.[11] The use of inhaled β2-agonists in patients with CF and bronchiectasis should be reserved for those who have demonstrated a clinical or lung function response to bronchodilator.[12]

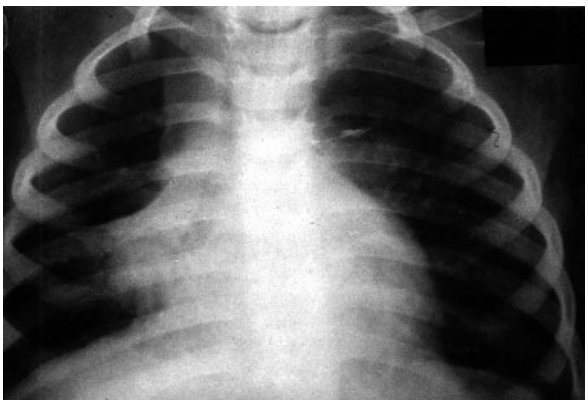

Figure 2 Atelectasis of the right middle lobe caused by extrinsic bronchial compression determined by enlargement of hylar lymph nodes.

Chest physiotherapy is commonly used in paediatric respiratory diseases: the benefit is thought to be due to mobilization of secretions by huffing and coughing from the smaller to the more central airways. Patients with chronic pulmonary diseases, such as CF and ciliary dyskinesia, usually improve with antibiotics and regular home chest physiotherapy. If atelectasis occurs in the intensive care unit, in the postoperative period or during mechanical ventilation, postural drainage and chest percussion, together with bronchodilator therapy, may help. When atelectasis persists, bronchoscopy should be used to investigate and remove the cause of the obstruction. Resistant atelectases have been successfully treated by positive pressure ventilation using a flexible bronchoscope inserted into a subsegmental bronchus with a small balloon cuff at the distal end of the scope.[13] The use of nebulized recombinant human Dnase I (rhDNase), which hydrolyses bacterial and cellular DNA present in sputum plugs, has been described in the management of a mechanically ventilated child with asthma.[14] It has also been used in patients with cystic fibrosis.[15] The diagnosis and early management of atelectasis in children is important so as to optimize resolution and prevent permanent atelectasis with the possibility of irretrievable lung damage.

PRACTICE POINTS

- In early childhood the airways are smaller and more collapsible.
- The signs and symptoms of atelectasis are often non-specific.
- The presence of atelectasis does not change the clinical picture unless the obstructed area is large.
- In the case of persisting atelectasis, bronchoscopy should be considered.

RESEARCH DIRECTIONS

- Differential diagnosis of migrating or rotating atelectasis.
- Greater understanding of mechanisms of surfactant insufficiency or inactivation.
- Evaluation of chest physiotherapy programs.
- Application of positive pressure ventilation via flexible bronchoscope.

REFERENCES

1. "Lung growth and development." In: Phelan, Olinsky, Robertson R (eds). *Respiratory illness in children*. Oxford: Blackwell, 1994; 1–7.
2. Redding GJ. Atelectasis in childhood. *Ped Clin North America* 1984; **31**: 891–907.
3. Hazinski TA. Atelectasis. In: *Chernick and Boat* (eds). *Kendig's disorders of the respiratory tract in children*. Philadelphia: Saunders Co, 1998; 634–641.
4. Kennedy SD. Lung function outcome in children of premature birth. *J Paediatr Child Health* 1999; **35**: 516–521.
5. Ruzal-Shapiro C. Cystic fibrosis: an overview. *Radiol Clin North Am* 1998; **36**: 143–161.
6. "Pulmonary complications of inhalation." In: Phelan, Olinsky, Robertson R (eds) *Respiratory illness in children*. Oxford: Blackwell, 1994; 252–268.
7. De Boeck K, Willems T, Van Gysel D, Corbeel L, Eeckels R. Outcome after right middle lobe syndrome. *Chest* 1995; **108**: 150–152.
8. Leistikow EA, Jones NE, Josephson KD, de Sierra TM. Migrating atelectasis in Werdnig-Hoffmann disease: pulmonary manifestations in two cases of spinal muscular atrophy type 1. *Pediatr Pulmonol* 1999; **28**: 149–153.
9. Wang LT, Wilkins EW, Bode HH. Bronchial carcinoid tumors in pediatric patients. *Chest* 1993; **103**: 1426–1428.
10. Swischhuk LE, John SD. *Differential diagnosis in pediatric radiology* Baltimore: Williams and Wilkins, 1995; 64–78.
11. Conway J, Fleming JS, Perring S, Holgate ST. Humidification as an adjunct to chest physiotherapy in aiding tracheo-bronchial clearance in patients with bronchiectasis. *Respir Med* 1992; **86**: 109–114.
12. Eber E, Oberwaldner B, Zach M. Airway obstruction and airway wall instability in cystic fibrosis: the isolated and combined effect of theophylline and sympathomimetics. *Pediatr Pulmonol* 1988; **4**: 205–212.
13. Krell WS, Prakash UB. Therapeutic bronchoscopy. In: Prakash UB (ed). *Bronchoscopy*. New York: Raven Press, 1994.
14. Greally P. Human recombinant Dnase for mucus plugging in status asthmaticus. *Lancet* 1995; **346**: 1423–1442.
15. Harms H, Matouk E, Tournier G. Multicenter, open-label study of recombinant human Dnase in cysti fibrosis patients with moderate lung disease. Dnase International Study Group. *Ped Pulmonol* 1998; **26**: 155–161.