Acute Non-Aeration of Lung: 
Pulmonary Edema versus Atelectasis

DAVID M. SPAIN, M.D., F.C.C.P.*
Brooklyn, New York

Probably no other diagnosis related to pulmonary disease has been abused as much as that of atelectasis. It is constantly being made where opacities are seen in roentgenograms of the chest in such situations as post-anesthetic states, barbiturate narcosis, chest wall injuries, prolonged rest in the supine position, poliomyelitis, bronchial asthma, pulmonary infections with retention of thick, tenacious mucus or exudate, and aspiration of foreign material or foreign bodies. Yet postmortem examination of the lungs from such conditions most often fails to reveal what is strictly defined as atelectasis. This discrepancy is probably due to misinterpretation of the actual alterations that usually transpire in the lungs in the aforementioned conditions. Atelectasis correctly defined refers to incomplete expansion of the pulmonary parenchyma at birth or to the collapse of varying portions of previously aerated lungs during later periods of life.¹ The usual mechanism which is thought to produce collapse of lung parenchyma is absorption of the alveolar air after the bronchus to a particular region has been obstructed. The other mechanism, compression of the lung, may be produced by alterations in the pleura, diaphragm, or chest wall. In the former mechanism, bronchial drainage is impaired, while in the latter it is not unless associated intrapulmonary disease is present. In microscopic sections, the walls of the alveoli, alveolar ducts, and respiratory bronchioles are closely opposed to form slit-like spaces with the walls often paralleling each other. The alveolar capillaries appear to be dilated, but it has been shown that less blood flows through an atelectatic lung than a normal expanded one. This report is concerned with experimental and clinical observations relative to the previously stated discrepancy between clinical and post-mortem findings.

Experimental Procedure and Observations:

Twelve healthy, mongrel dogs of approximately the same weight and age and kept under the same conditions were put under light sodium nembutal narcosis. Through a bronchoscope, the major bronchus to a left lower lobe in each dog was obstructed with a plug of absorbent cotton (Tampax) saturated with 1 per cent procaine hydrochloride. This was pushed down into the bronchus so that it would fit snugly. The dogs were then allowed to come out of the narcosis and were all alert within one hour of the procedure. Three dogs were sacrificed at 6, 12, 24, and 36 hour intervals following obstruction to the bronchi. The dogs were killed within

*Beth El Hospital, Brooklyn, New York. Formerly with Department of Laboratories and Research of Westchester County and the Division of Pathology of Grasslands Hospital, Valhalla, New York.

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the period of a few minutes by use of a carbon monoxide chamber. Immediately following sacrifice, the dogs were autopsied. The trachea was tied off in the neck before entering the chest and the lungs, trachea and heart were then removed in toto. The bronchus with the plug of cotton in situ was identified in all instances. After gross examination, numerous sections were taken from the position of lung distal to the obstructed bronchus, as well as from the non-obstructed lobes on the same and opposite sides. These sections were stained with hematoxylin and eosin and Masson's trichrome stain.

In the three animals sacrificed at six hours, no significant gross changes were found in the areas of lung obstructed as compared to the contra-lateral lungs and non-obstructed lobes on the same side. Microscopic examination revealed the only alteration to consist of congestion of the alveolar capillaries of the obstructed lobe. There was no reduction in size of the obstructed lobe. The alveoli still appeared to be normally distended. The other lobes were not congested (Figure 1).

Three animals sacrificed at 12 hours revealed essentially the same findings as the six hour animals with the exception that the congestion was more pronounced and in some of the alveoli there were accumulations of transudate. This was not present in the other areas of lung tissue.

In the 24 hour animals, there were slight reductions in the size of the lobes of lung distal to the obstructions. Practically all of the alveoli in the obstructed regions were filled with fluid. The alveolar walls were not collapsed against each other and the reduction in the size of the alveoli was slight (Figure 2). The other areas of the lungs did not reveal any significant congestion or edema.

