

Acute Lobar Atelectasis



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Lobar atelectasis (or collapse) is an exceedingly common, rather predictable, and potentially pathogenic companion to many forms of acute illness, postoperative care, and chronic debility. Readily diagnosed by using routine chest imaging and bedside ultrasound, the consequences from lobar collapse may be minor or serious, depending on extent, mechanism, patient vulnerability, abruptness of onset, effectiveness of hypoxic vasoconstriction, and compensatory reserves. Measures taken to reduce secretion burden, assure adequate secretion clearance, maintain upright positioning, reverse lung compression, and sustain lung expansion accord with a logical physiologic rationale. Both classification and logical approaches to prophylaxis and treatment of lobar atelectasis derive from a sound mechanistic knowledge of its causation.

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Collapse of major segments of the lung, acute lobar atelectasis (ALA), ranks among the most commonly encountered problems of critical care. Although clinical consequences are often significant, ALA development is rather predictable. For example, the varied stresses associated with thoracic or abdominal surgery alter the normal balance of forces that keep the lung inflated while interfering with effective airway clearance. In such settings, special precautions must be taken to avoid segmental collapse in patients predisposed by advanced age, obesity, copious airway secretions, weakened respiratory muscles, or lung edema. Both classification and logical approaches to prophylaxis and treatment of ALA derive from a sound mechanistic knowledge of its causation. Recognition begins by understanding the underlying segmentation of the healthy lung.

Lobar Anatomy

One might wonder why the lung is divided into lobes by fissures that are relatively impermeable to gas transfer. One rational explanation is that separation of lung compartments allows containment of inflammation and tissue damage (eg, infection) to the region of the gas-exchanging parenchyma already affected.¹ Such compartmental separation undoubtedly served a vital and life-prolonging function in the preantibiotic and pre-life support eras that preceded our own. Occasionally, some fissures remain incomplete, allowing collateral ventilation and peripheral air drift that slow or prevent segmental collapse despite predisposing factors such as proximal obstruction or locally reduced transpulmonary pressure.² Anatomically, the contours, volumes, and connections to the

ABBREVIATIONS: ALA = acute lobar atelectasis; FRC = functional residual capacity; PEEP = positive end-expiratory airway pressure

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trachea of the individual lobes are distinct from one another. In the adult, lower lobe gas volumes exceed those of the upper lobes, with right lung volumes greater than left lung volumes.^{3,4} The narrower main bronchus serving the left lung angulates more sharply and divides more peripherally than that serving the right. When semi-upright, gravity predisposes mucus to collect dependently. Collapse of the lower lobe occurs approximately twice as commonly on the left as on the right,⁵ even though caudal drainage of tracheal secretions anatomically should favor the opposite. One reasonable explanation highlights the localized effects of cardiac compression and deformation on gravitationally dependent bronchi.⁶ Predisposition for lower lobe collapse does not hold for very young children, in whom upper lobe collapse is strikingly dominant, especially on the right.^{5,7,8}

The pediatric predisposition to ALA has several explanations. The lungs of young children are surrounded by differently configured chest walls with greater flexibility, characteristics that diminish the outward recoil that aids lung expansion in the adult.^{8,9} They also have less well-developed collateral ventilation and compressible, small-diameter bronchi. The bronchi are also oriented differently among young children, favoring mucus clogging of the dependent upper lobe.

Pathogenesis

Normal

Competition between the tendencies of the lung to collapse and the chest wall to expand strikes the balance of functional residual capacity (FRC). FRC normally declines only marginally (if at all) with age, owing to counterbalancing structural effects of lung and chest wall on lung recoil.¹⁰ At all ages, however, FRC falls impressively in recumbent body positions, due to alterations of chest wall configuration, hydrostatic forces, and abdomen-impeded diaphragmatic descent.^{11,12} Dependent transpulmonary (alveolar minus pleural) pressures are lower than those situated above them. The lung volume at which closure of dependent airways begins is termed the closing volume. As aging proceeds, FRC may slip below the closing volume, especially in recumbency.¹² In the transition from the upright (90°) to a horizontal (0°) position, the loss of FRC volume is due primarily to the diaphragmatic ascent caused by increasing infradiaphragmatic abdominal pressure. These positional changes of pressure and volume are most pronounced over the

