Postoperative Pulmonary Atelectasis*

ROSE K. L. WONG**
Portland, Oregon

The astounding number of surgical operations performed by physicians today has resulted in a greater awareness of atelectasis, a common postoperative pulmonary complication. Although easily recognized and prevented, atelectasis is frequently overlooked, resulting in serious pulmonary impairment and occasionally surgical mortality.

The word, atelectasis, is derived from the Greek, "atelect-asis," which denotes incomplete expansion. Although originally used to describe the non-expanded lung observed in stillborn babies, it is now more specifically defined as an airless state of the lung parenchyma resulting from an obstruction of the tributaries serving the lung and a subsequent absorption of the entrapped air by the circulating blood. That this condition occurs following surgery was first observed by Pasteur in 1910. In 1925, Lee and Jackson first demonstrated by bronchoscopy a complete bronchial obstruction by thick, tenacious secretions in a postoperative atelectatic patient.

How frequently postoperative atelectasis occurs has been found to be dependent upon factors such as: pre- and postoperative therapy, type of surgery, and age, sex, and health of the patient. It is more often encountered after upper abdominal or time-consuming operations, in patients who smoke, have respiratory infections, or are aged, and during seasons of increased acute respiratory infections.

According to Dripps and Deming' and Thorén' there is a higher incidence of atelectasis in men. DeWeese,' on the other hand, believes atelectasis to be twice as common in women because of their thoracic rather than abdominal type of respiration.

Because of the use of grossly divergent diagnostic procedures, surgical techniques, and selection of patients by the various investigators, incidence rates ranging from 5 to 47 per cent have been reported in the literature. Thus it may well be sufficient to state that the incidence of postoperative atelectasis is high, and that this clinical condition represents from 90 to 94 per cent of all postoperative pulmonary complications.

The primary cause of atelectasis is at present a controversial subject focused upon three proposed possibilities.

First, it is believed by most of the recent investigators, that atelectasis is primarily the result of broncho-occlusion by secretions of the respiratory tract which are normally evacuated by the cilia, the cough mechanism, and the expulsive force of respiration. If these secretions should accumulate in the respiratory tract and plug a bronchus, aeration of the involved lung segment would be impaired, and the entrapped air would thus cause collapse of the lung.

Second, atelectasis may be the result of reflex nervous stimuli. This is based upon the assumption that the lung and bronchi are neurologically controlled, myoelastic organs which become atelectatic by active broncho-

---

*Third Prize (tie), 1958 Essay Contest, American College of Chest Physicians.
**University of Oregon Medical School.
spasm or lung contraction. Evidence favoring this etiology is indirect, and includes the work of Bergamini and Shepard and of Santee, who found no demonstrable bronchial occlusion in several cases of massive postoperative atelectasis examined post mortem.

The third possibility is that atelectasis may be due primarily to postoperative hypoventilation. Because respiration is painful and guarded after surgery, it is shallow and rapid and predisposing to fluid accumulation in the small bronchioles. While Beecher gives evidence to support this etiology, Brattström’s studies on gross ventilation before and after high laparatomies, show hypoventilation to be insignificant in the production of atelectasis.

With present experimental evidence, it is not possible to determine which of these etiologies is of paramount importance. More specific etiologic agents to consider are: a decreased ability to expel bronchial secretions due to impairment of the cough reflex by pain or narcotics, a depression of ciliary action by anesthesia, an increased production of secretions after surgery, and a decreased caliber of the bronchial tree resulting from a swollen mucosa, barbiturate, or cyclopropane.

In Table 1 is presented a modification of Brattström’s list of presumed etiologic agents of atelectasis.

The diagnosis of atelectasis can be made from a study of the clinical course and symptoms of the postoperative patient. No symptom is evident if only a small area of lung is atelectatic, whereas variable physical findings, changing hourly, may be found in multiple small areas of involvement or patchy atelectasis.

In moderate cases of atelectasis the onset may be sudden or insidious, with subjective symptoms present usually within three days of the oper-

---

**TABLE 1 — ETIOLOGICAL AGENTS OF ATELECTASIS**

<table>
<thead>
<tr>
<th>Bronchitis</th>
<th>Bronchitis</th>
<th>Bronchitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Irritating anesthesia</td>
<td>Irritating anesthesia</td>
<td>Irritating anesthesia</td>
</tr>
<tr>
<td>Inhalation of stomach contents</td>
<td>Inhalation of stomach contents</td>
<td>Postoperative pain</td>
</tr>
<tr>
<td>Postoperative pain</td>
<td>Autonomous reflex</td>
<td>Decreased diaphragm movement</td>
</tr>
<tr>
<td>Autonomic reflex</td>
<td>Chronic and upper resp. infections</td>
<td>Decreased bronchial movement</td>
</tr>
<tr>
<td>Infection</td>
<td>Dehydration</td>
<td>Decreased muscle tone</td>
</tr>
<tr>
<td>Dry atmosphere</td>
<td>Increased mucin</td>
<td>Anoxia</td>
</tr>
<tr>
<td>Surgical trauma</td>
<td>Atropine</td>
<td>Carbon dioxide excess</td>
</tr>
<tr>
<td>Intubation</td>
<td>Scopolamine</td>
<td>Viscous sputum</td>
</tr>
</tbody>
</table>

