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Concepts of atelectasis

Donald Malcolm McMillan
University of Nebraska Medical Center

CONCEPTS OF ATELECTASIS

Donald Malcolm McMillan

advisor: John R. Jones, M.D.

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Atelectasis is a loss of lung volume due to a state of airlessness and collapse of alveoli. In the normal situation, a balance of forces exists which maintains the patency of the alveoli. Subatmospheric intrapleural pressure acts to keep the alveoli open, whereas the elastic recoil and active contraction of the pulmonary tissue along with the forces of surface tension generated at the interface between the alveolar air and the thin layer of fluid act to collapse the alveolus. Various physical and chemical alterations occur that precipitate the development of atelectasis.

Atelectasis may develop at any time; however, it is a common postoperative phenomenon and is estimated to comprise 90% of all postoperative pulmonary complications.[1] Atelectasis should certainly be suspected in any postoperative patient with elevation of pulse, temperature or respirations in the first twenty-four hours. At this time, it is usually the result of inadequate respiratory excursion secondary to sedation or splinting.

The more carefully the presence of atelectasis is sought the more frequently it is found. Roentgenographic examination will show atelectatic areas in lungs not evident on physical examination. Pulmonary

function studies have demonstrated some lung collapse in approximately 80% of patients undergoing laparotomy that was not evident on physical or x-ray examination.

Physiologic Atelectasis

Physiologic atelectasis has been described in the newborn infant prior to the time of pulmonary respiration. Unless depressed for some reason such as medication, hypoxia, acidosis, or hypothermia, an unknown mechanism causes spontaneous respiration. In the presence of a patent airway, this results in the inflation of previously collapsed or "atelectatic" lungs. In this paper, we are primarily interested in the atelectasis occuring in previously functioning alveolar tissue.

Obstructive Atelectasis

A very common mechanism which acts to produce collapse of small areas of lung parenchyma is the absorption of air in groups of lobules after the bronchioles leading into them have been obstructed. This is known as obstructive atelectasis and is usually the result of mucus plugs, swollen bronchial mucosa or various foreign bodies. Postoperatively, there is frequent retention of secretions in segmental bronchi.

The movements of cilia usually act to keep these bronchi clear; however, ciliary action can be impaired by anesthetic agents, increased viscosity due to dehydration or noxious substances such as bacterial toxins.[2] When the activity of cilia becomes ineffective, coughing is initiated to produce removal of obstructing material by rapid air flow in the bronchioles. The effectiveness of the coughing mechanism can be reduced by postoperative pain or sedation and preoperative debility or lung disease. Thus vital capacity is reduced to a level where secretions are not moved.

The existance of peripheral collateral pathways permitting free passage of air to and from the alveoli with an obstructed bronchus prevents obstructive atelectasis from occurring more frequently than it does.

This movement of air, known as collateral ventilation, takes place through adjacent segments, presumedly through defects in alveolar septa (pores of Kohn), since no movement of gas occurs between adjacent lobes.[2] This pathway permits air to get behind secretions aiding in removal by coughing and explains the resolution of atelectasis after the removal of a minimal amount of secretion.

The development of atelectasis after obstruction is dependent upon the functional circulation remaining in the atelectatic area. Oxygen, a very absorbable gas, is quickly removed by the blood stream with the potential development of atelectasis in fifteen to thirty minutes after obstruction of airways in areas of normal pulmonary capillary blood flow.[3] With the presence of a poorly absorbed gas or impaired pulmonary circulation, the alveoli may remain patent for many hours after obstruction.