range of 60° to 0° from horizontal.^{11,13} Maintaining alveolar patency in well-perfused dependent lung zones requires periodic increases of regional transpulmonary pressure and stretching forces, as well as continual refreshment by ventilation at a rate sufficient to counter the capillary absorption of oxygen and maintain steady composition of alveolar gases.¹⁴⁻¹⁶ Monotonously small tidal volumes predispose to emergence of atelectatic zones,¹⁷ a tendency countered by periodic sighs and/or positive end-expiratory airway pressure (PEEP).^{13,16,18}

Disease

In acute illness, multiple factors unite to promote regional collapse. Bronchospasm, edema of airway mucosa and lung parenchyma, recumbency, ineffective clearance of secretions, compressive abdominal forces, and impaired diaphragmatic function are examples of such predispositions.^{19,20} Neuromuscular diseases impair depth and distribution of respiration as well as cough, favoring ALA.^{13,21} Normal diaphragmatic action during both phases of the tidal cycle preferentially stretches and ventilates contiguous dependent lung regions.²² Persistence of diaphragmatic activity during expiration slows the rate of deflation, helping to prevent closure of dependent small airways.²³ Conversion to positive pressure ventilation nullifies that preferentially dependent distribution of ventilation, particularly when inflation proceeds passively.²² Following an acute lung injury, monotonous and relatively small (“lung-protective”) tidal volumes encourage progressive collapse.^{17,24} Moreover, prolonged diaphragmatic rest promotes weakness and atrophy that linger into the recovery stage postextubation.²⁵

Surgery that involves the thorax and/or abdomen strongly predisposes to postoperative atelectasis.^{26,27} Anesthesia impairs the mucociliary escalator and promotes secretion generation.²⁸ Meanwhile, intubation degrades coughing effectiveness in several ways: prevention of glottic closure; cuff-blockaded transfer of tracheal secretions to the pharynx; and co-axial positioning of the tube’s narrow opening. Simultaneously, incisional pain restricts inspiration.^{19,29,30} Although well-titrated analgesia may help address pain and aid coughing, excessive analgesia reduces the drive to breathe and blunts coughing reflexes.³⁰ Opiates tend to elevate the set point of arterial CO₂, but moderate doses typically preserve or increase tidal excursions while reducing breathing frequency.^{31,32} Independently of any associated pain, incisions made in the lower or midline thorax and or upper abdomen

adversely affect diaphragmatic contraction.²⁶ By comparison, lower abdominal incisions are better tolerated, and laparoscopic procedures exert very limited impact on diaphragmatic functioning.³³

Pathogenetic Categories of Lobar Atelectasis

Categories of ALA, which often overlap in the individual, are usually classified according to the primary mechanism of their pathogenesis: absorptive (resorption), compressive, surfactant depleted/adhesive, and passive (Table 1).^{7,29}

Absorptive Collapse

The absorptive mechanism is frequently encountered in the ICU because mucus retention favors plugging, and enriched inspired oxygen favors gas absorption.¹⁵ Mucus production is often increased and the mucociliary escalator made dysfunctional by inflammatory diseases and the endotracheal tube.^{34,35} Bronchospasm has been reported as the sole or primary cause of ALA,³⁶⁻³⁸ but this mechanism seldom occurs without simultaneous secretion plugging. During room air breathing, venous blood flowing past normally functioning alveoli harbors gas tensions that sum to approximately 50 mm Hg below atmospheric levels, largely due to the arteriovenous differential of oxygen tensions.^{7,11,39} Maintaining this ongoing pulmonary capillary to alveolus diffusion gradient while assuring sustained alveolar patency requires continual gas refreshment and maintenance of adequate transpulmonary pressure to counter inward lung recoil.⁴⁰

Breathing supplemental oxygen promotes gas absorption in rough proportion to the compensatory decline of alveolar nitrogen tension. When breathing pure oxygen, absorptive collapse of a lobe with compromised ventilation may take as little as 60 min, whereas complete absorption while filled with room air may require 12 to 24 h.^{15,39} Seriously reduced regional