In the 36 hour animals, the lungs distal to the obstructions were distinctly reduced in size, but not completely so, and on histologic section revealed alveoli reduced in size, marked congestion of the alveolar capillaries, and intra-alveolar fluid containing numerous polymorphonuclear leucocytes (Figure 3). The unobstructed lobes did not reveal anything unusual.

Thus the progression of events beginning with the six hour period: At first congestion of the alveolar capillaries; a few hours later pulmonary edema developed; subsequent to this there was reduction in distention of lung tissue which contained fluid with numerous polymorphonuclear leucocytes.

Post-mortem Observations On Human Lungs:

There were 20 cases of barbiturate poisoning, 30 of brain injuries with deaths occurring immediately following the injury to several days later, 20 fatal cases of poliomyelitis, 10 with chest injuries of varying types and degree, 4 deaths from bronchial asthma, a few cases of various types of bronchial obstruction, and several anesthetic and post-anesthetic deaths. All of these were examined carefully to determine the degree of pulmonary edema, congestion, atelectasis, and pneumonia that might be present. In all of the barbiturate cases, regardless of the time interval from the
Figure 1: Photomicrograph (H & E—250 x) of lung in dog sacrificed six hours after obstruction to bronchus. This shows alveolar capillary congestion. Figure 2: Photomicrograph (H & E—250 x) of lung in dog sacrificed 24 hours after bronchial obstruction. This shows edema and congestion. Figure 3: Photomicrograph (H & E—250 x) of lung in dog sacrificed 36 hours after bronchial obstruction. This shows some alveolar collapse, edema, and extensive infiltration with leucocytes.
ingestion of the barbiturates to the time of death, areas of colapsed lung were not found. Considerable congestion and varying degrees of focal and diffuse edema, and occasionally lobular pneumonia were present (Figure 4). In the traumatic cerebral cases, even when death was almost instantaneous, the congestion and edema of the lungs was diffuse and extensive. Atelectasia was not found in the cases surviving several days, despite the accumulation of thick and tenacious exudate in many of the bronchi and bronchioles. Areas of lung distal to these bronchi were markedly edematous and congested and in numerous instances revealed pneumonia. In the poliomyelitis cases, a somewhat different picture was present. In all of them, therapeutic drainage of the bronchial tree was maintained through suction, bronchoscopy, or a tracheotomy tube. The lungs varied in appearance. In some, there were areas of congestion and edema. In the posterior lower aspects of the lungs, areas of edema were often intermingled with small focal areas of atelectasis in which the alveolar walls were actually in contact with each other (Figure 5). This occurred regardless of the presence or absence of exudate in the bronchioles. In the chest injuries, the usual findings consisted of varying degrees of hemorrhage in the alveoli beneath the pleura immediately adjacent to the site of the injury with areas of edema adjacent to this (Figure 6). Pulmonary edema was also present remote from the site of injury. Atelectasis was not found. Two cases of bronchial obstruction were of particular interest. The first, a robust, adult Negro male with a compression injury of the chest was given oxygen by means of a mask. While receiving oxygen, his condition
rapidly grew worse and he expired after receiving oxygen for eight hours. At postmortem examination, it was found that the plastic disc in the oxygen mask had slipped off the valve and had become lodged in the carina completely obstructing the main bronchus to the right lung. In this lung, there was marked congestion and edema, but reduction in the size of the lobe was scant. In the other case, the obstruction was of a more chronic nature. This case was one of bronchial asthma in which the mucoid exudate in a bronchus to an upper lobe segment had become inspissated and obstruction developed over a period of time. Behind this obstruction to the bronchus, which was ulcerated at the region of the mucoid impaction, there was edema, mononuclear cell infiltration, and beginning organization of the exudate. There was no atelectasis. The constant findings in these groups of cases were varying degrees of congestion, edema, occasional small focal areas of atelectasis, and pneumonia. The focal atelectasis was limited almost entirely to the cases of poliomyelitis.