Bronchial obstructions have been considered primarily as intramural in origin. However, extramural obstruction may be caused by a lymph node. Mural obstruction due to a bronchial adenoma, stenotic bronchus secondary to tuberculosis or edema associated with infection is also an important obstruction mechanism.[3]

Some investigators feel that the tachycardia, tachypnea and fever classically associated with obstructive atelectasis are the result of an infectious process taking place in the distal respiratory tree since the phenomena can be suppressed with antibiotic therapy or removal of a mucus plug.[4]

Compressive Atelectasis

Another mechanism frequently causing atelectasis is compression of the lung by external forces such as fluid, air, or solid masses. This is sometimes associated with obesity or abdominal distention when the arch of the diaphragm is forced upward compressing the bases of the lung. Neoplasms of the lung and mediastinum may exert direct pressure on lung parenchyma or cause obstruction of bronchi with subsequent atelectasis. More current phenomena also acting to cause lung compression are underwater snorkeling, chest binding, or inflation of a pressure suit. In a combination of several factors, forward acceleration may also bring compressive forces on the lung.[5] The sudden development of a pneumothorax may cause precipitous collapse of a lung.

Gas volume is reduced by squeezing it out of airways to a point where some of the alveoli collapse. This precipitates effective shunting of capillary blood and contributes to arterial unsaturation.[5] The loss of functional pulmonary tissue due to atelectasis usually means there is a reduction in pulmonary reserve and the patient experiences no or few respiratory symptoms in a resting state. A stressful situation with increased

demand on arterial oxygen saturation would reveal the loss of functional pulmonary reserve and precipitate respiratory symptoms.

Atelectasis Secondary to Constant Tidal Volume Respiration

The significance of breathing patterns and atelectasis was first demonstrated by Mead[6] when he used dogs to show that there was a progressive decrease in pulmonary compliance with quiet breathing. This process was reversible following passive hyperventilation. The post mortem appearance of the lungs suggested that closure of air spaces was related to reduction of compliance. Measurements of total and ventilatory lung gas volumes indicated that the closed spaces were essentially atelectatic.

Bendixen et al[7] in a series of eighteen patients undergoing general anesthesia with controlled ventilation demonstrated impaired oxygenation and decreased lung compliance if periodic deep breaths were not initiated. According to their calculations, there were a drop in lung compliance of 15% and a fall in oxygen tension of 22%. At the end of the controlled breathing period, normal lung compliance and oxygenation was achieved by successive hyperinflations of the lungs.[7] The relationship of oxygen tension and tidal volume permitted adequate oxygen tension to be maintained by hyperinflation but shallow respirations resulted in impaired oxygenation and atelectasis. Since the above findings were accomplished with a mixture of nitrous

oxide and oxygen, it is assumed that a postoperative patient may become hypoxic with normal minute ventilation if pain medication prevents periodic deep breaths.

In a later study, Bendixen et al[8] used a series of twenty-five patients under oxygen and ether. Allowing them to breathe spontaneously for approximately thirty minutes, it was found the average arterial oxygen tension was 402 mm. of Hg. After three to five minutes of controlled ventilation with large tidal volumes, the arterial oxygen tension rose to an average of 553 mm. of Hg. They concluded that spontaneous ventilation in anesthetized patients should be supplemented with periodic deep breaths even though this ventilatory rate is adequate for carbon dioxide elimination.

Although low surface tension is necessary in the presence of low distending pressures, apparently the lung cannot maintain a low surface tension for prolonged periods of time. Therefore, if the surface tension is high in areas of abnormal surface, an increase in distending pressure achieved by a deep breath may replenish the surface. This mechanism likely resolves the atelectasis that occurs with normal shallow breathing.[9]

Some investigators have postulated the importance of nitrogen in the mechanism just described.[10] The poor diffusibility of nitrogen as compared to oxygen would

would leave the alveoli partially distended. Thus nitrogen prevents total collapse for a period of time until a deep breath restores the normal pressures in the alveoli.

Atelectasis Secondary to Alteration of Alveolar Surface Tension

After there has been a reduction of lung volume by either passive or active means, surface active forces become significant in creating further atelectasis. The estimated seventy square meters of internal surface area in the adult indicates somewhat the dimension of these forces.[11] Also the mechanical advantage of these forces is enhanced by the anatomical arrangement of the air pathways in the lung.

von Neergaard[12] just demonstrated surface tension was much of the retractive force in the lung by reexpanding a lung emptied of air with saline. This showed that tissue elasticity was only a fraction of the total lung elasticity, the remainder being due to surface forces.