TABLE 1] Classification and Mechanisms of Acute Lobar Atelectasis

Type	Example
Obstructive	Central mucus plug
Passive (relaxation)	Large pleural effusion
Compressive	Pneumothorax
Adhesive	Surfactant depletion/ inactivation
Mixed Forms	Lobar pneumonia

ventilation encourages critical closure in those low ventilation/perfusion areas.^{39,41} Alveolar closure is favored when the recoil forces of surface tension rise in response to surfactant depletion or inactivation. Once closed, the lining surfaces of atelectatic lung adhere, substantially raising the transpulmonary pressure required to re-open those “sticky,” surfactant-deficient units.^{39,42}

Passive and Compressive Collapse

Airway occlusion is not a prerequisite for ALA development or persistence. Regional ventilation may be critically compromised by external lung compression (eg, pleural effusion, increased abdominal pressure) or by failure to maintain sufficient regional expansion and ventilating forces.^{7,39,43} During health, periodic sighs and adjustments of body position counter this local tendency for collapse and prevent its progression.^{13-16,44} Conversion from a normal pattern of spontaneous breathing in which the diaphragm actively contracts to passive inflation compromises caudal and gravitationally dependent ventilation.^{21,22}

Positional Factors

Recumbent and semi-recumbent positions impair resting lung volumes. Depending on lean body size and age, the healthy adult loses 700 to 1,200 mL of FRC in the transition from sitting to fully supine.⁴⁵ Somewhat less may be lost in transition to lateral positions. These positional losses of lung volume are not equally shared; compression by the heart and mediastinal contents as well as the upward thrust of the abdominal organs further restrict dependent expansion when horizontal. Such compressive factors are reinforced by massive obesity.⁴⁶ In the fully supine (0°) position, the weight of the heart and mediastinum narrow and pinch the left lower lobe bronchi.⁶ Fully lateral positions favor expansion of the uppermost lung at the expense of contralateral compressive collapse and secretion pooling. Prone positioning helps even the distribution of transpulmonary pressures and promotes mouthward migration of secretions from dorsal regions.⁴⁷ With the larger lower lobes uppermost, the incidence, volume losses, and consequences of ALA when prone are presumably less than in the supine or lateral postures.

Consequences of Lobar Atelectasis

Although pressures within the collapsed lobe have not been well studied, they should vary according to the mechanism of atelectasis: lower when obstruction and absorption occur than when external compression is the

cause. When relatively negative, interstitial pressures within the perfused but collapsed lobe tend to draw fluids into the interior and distend embedded blood vessels,⁴⁸ helping to account for the opacity and volumetrically incomplete collapse of the airless lobe on most radiographic images. This pooled fluid provides a potentially rich source of nutrients for inoculated organisms, and once airless, the collapsed lobe is not easily cleared of obstructing mucus. Surfactant functionality declines and mild local inflammation develops,⁴⁹ often signaled by very-low-grade fever.⁵⁰ Higher fevers indicate active infection and herald progression to pneumonia.⁵¹⁻⁵³ Accompanying influx of activated leukocytes promotes vascular permeability.^{54,55} During full-blown inflammation, cellular breakdown and released DNA encourage stickiness of luminal mucus plugs. Apart from their internal effects within the lobe, the relatively negative local pressures that surround the atelectatic segments favor shift of contiguous anatomic structures.^{56,57}

Physiologic consequences of lobar collapse include impaired global lung compliance, arterial hypoxemia, and increased ventilatory workload. Although ALA is generally well tolerated hemodynamically, extensive collapse accentuates right ventricular afterload due to mechanical vascular occlusion and reactive vasoconstriction.⁵⁸⁻⁶¹ The degree to which these effects become clinically manifest depends on the extent of collapse, vasoreactivity of the aerated lung, and the underlying condition. Therefore, ALA often is detected first on a chest radiograph performed for unrelated reasons. Symptoms and hypoxemia are most likely when massive collapse occurs suddenly and compensatory hypoxic pulmonary vasoconstriction proves inadequate.^{15,41,60-62} Initial hypoxemia tends to resolve with passing time.^{15,61}