Discussion:

The findings of congestion, edema, and some reduction in the size of lung with an infiltration of inflammatory cells in the dogs with obstructed bronchi would indicate that in many instances shadows appearing in the chest roentgenograms are at first primarily caused by congestion and edema. The reduction in the size of the lungs and the inflammatory

**FIGURE 6**

*Figure 6:* Photomicrograph (H & E—250 x) of lung in case of blunt trauma to chest cage. This shows edema and intra-alveolar hemorrhage.

**FIGURE 7**

*Figure 7:* Photomicrograph (H & E—250 x) of lung in case of mucoid impaction of the bronchus in bronchial asthma. This shows edema, leucocytic infiltration, and some fibrosis.
acutely appear somewhat later. Within 36 hours it was necessary to consider the process as pneumonia. Drinker in his experimental observations states that the development of the edema is due to anoxia. The alveolar capillaries depend for their oxygen supply upon the alveolar air. If this is shut off or reduced, the alveolar capillaries are deprived of the oxygen and the capillary endothelium becomes more permeable. This permits fluid and cells to accumulate in the alveolar spaces. Another explanation offered for the development of edema following bronchial obstruction is a mechanical one. It is claimed with absorption of air distal to the obstruction a negative pressure is exerted on the alveolar capillaries with the resultant sucking of fluid from the capillaries into the alveolar spaces. It would seem from most experimental and clinical evidence that the former explanation is the most important and valid one. In some other experimental studies, obstruction of the larger bronchi has produced real atelectasis without edema. However, in these studies, the bronchi were obstructed through an open chest and after the lung was collapsed from the pneumothorax created by the procedure. This would explain the differences in the findings of this study as compared to that of others.

Analysis of the postmortem findings in the human lungs is consistent with that observed in the dogs. In addition to the experimental procedure described in this report, others have produced pulmonary edema in dogs with acute peripheral blood loss, with blunt injuries to the chest wall, with the maintainance of dogs in a state of narcosis and in the supine position. Increased intracranial pressure in dogs also results in pulmonary edema. In all of these animals, the predominant findings were congestion and edema and only insignificant degrees of atelectasis. The case of bronchial asthma with a bronchus obstructed by laminated inspissated mucoid exudate is similar to other cases reported by Shaw. In this situation, the findings undoubtedly represent the earlier stage of what McDonald et al. have described as obstructive pneumonitis of neoplastic origin. These authors point out the error of referring to these changes as atelectasis. In an extensive personal experience with neoplastic obstruction of the bronchi, the experience is similar to that reported by McDonald. In most of the aforementioned clinical conditions there are multiple factors present which facilitate the development of the described changes. For instance, in cases of trauma to the brain there are neurogenic and neuromuscular factors involved in the production of pulmonary edema, as well as interference with pharyngeal, laryngeal, and cough reflexes. Thus a combination of events which produce edema and interference with the removal of this accumulated fluid is present. Patients in these various categories are usually in the supine position so there would be the development of congestion and edema in the dependent portions of the lungs. The same multiple factors are present in barbiturate narcosis, post-anesthetic conditions and poliomyelitis.