A fluid film has a contractile tendency like a stretched membrane. According to La Place,[13] the effect of surface forces increases as the surface curvature increases. His expression P=2 T/r (P=pressure, T= surface tension, r=radius of curvature) indicates the pressure is directly proportional to the surface tension and inversely proportional to the radius of surface curvature. This would imply that air in a small alveolus would be lost to a larger alveolus if there is free com-

munication between them and no other properties are acting.[14]

To maintain an alveolus in an expanded condition, two of the forces that must be overcome are the elastic network and the film lining the alveolar surface.

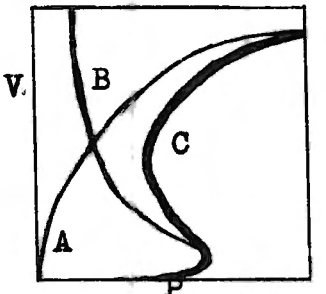


fig.1

Pressure-volume characteristics of a spherical elastic network (A) and a liquid film surface lining this sphere (B). Combined characteristics illustrating the theoretical behavior of an alveolus are shown by line (C). (From Rahn, H.; Fachi, L.E.: Gaseous Environment and Atelectasis, Federation Proceedings 22:1035-1041, Jul Aug 1963

There is a region of instability where the slope reverses. Excised lung shows less elastic recoil when filled with physiologic saline than with gas. Thus the tissue gas interface or surface tension is the apparent force tending to collapse lungs.[12] Air pressure in the alveolus must be sufficient to counteract the surface forces since below a certain critical size surface tension would be sufficient to collapse the alveolus.[15] The elastic properties also enter into alveolar compliance. Therefore, a reasonable assumption is that tissue forces predominate while the alvoli are distended. However, at small volumes surface forces are more significant.

Since there are no structural components of support to prevent collapse, the alveolar lining layer thus becomes very important. It has subsequently been demonstrated by several investigators through observation of bubbles[16] and saline extracts[17] from lungs that the alveoli are lined with a substance that reduces surface tension. This substance, called surfactant, works as an antiatelectasis factor.

Macklin[18] was the first to suggest the alveolar lining layer was a mucoprotein film. Further investigation has shown that the material is a lipoprotein, primarily a phospholipid. Various substances have been tested to evaluate their effect on this material but only those that are surface active themselves seem to be significant. Saline extracts of normal lungs loose ability to lower surface tension when treated with non-ionic detergents, phospholipase C, serum, fibrinogen, and heating to forty-four degrees Centigrade.[19] The effects of these agents in the normal lung would thus be highly contributory to the formation of atelectasis and it has thus been proposed that certain anesthetic agents may predispose the lung to the formation of postoperative atelectasis.[20]

The lack of surfactant has been suggested as the cause of the severe atelectasis that is present in