Re-expansion edema may become evident for a short period following reinflation of a long-collapsed and surfactant depleted lobe.^{63,64} Although this phenomenon should be an expected consequence of pathophysiologic mechanics and surfactant depletion, overt flooding occurs uncommonly unless highly negative pleural pressures arise from overly aggressive thoracentesis or pleural suctioning.⁶⁴ In the absence of other pathology or abnormality, lobar collapse seldom generates pleural effusion. If already present, however, segmental atelectasis alters the distribution of intrapleural pressure and pleural effusion, often confounding imaging interpretation.^{65,66}

Epidemiology of ALA

Higher risk of developing ALA accompanies diseases and disorders that distort the normal thoracic configuration or impair respiratory muscle strength, endurance, and cough efficiency.^{29,67,68} Open surgical interventions that involve the heart, spine, or upper abdomen are especially hazardous.²⁶ Some degree of lobar collapse may complicate as many as 90% of cardiac operations, 75% of spinal interventions, and 25% of upper abdominal procedures.^{7,29,39} Associations of ALA with massive obesity and advanced age are surprisingly weak. Once BMI exceeds a certain threshold, further elevations correlate poorly with atelectatic postoperative complications.⁶⁹⁻⁷¹ Of itself, advanced age does not significantly affect resting lung volume,⁷² nor is it a strong risk factor for ALA. However, elderly status predisposes to comorbidities (eg, chronic bronchitis, esophageal reflux) that do promote collapse.

Diagnosis

Physical Findings

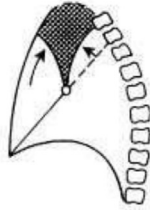
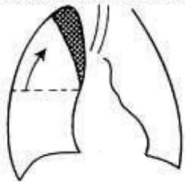
On physical examination, localized dullness to percussion together with normal or hyperresonance over uninvolved lobes is characteristic of ALA. Dullness over the affected lobe may not extend all the way to the lateral rib margins, depending on the lobe(s) that collapsed.^{7,69} Increased (“bronchial” quality) breath sounds are typically heard directly over the involved segments when the conducting bronchus to the collapsed lobe remains patent. However, absent breath sounds accompany dullness to percussion when the ventilating conduit is blocked, as by a secretion plug.⁶⁸ In semi-upright positions, a large pleural effusion can be distinguished by projection of bronchial breath sounds to the compressed but partially aerated zone immediately above percussion dullness. Unlike ALA caused by other mechanisms, effusion-related dullness shifts with position, provided that the fluid collection is subtotal and freely mobile. Whole lung collapse often produces hyperresonance of the uninvolved side, but mediastinal shift is less reliably detected in adults than in children.⁸ Whole lung collapse that occurs acutely almost invariably results from occlusion by a misplaced endotracheal tube or retained mucus. In such cases, bronchoscopic inspection offers both a high-yield diagnostic and a therapeutic intervention.^{50,73,74}

Imaging

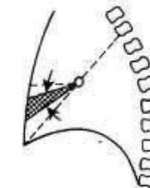
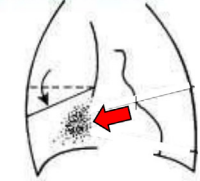
Radiographic imaging has been instrumental for diagnosing ALA for more than a century.⁷⁵ Although

CT imaging is now the definitive test,⁷⁶⁻⁷⁸ the bedside chest radiograph remains the imaging modality that usually raises initial concerns regarding lobar collapse (Fig 1). A rich literature describes varied radiographic

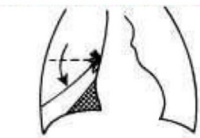
Right Upper Lobe Collapse



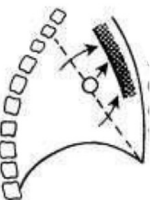
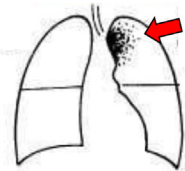
Right Middle Lobe Collapse