The occasional cases of acute massive collapse of the lung which occur usually in anesthetic and post-anesthetic states do not fit into the above categories. In this situation the lungs become collapsed within a matter
of a few minutes and real atelectasis without significant edema is present. Among the cases studied, there was one which belonged in this group. This was a young female who was given nitrous oxide anesthesia during the final stages of labor. After the anesthesia was given for a period of 20 minutes, it was noted that the patient was dead. Postmortem examination revealed massive collapse of both lungs with numerous loose mucus plugs diffusely distributed throughout the bronchial tree. Microscopically, atelectasis was present. The alveolar and bronchiolar walls were collapsed against each other. There was no edema. There have been numerous attempts to explain this development on a rational basis. None has been completely satisfactory. A logical and ingenious explanation has recently been offered by Viswanathan. He contends that the mucus secretions in the bronchioles instead of producing complete obstruction act like ball-valves allowing air to get out from the lungs during expiration and preventing air from entering during inspiration. This may cause collapse of the lung within a few minutes even before edema can develop. The ball-valve action is possible because the bronchi are not of the same caliber throughout their length. The mechanism is accelerated by the tendency of the lung to shrink owing to its elasticity. Absorption of air behind a completely blocked bronchus obviously cannot explain the sudden collapse since this would take at least several hours. In all of these conditions, one of the most important defense mechanisms necessary to maintain adequate aeration of the lungs, that is, collateral ventilation, is interfered with. Maintenance of adequate collateral ventilation is dependent upon a proper functioning neuromuscular apparatus, as well as patency of the smaller bronchioles. In both the experimental studies and in the human cases, congestion and edema developed very early from multiple causation. Any subsequent reduction in the size of the lung would then be superimposed on this pre-existing edema and congestion. The sum total of the final picture, depending on the duration and circumstances involved, is a combination of congestion, edema fluid, inflammatory cell infiltration, and varying degrees of reduction in size of the lungs due to the absorption of air. Without antibiotic therapy, this is the ideal background for the development of pneumonia. Simple collapse of the lungs, as in compression atelectasis following artificial pneumothorax in which none of the above factors are operating and in particular in which the bronchial drainage is not impaired does not predispose towards infection. It is important to note that despite the terminology one wishes to choose, whether this be atelectasis, wet atelectasis, collapse of the lung, edema, or atelectatic pneumonia, what the mechanism and pathogenesis of the lesion is and what the ultimate consequences might be. The use of the term acute non-aeration of lung is suggested to describe these x-ray picture shadows because it is non-specific and yet recognizes the fundamental functional alteration that is present. In any individual situation of this sort it is difficult to be certain of the proportions of congestion, edema, pneumonia, and actual collapse present on clinical and roentgenographic grounds.
SUMMARY

1) Sudden complete obstruction of major bronchi in dogs with intact chests resulted in a series of events over a 36 hour period. Pulmonary congestion, edema, partial collapse, and infiltration with leucocytes (pneumonia) developed in the sequence listed.

2) Post-mortem observations on the lungs from such conditions as bronchial obstruction, barbiturate narcosis, chest injuries, poliomyelitis, and post-anesthetic states revealed congestion, edema, and pneumonia. The degree of lung collapse, if present, was usually variable and insignificant.

3) The term acute non-aeration of the lung is suggested to replace the term atelectasis in those situations where it is used erroneously.

RESUMEN

1) Como consecuencia de la obstrucción repentina de los bronquios principales de perros con tórax intacto, ocurrieron algunos cambios dentro de un período de 36 horas. Se presentaron: congestión pulmonar, edema, colapso parcial e infiltración con leucocitos (neumonia) en el orden citado.

2) Los exámenes de pulmones a la autopsia cuando los sujetos habían presentado afecciones tales como obstrucción bronquial, narcosis con barbitúricos, lesiones del tórax, poliomielitis, y estados postanestésicos, demostraron la presencia de congestión, edema, y neumonia. El grado del colapso pulmonar si existía, era habitualmente variable e insignificante.

3) Se sugiere el término “no aereación aguda del pulmón,” para substituir el de atelectasia en esas situaciones, en las que ha sido erróneamente aplicado.

RESUME

1) Une obstruction soudaine et complète de la bronche souche chez le chien, sans lésion pulmonaire, survint dans une période de 36 heures à la suite de manifestations variées. On constata congestion pulmonaire, oedème, colapso parcial, et infiltration leucocytaire (pneumonie).

2) L'examen post-mortem des poumons atteints d'obstruction bronchique, d'anesthésie barbiturique, de traumatismes thoraciques, de poliomyélite et dans la phase post-anesthésique, mit en évidence congestion, oedème et pneumonie. L'importance du colapso pulmonaire, si toutefois il existe, était généralement variable et insignifiant.

3) L'auteur propose que l'expression d'atélectasie soit remplacée dans les cas où elle est utilisée d'une façon erronée, par celle de “inaération aigue du poumon”.

REFERENCES