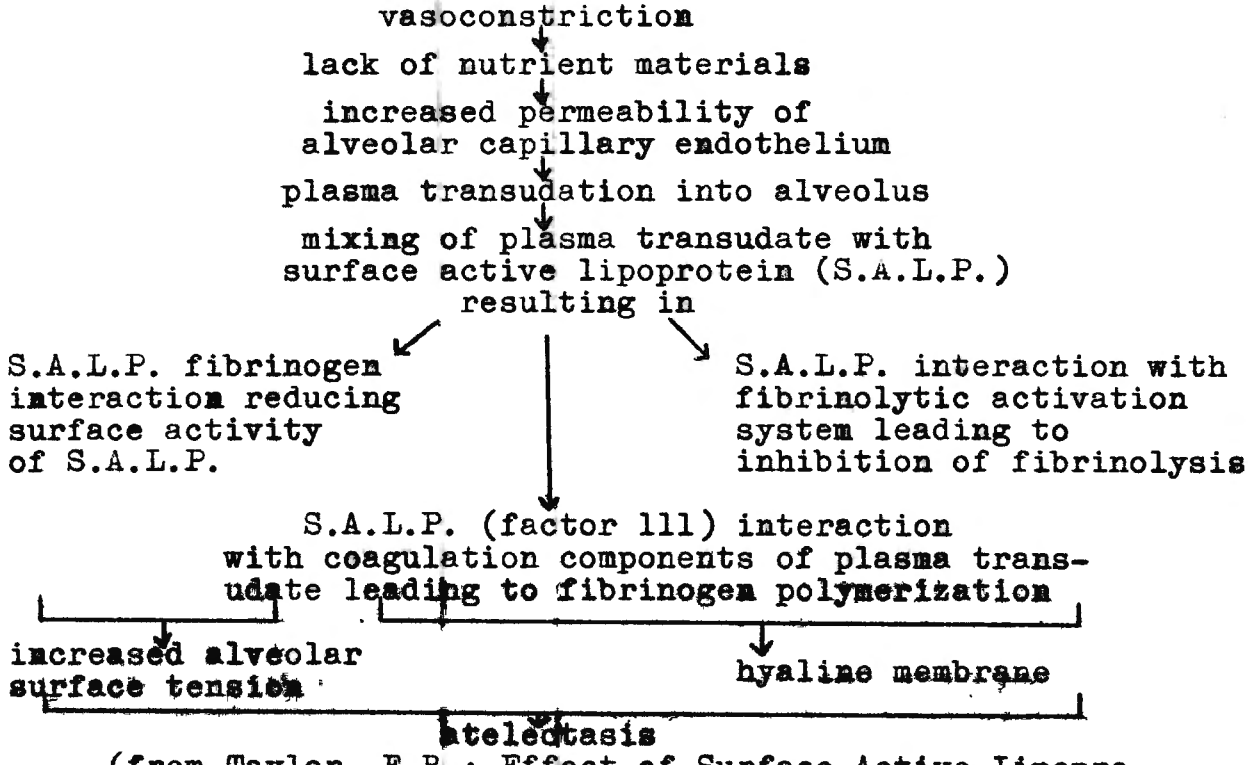
hyaline membrane disease because there is a loss of normal surface activity in extracts from lungs of infants with this condition.[21] Due to the inability to stabilize the patency of small alveoli, the newborn with a predominance of these alveoli will subsequently develop wide-spread atelectasis and the airless "liver like" condition of these lungs at autopsy.[17] The pressure volume characteristics of these lungs is markedly changed as they are not distendable except under high pressure and trap little air when deflated.[14]

The reason for this apparent absence of alveolar lining layer is not yet determined. Pattle,[16] after experimenting on mice, rats and guinea pigs, suggested that this film appears in the fetus when cuboidal epithelium attenuates and alveoli appear. This is consistent with the observation that atelectasis is prominent in infants less than 1000 grams at birth, thus suggesting a factor of immaturity. Membranes are more common in heavier infants. Thus if membrane formation is dependent on circulatory fibrin, the development of a capillary network is necessary. Because membranes are not present in infants that have breathed less than two hours, it is assumed membrane formation is secondary and not primary with distal atelectasis.[14]

These phenomena can and do occur in larger infants

nearer term, also the corresponding histological change occurs in man prior to twenty-four weeks of fetal life.[16] This prevents the lack of lining layer to be ascribed solely to fetal developmental age.[14] Toxicity or fetal insult have also been implicated because of a statistically significant occurrence of maternal diabetes or hemorrhage.[21] Such an insult could denature or prevent the formation of the lining layer.

Taylor[22] has observed that the development of hyaline membrane disease apparently involves the transudation of plasma into the alveoli with the subsequent polymerization of fibrinogen to fibrin. The fibrinolytic enzyme system acts to hydrolyze this membrane and there is a loss of surface activity. He proposes the following theory of pathogenesis as diagrammed.



(from Taylor, F.B.: Effect of Surface Active Lipoprotein on Clotting, Amer. Jour. of Med. 40:346 mar 1966.)