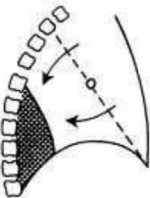
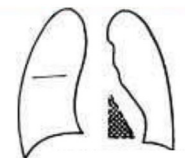
Right Lower Lobe Collapse



Left Upper Lobe Collapse



Left Lower Lobe Collapse



Lingular Collapse

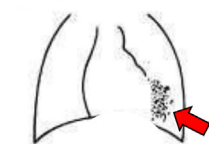


Figure 1 – Schematic drawings depicting frontal and lateral views of chest radiographs of acute and nearly complete lobar atelectasis that result from absorptive mechanisms of collapse. Dark shading indicates the airless and contracted collapsed lobes. Large arrows indicate zones of typically indistinct radiographic margins of the atelectatic tissues (stippled pattern).

TABLE 2] Radiographic Signs of Acute Lobar Atelectasis

Increased opacity of affected lobe
Bronchovascular crowding
Narrowing of the ipsilateral intercostal spaces
Compensatory hyperinflation
Compensatory shift of adjoining structures and/or diaphragm
Hilar displacement
Ipsilateral hemithoracic contraction (in massive collapse)
Silhouette sign affecting contiguous mediastinal structures

presentations.^{7,56,57,76,78} Direct signs of ALA on chest films include fissure deviation, parenchymal opacification with unbroken linear borders, and vascular displacement (Table 2). In about one-third of cases (those not caused or maintained by an absorptive mechanism), air bronchograms penetrate the otherwise airless zone.⁵⁰ Opacification of the lobe may be partial early in this process, allowing air-tissue contrast to reveal crowding of major vessels. Indirect signs of ALA include the following: ipsilateral diaphragmatic elevation; hilar displacement; shift of the heart, mediastinum, and trachea toward the affected area; compensatory hyperinflation; rib approximation; and “silhouette” obliteration of normal air-tissue boundaries.^{56,57} The extent to which these indirect signs of ALA are detectable on the plain chest radiograph depends not only on patient orientation, body positioning, and film penetration but also on the relative mobility of lung, diaphragm, and abdominal contents. Whenever there is a serious diagnostic question, a chest CT examination is indicated. IV contrast helps distinguish pleural effusion from collapsed tissue. Only CT imaging provides the necessary spatial resolution to confirm or exclude the ALA diagnosis.⁷⁶⁻⁷⁸ Although bedside ultrasound may distinguish pleural fluid from primary collapse, distinguishing signs are often subtle.⁷⁹⁻⁸¹ Regionally absent lung sliding, lung pulse, and static air bronchograms are specific but inconsistent ultrasonic indicators of ALA. Although electrical impedance tomography may aid in ALA diagnosis and monitoring of therapy,^{82,83} access to this technology is not widespread.

Management

Although there are numerous suggestive reports, convincing data regarding efficacy are not plentiful.⁸⁴

Physiologic principles suggest, however, that the management of ALA should center on restoration and maintenance of airway patency and providing adequate regional transpulmonary pressure to overcome the forces of recoil. Once reversed, prophylactic measures are usually required to prevent recurrence in those patients remaining vulnerable to relapse.

Restoration of Adequate Transpulmonary Pressure

Relief of compression due to increased abdominal pressure, pneumothorax, or pleural effusion must be strongly considered as the first intervention step when these extrapulmonary factors play a pathogenetic role. Vital capacity maneuvers, sighs, adequate tidal volume, and sufficient end-expiratory airway pressure (PEEP or CPAP) are clearly important once patency of the conducting airway has been assured. However, when used alone in the presence of a mucus plug, these same measures typically prove ineffective. Massive obesity presents special challenges, as dependent intrathoracic pressures may be impressive and require high levels of PEEP to offset.⁸⁵