The development of atelectasis due to a change in surface activity in association with the alteration of certain physical or chemical conditions deserves some consideration. It has been demonstrated that prolonged sustained overinflation of the alveoli can interfere with the activity of surfactant. This is a possible mechanism in the development of respiratory insufficiency and atelectasis associated with the use of fixed volume piston type respirators.[23] It has also been shown that there is a loss of surface activity and increase of surface tension subsequent to diminution of pulmonary blood flow.[24,25] The reason for or significance of these phenomena is not clear yet. It has been stated that atelectasis may cause rather than result from a decrease in surfactant. This is assumed to be the probable relationship during short term compressive or obstructive atelectasis when there is inadequate perfusion of alveoli.[19]

Most important, however, has been the relationship of the infectious process and subsequent loss of surface activity.[24,26,27] This is evidently due to a destruction of surfactant or the proliferation of an antagonist. Whether this is an action of some factor from the infecting organism or the result of host response has not yet been determined. It has been suggested that a sur-

factant antagonist does exist in the lung tissue itself. However, it seems more likely that the proteolytic enzymes found in various infecting organisms are more important. Elevated temperatures have caused changes in surface activity also but these temperatures were higher than found in infectious processes, thus probably not part of the mechanism of surfactant alteration.[19] It is definitely felt that a substance exists in pneumonic lungs which causes alteration in surfactant and frequently results in atelectasis.[26]

The following diagram illustrates the relative effect the loss of surfactant has on the pressure volume behavior of alveoli in comparison to atelectasis occurring by compression absorption secondary to obstruction

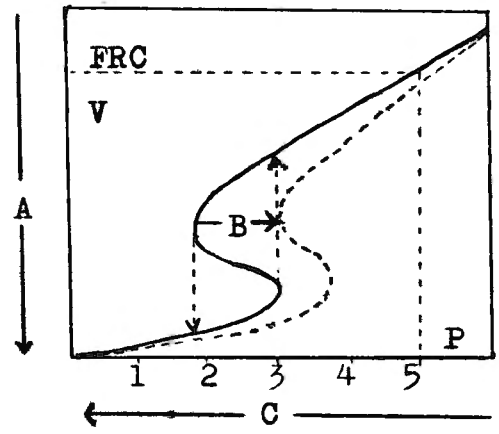


fig.2

Hypothetical pressure volume behavior of an alveolus (line C of fig. 1). Its normal stable volume is located at its FRC supported by a pressure difference of 5 cm. H_2O. Heavy arrow, A, indicates the direction of changes which accompany absorption atelectasis; arrow C, the forces which lead to compression atelectasis, while arrow B indicates the consequences of surfactant loss which also leads to atelectasis.

(from Taylor, F.B.: Effect of Surface Active Lipoprotein on Clotting, Amer. Jour. of Med. 40:1037 mar 1966.)

Congestive Atelectasis

Congestive atelectasis is an acute form of respiratory insufficiency secondary to non-obstructive collapse of pulmonary alveoli with intense interstitial capillary congestion associated with parenteral fluid therapy. Even though there is a lack of specificity in the clinical disease state, the pathological condition is constant and is usually referred to as "intense congestion of the lung".

Gross features of the lungs include blue-black discoloration bilaterally with increased weight of lung tissue. Microscopically, there is the appearance of gross hemorrhage with engorgement of uniformly dilated venules, arterioles and capillaries. There is loss of normal alveolar arrangement and epithelial cells are closely packed, obliterating alveolar air spaces.

Acute respiratory distress in this condition is apparently due to disturbance of the normal transport zone between blood and air. Two questions not yet satisfactorily resolved are how does this condition develop and what physiologic disturbances are present preventing adequate response to treatment with subsequent high mortality rates.

The clinical manifestations of this condition include intense dyspnea, cyanosis, tachypnea, and progressive

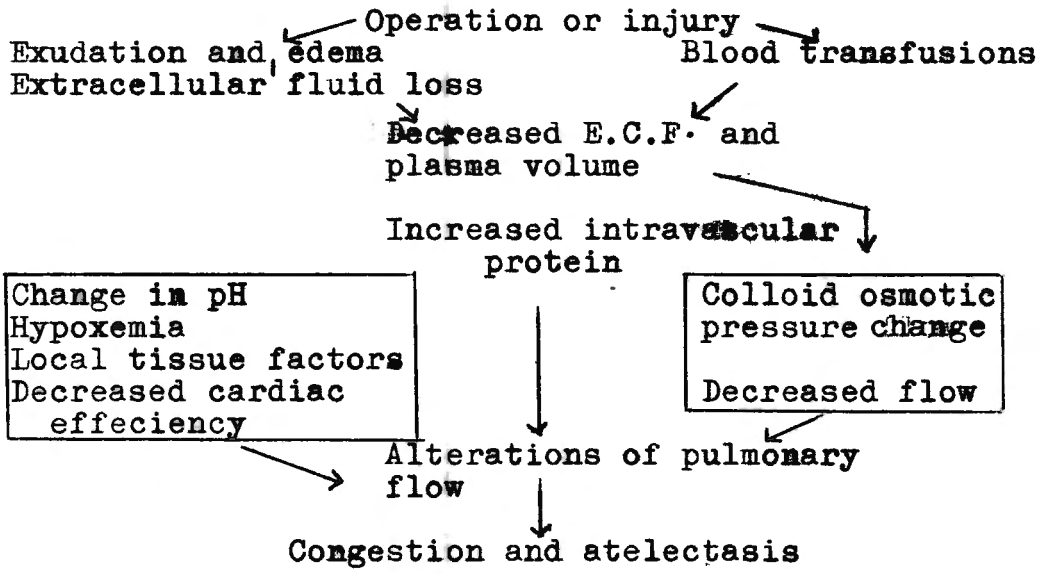
hypotension. The clinical state does not lend itself to investigative technique thus preventing adequate assessment of the chain of events that are active; however, several specific areas have been investigated.

Considering the significance of a change in environmental barometric pressure, it was found that congestive atelectasis may develop without an obstructive component. It was also shown that a small increase in the intra-pulmonic pressure may obviate the conjectured "critical closing pressure" of the alveoli. At the present time, there is not enough evidence to implicate a change in surface tension in a contributory or sustaining component of congestive atelectasis. Some authors[28] suggest that pulmonary edema leads to regional impairment of pulmonary surface activity and subsequent alveolar closure.

Humoral factors have not been well evaluated yet but it has been shown that histamine and acetylchloine have an effect on pulmonary vasculature which is considerably greater than that of anoxia. Seratonin, which is released by intravascular clotting, causes a marked increase in pulmonary construction. Most significant is the effect of hypoxia and acidosis on lung capillaries and their capacity to dilate and become congested.[29]

Congestive atelectasis is a complication of parenteral fluid therapy but the reason has not been explained. With

all the variables considered, it is concluded low blood pressure is a sign, not a cause, of the problem. The following scheme illustrates the potential factors.



(from Berry, R.E.L.; Sanislow, C.A.: Clinical Manifestations and Treatment of Congestive Atelectasis, Archives of Surgery 87:162 jul 1963.)

Conditions that should cause anticipation of congestive atelectasis in particular are a significant inflammatory component, chronic anemia, metabolic deficiency, severe trauma with hypotension, period of anesthesia over six hours, and excessive obesity.

In diagnosing this condition, it is emphasized that what is absent in considering signs and symptoms is as important as what is present. This is especially true when differentiating from obstructive atelectasis, acute hypovolemic shock, and acute left heart failure. Clinical

features of congestive atelectasis include:

1. Cyanosis: deep and refractory to usual O_2 therapy.
2. Dyspnea: severe with limitation of expiratory phase and tachypnea, early and severe.
3. Hypotension: progressive, severe, unresponsive to and aggravated by parenteral fluid especially blood. There is peripheral cold shock.
4. X-rays: little early change; later, patchy involvement.
5. Auscultation: few early findings.
6. Distention of neck veins: none early.
7. Bronchoscopy, phlebotomy, and digitalization: no benefit.

Several signs occurring during an operation may suggest the development of congestive atelectasis. These include the apparent necessity to administer greater amounts of blood than that lost for maintenance of circulatory stability, adequate relaxation accompanied by poor color, increased pulse rate and undesirable fall in blood pressure, narrowing of pulse pressure.