Position

External traction on the affected anatomic segments can be applied and maintained by appropriate body orientation. Elevation of the affected lobe and secretion drainage by positioning might be expected to re-open nondependent lung segments in the setting of ALA (as in ARDS), but repositioning alone seldom proves effective if the atelectasis is adhesive or if the relevant bronchus remains plugged. Body positioning is generally more effective as prophylaxis or when combined with an effective secretion-mobilizing modality.^{86,87} Semi-upright positioning at times proves counterproductive, with or without an occluded conducting passage.⁷⁰ For example, a large, position-compressed abdomen may increase dependent pleural pressure. However, once airway occlusion has been relieved, fully upright body positioning is a valuable preventative intervention. Upright positioning to prevent airway compression and collapse is especially important for massively obese patients, in whom routine 30° to 45° postures fail to avoid dependent airway closure.⁸⁸ Upright positioning with directional reversal of the cephalad forces imposed by the abdominal contents assumes added value in the presence of diaphragmatic paralysis or weakness following lengthy mechanical ventilation.^{25,26} For quadriplegia, however, this general mandate to maintain verticality may be questioned. In such patients, upright orientation increases end-expiratory transpulmonary

pressure but tends to flatten the relatively intact diaphragm.^{89,90} Reversal of pharmacologic paralysis restores the spontaneous breathing pattern that favors peri-diaphragmatic expansion.^{21,22} In recumbent positions, PEEP helps preserve and restore ventilation in dependent lung zones, especially when the lung has been acutely injured.

Reversal of Airway Occlusion

Standard measures to “uncork” a mucus-occluded airway include directed suctioning, physiotherapy, and therapeutic bronchoscopy.^{35,50,68,74} Infraglottic instrumentation of the airway may help direct aspiration of proximal airway secretions and promote cough. The ease and relative comfort of airway suctioning clearly depend on intubation status. For those not intubated, lubricated nasal “trumpets” can guide glottic entry but may also constrict the nasal passage, obligating the use of smaller, less effective suction catheters. Conventional closed-circuit aspiration catheters may have limited effectiveness in left lower lobe atelectasis, especially when they fail to provoke effective coughing, as during neuromuscular paralysis. Moreover, without peripheral air to push secretions mouthward during expiratory efforts, even forceful coughing may fail to dislodge a mucous plug lodged at the entrance of an occluded lobe. Under such circumstances, adequate airway humidification and the use of directed (coudé-style) catheters are logical to use as initial measures.

Physiotherapy is a time-honored technique whose documented value has been questioned but whose effectiveness seems to have backing from extensive clinical experience.⁹¹⁻⁹³ Deep breathing, assisted coughing, postural drainage, and chest percussion are sometimes contraindicated (eg, spinal injury) or difficult to apply in the acute care setting. Timed manual abdominal thrusts or device-actuated coughing by slow insufflation with positive airway pressure coupled to rapid exsufflation by suction (“in-exsufflation”) clearly help routine secretion management in high-risk patients.⁹³⁻⁹⁷ Unfortunately, these same measures often prove ineffective once atelectasis becomes firmly established. In recent practice, vibratory percussion of the chest wall and/or air column have been used increasingly (discussed later). Certain specialty beds provide external vibration as a convenient elective feature. Beds that prioritize variation of position (ie, kinetic therapy) may shift the body through a > 60° angle around the horizontal axis, and although there

seems to be little consensus regarding efficacy, some reports strongly support their use.^{98,99}

Even though their outcome benefit sparks debate,⁹³ the combination of deep breathing, coughing, airway-draining body positions, and chest percussion do help reverse ALA and prevent its redevelopment, provided that the patient can tolerate such manipulation.¹⁰⁰ For example, when mucus is the primary reason for lobar collapse, one vigorous chest physiotherapy treatment reverses ALA as effectively as fiberoptic bronchoscopy.⁵⁰ Because airways serving the collapsed region are not filled by radiodense mucus, neither respiratory therapy measures nor fiber-optic bronchoscopy are immediately productive when a branching air bronchogram penetrates the atelectatic lobe.^{50,101} With whole lung collapse, however, mucoid secretions invariably replace the air column, portending the effectiveness of directed suctioning. In such settings, therapeutic bronchoscopy is the logical next step when coughing efforts and catheter suctioning fail to restore airway patency. For patients with a very narrow main airway (eg, as in some young children) or unusually adherent secretions that cannot be extracted through the small channel of the fiber-optic instrument, ventilating (rigid) bronchoscopy under deeper anesthesia may be required.⁷³ Well-established atelectasis often proves refractory to quick re-expansion, even after the blocked passage has been cleared. Aggressive instrumentation that selectively occludes the involved airway and sustains a high regional airway inflation pressure that meets or exceeds those associated with total lung capacity may reverse atelectasis, with variable impact on gas exchange.¹⁰²⁻¹⁰⁷ The indications that mandate such aggressive measures and the relative safety of such maneuvers have not been convincingly delineated or verified.

Adjunctive Measures

The pharmacotherapy of ALA consists of airstream hydration, bronchodilation, mucus lubrication, reduction of inflammation, and chemical disruption of the mucus plug itself.⁶⁸ With these ends in mind, antibiotics, corticosteroids, guaifenesin, and mucolytic agents have been extensively used.^{58,108-111} Although there is limited evidence to support inhalation of hypertonic saline, acetylcysteine, and dornase aerosols, direct instillation of these agents has reportedly met with greater success. Such drugs and delivery methods show benefit in chronic clinical settings such as cystic fibrosis but have been variably effective for ALA.¹⁰⁹

Prophylaxis

Effective prophylactic physical measures against ALA are those that address its pathogenic mechanisms. Upright positioning, patient mobilization, and effective secretion clearance have been standards of practice in the postoperative setting for many years.⁹⁵ Maintenance of transpulmonary pressure and lung volume by applying PEEP and CPAP makes better sense than short-lived incentive spirometry,¹¹²⁻¹¹⁵ especially when end-expiratory airway pressure can be sustained. For those not intubated, even intermittent CPAP by mask is reportedly superior to incentive spirometry,^{18,116} as much for its assured utilization without patient cooperation as for its direct assistance in improving lung mechanics. Adding an inspiratory pressure boost (Bi-Pap) is logical¹¹⁷ but cannot be considered of proven added benefit over CPAP for this purpose.^{35,95} Nasal masks and helmets offer well tolerated, noninvasive options for elevating airway pressure.¹¹⁸⁻¹²⁰

Regarding incentive spirometry, use of a one-way valve to tidally “breath stack” volume toward total lung capacity during spontaneous breathing may increase the depth and duration of tractive forces in semi-cooperative patients whose primary problem is weakness.¹²¹ Assuring adequate hydration, prevention of aspiration, and early treatment of respiratory infections have intuitive value in reducing the secretion burden. During conventional mechanical ventilation, inspiratory flow profile, inspiratory time fraction, and tidal volume alter the ratio of peak expiratory to peak inspiratory flows and thereby may help (higher ratio) or hinder (lower ratio) the mouthward migration of small airway secretions.¹²² Promotion of coughing by in-exsufflation devices seems especially helpful in patients with neuromuscular debility.¹²³ High-risk individuals with low ventilating reserve (those with neuromuscular weakness, tracheostomy, or cystic fibrosis) may dislodge and mobilize their high secretion loads by percussive therapy applied to the chest wall or airway.^{86,109,124,125} High-frequency percussive ventilation, a proprietary ventilation technique, has been successfully applied for this purpose in diverse ICU settings.¹²⁶ Vest vibration can be applied during spontaneous breathing or superimposed on conventional ventilation but is not well tolerated by many hospitalized patients or effectively applied in most patients with major chest deformation, burns, or rib fractures. Chest pummeling during postural drainage (“stir-up” regimens), entrenched components

of physiotherapy, and prophylaxis^{127,128} lack convincing documentation of effectiveness.^{95,97,109,129,130}

Conclusions

ALA is a predictable, readily diagnosed, and pathogenic companion to acute illness, postoperative care, and chronic debility. Consequences from lobar collapse may be minor or serious, depending on extent, mechanism, patient vulnerability, abruptness of onset, effectiveness of hypoxic vasoconstriction, and compensatory reserves. Measures taken to reduce secretion burden, assure adequate secretion clearance, maintain upright positioning, reverse lung compression, and sustain lung expansion accord with a logical physiologic rationale.

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