Treatment of this condition is often ineffective, probably because of the advanced condition when recognized with severe acidosis and oxygen debt. This results in an inability to reverse the congestive process. The only effective measures are prompt cessation of parenteral fluid administration and 100% oxygen administered under positive pressures on inhalation and exhalation. Fluids are not stopped because of the hypotension. These measures would

prevent the collapse of the alveoli by maintaining intra-alveolar pressure at a level that is not overcome by the external pressure of increased amounts of blood present.[30]

Contraction Atelectasis

In an effort to understand the problem of nonobstructive alveolar collapse, one explanation that has received considerable attention recently is that of active contraction by alveolar smooth muscle. Contraction atelectasis was first described as following a segmental distribution in the lung with the assumption that such pulmonary spasm was likely related to neurovegative activity. Several investigators consider this to be due to the presence of "physiological pulmonary tonus" which is controlled by the autonomic nervous system; thus active pulmonary contraction is the result of an increase in this tonus.[31]

It is generally agreed that increased neuromuscular reflex activity in the lung can result in bronchospasm and atelectatic changes. There is, however, disagreement regarding the involvement of peripheral lung units in this phenomenon. Since it has been generally accepted, until recently, that no muscle fibers were present in alveolar tissue, investigators assumed that no spasm could occur.[32]

Early investigators and proponents of the concept that muscle fibers are present and active in lung parenchyme include Baltesberger, Bronkhorst and Dijkstral. The significance of early findings has been debated.[31] In an effort to establish the existence of alveolar musculature, recent investigators have attempted various

approaches to the problem which, if solved, would be clinically quite significant.

Corssen[32] approached the problem by using lung tissue taken in situ and subjecting these specimens to various staining techniques in anticipation that smooth muscle fibers could be visualized. Biopsy specimens were taken from various disorders, including bronchogenic carcenoma, emphysema, chronic bronchitis, and pulmonary hypertension. He observed that smooth muscle elements were detectable in all specimens including both normal and diseased; however, there seemed to be less musculature in normal tissue. Single and grouped smooth muscle cells were identified in the alveolar ducts and walls along with bundles of interstitial muscle fibers in alveolar septa. In lungs that were emphysematous, interstitial musculature seemed to predominate. However in the lungs of those with pulmonary hypertension, the smooth muscle fibers were observed more frequently in the alveolar ducts and walls.

Corssen considered his findings as sufficient evidence of alveolar musculature capable of active contraction under control of the autonomic nervous system. He also speculated that the increased amounts of musculature found in people with pulmonary hypertension may predispose them to "contraction" atelectasis.[31, 32]

Impressive evidence for the mechanism of active contraction of the lung has been assembled by several observers.

"In brief, it is to be noted that:

1. When the lung is directly observed by thoracoscopy, touching the surface of the lung produces dimpling with active retraction of the spot that had been stimulated. The dimple appears by closure of the bronchiole and of the peripheral acinus arising from it. The recoil and contraction of this healthy lung surface is thought to be neurogenic in origin because pretreatment of the surface with procaine prevents this contractile response.

2. Similarly, it was noted by thoracoscopic observation that a lung could suddenly diminish its volume without apparent cause, detach itself from the chest wall and, while floating freely in the chest cavity, would continue its respiratory excursions. Areas of function (breathing zones) were often observed within larger areas of collapsed lung. These phenomena were interpreted as indicative of regional areas where tonicity of lung tissue would alter with resulting changes in volume.

3. Direct measurements of the pressure and volume within pulmonary cavities often demonstrate increases or decreases which are independent of atmospheric or intrapleural pressures. Such changes seemingly result from active contractility with some change of tonus of the functioning lung parenchyma which surrounds the cavities.

4. It has been demonstrated that stimuli applied to abdominal viscera, cranial centers, the parietal or visceral pleura, or to the bronchus can result in a sharp reduction of the volume of the lung, while there may actually be bronchodilation. On numerous occasions during the initial induction of a therapeutic pneumothorax, it has been observed that as little as 150 cc of air intrapleurally could result in a rapid and complete contraction of the lung with intrapleural pressures reaching minus 40 cm. of water.

Similarly, during routine bronchography, instances have occurred in which immediate atelectasis of a lobe or of a lung appeared while there was

obvious patency of the bronchi in the roentgenograms. Re-expansion occurred just as rapidly although there was no expulsion of the Lipiodol and the bronchi remained open and filled with contrast medium. This rapid filling and emptying of the lung cannot be explained by passive resorption of air following fortuitous obstruction of a major bronchus because, when a major bronchus is occluded by a ligature, at least 8 hours elapses before sufficient air is absorbed to produce x-ray opacification. Yet, immediate contraction of the lung and retraction of the hemithorax is observed following ligation of the bronchus. This demonstration clearly separates the immediate reflex contraction from the later resorptive phenomena.

5. There is agreement that smooth muscle bundles are present in human lungs as far distal as the alveolar ducts. The apparent absence of muscle fibers in the alveolar walls does not prevent active contraction, as the "plasmatic alveolar cell" is an adequate functional substitute for muscle fibers.

6. In any case, when acetylcholine in greatly diluted quantities is applied to the lung surface, strong contractions occur with corresponding loss of lung volume. This demonstrates that, in spite of any controversy over the exact mechanisms involved, actual pulmonary contraction does occur."[15]

(from Burbank,B.; Cutler,S.S.; Sbar,S.: Non-obstructive Atelectasis, Jour. of Thoracic and Cardio-Vascular Surgery 41: 710-711 June 1961.)

It is postulated that atelectasis of reflex origin which is culminated by contraction of distal lung units is mediated through an afferent arc in the chest wall, visceral or parietal pleura, the abdominal viscera, or the bronchi. The afferent arc stimulated contraction of the distal lung units, causing the radii of the alveoli to decrease to the point where intra luminal air pressure is overcome by surface tension, tissue elasticity and contractility resulting in complete collapse. Consider-

ing the contractile properties of the lung units including bronchi and alveoli with the evidence of pulmonary reflexes, it is speculated that reverse peristalsis and vomiting of air may be significant in rapid deflation of lungs.[15]

Atelectasis Associated with Anesthesia and Pneumonia

The association of atelectasis and anesthesia has been stressed with a search for incriminating factors. Some investigators feel there is little relationship to the development of atelectasis postoperative and the anesthetic agent used. Although differences of opinion exist, it has been shown that regional or spinal anesthetics have the same incidence of postoperative respiratory complications as general anesthetics if given the same postoperative care.[1] One study was done to establish the hypothesis that thorough ventilation of lungs with gas of low solubility at the termination of anesthesia would protect against postoperative atelectasis; however, the findings did not verify this.[33]

The frequent development of atelectasis with pneumonia has been recognized by physicians for many years. Numerous theories have been proposed, although the mechanisms of this phenomenon have never been elucidated. Among the possibilities mentioned is a weakness in inspiratory power when it was noted children with diphtheria developed lower lobe atelectasis with diaphragmatic paralysis and upper lobe atelectasis with intercostal muscle paralysis.[34] Other mechanisms proposed include contraction of partially consolidated lungs, bronchial occlusion with exudate, shallow breathing with acute illness, loss

of lung resiliency from previous infection, spontaneous contraction of a bronchus, and acute parenchymal necrosis.[26] These factors, along with the aforementioned antagonism of surfactant, are certainly associated and perhaps collectively responsible for this condition. Further investigation will develop more understanding of atelectasis and aid in the treatment of this important pathologic condition.

Summary

For many years, physicians have been concerned with the problem of atelectasis, its causes, and treatment. Traditionally, atelectasis was categorized as physiologic, compressive or obstructive. More recently, it has been established atelectasis can result from constant tidal volume respiration or alteration of alveolar surface tension. Two new concepts still being evaluated are that of atelectasis secondary to intense interstitial pulmonary capillary congestion and atelectasis resulting from active contraction of lung parenchyma. This paper is a discussion of some current concepts regarding the pathophysiology of atelectasis.

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