---

**REDUCTION IN BRONCHIAL CALIBER**

**STAGNANT BRONCHIAL SECRECTIONS**

**DEFICIENT VENTILATORY AND EXPULSIVE MECHANISM**

---

**ATELECTASIS**
ation. At first, there may be only tightness in the base of the lung or uncomfortable breathing sensations, but these are soon followed by difficulty with expectoration, chest pains on breathing, coughing and moving, listlessness, and occasionally pleural pain. Elevations of temperature, pulse and respiration during the first, second and third postoperative days should immediately suggest the possibility of atelectasis. The temperature usually rises to 101°F or higher and the pulse increases disproportionately to 120 or higher. After the first three days the temperature and pulse may decrease, the pulse decreasing more rapidly than the temperature. In the presence of secondary pneumonitis, the temperature remains elevated.¹

In a massive collapse of the lung, the onset of atelectasis is acute, and the patient appears dangerously ill with mounting fever, sudden, extreme breathlessness or a sensation of suffocation, pleural pain, and profound cyanosis. Unless adequately treated, death can occur within a few hours.¹²

The physical examination of an atelectatic patient presents at first findings of dulness to flatness on percussion, no tactile fremitus or breath and voice sounds, and no rales over a large area. These signs rapidly change to those of solidification, making differentiation from postoperative pneumonia and infarction difficult. Atelectasis is diagnosed when an elevation of the diaphragm and a displacement of the heart and mediastinum to the side of the lesion are detected by physical examination or by the use of x-ray films. A downward inclination of the ribs, a narrowing of the intercostal spaces on the affected side, and a sharply defined segment of non-aerated lung⁴ may also be seen on x-ray films.

Because the atelectatic patient is often too ill to be moved and properly positioned, the use of x-ray films is somewhat limited, and the physical signs of percussion and auscultation often cannot be elicited.

In doubtful or serious cases of atelectasis, bronchoscopy may be indicated, not only for diagnostic purposes, but also for treatment, if broncho-oclusion is present.

Recovery from atelectasis is spontaneous in the majority of cases. Although in beginning atelectasis the lung is readily re-expanded, prolonged atelectasis may result in superimposed pulmonary infections, bronchopneumonia, bronchiectasis, and lung abscess. Thus one should be continually aware of atelectasis in the postoperative patient, and clues such as any unexpected fever or delayed convalescence should be investigated promptly.

In early atelectasis, treatment is simple and effective, consisting of procedures to liquefy the mucus plug, release the bronchospasm, and allowing the liquefied plug to slip out either by coughing or by posturing of the patient.

The commonly used therapeutic measures for relief from atelectasis include the following:¹³-¹⁶

1) inducing a natural drainage of the lung by frequent changes in posture and by having the patient lie on the side of the unaffected lung.
2) encouraging the patient to cough even though it is painful, by assuring him that the wound is firmly sutured, by giving support to his abdominal incision while coughing, and by restoring the cough reflex by discontinuing the use of narcotics,

3) combating shallow postoperative breathing by having the patient take deep breaths or by the cautious use of carbon dioxide hyper-ventilation,

4) administering epinephrine to relieve bronchospasm and sulfa drugs or antibiotics to treat pre-existing infections, and

5) aspirating bronchial secretions by bronchoscopy if the occlusion is not decreased within 12 hours after the use of the other procedures.

Recently, newer methods for the treatment of atelectasis have been proposed. Camarata et al.\textsuperscript{9} use the enzyme, trypsin, or a wetting agent, the detergent, triton A-20, to liquefy the mucus plug; Baker et al.\textsuperscript{10} administer sodium iodide to the postoperative patient to produce an easy and rapid evacuation of the viscid secretions formed during anesthesia; and Marshall\textsuperscript{11} promptly relieves atelectatic symptoms by a compression of the patient's thorax while he is coughing to dislodge any bronchial plugs. Although effective, these methods are not as yet used routinely.

Atelectasis can be prevented if the necessary precautions are taken in the surgical preparation and the postoperative care of a patient. To decrease the incidence of postoperative atelectasis, routine prophylactic procedures, such as those described by Thoré,\textsuperscript{1} and Marshall,\textsuperscript{12} are practiced in most hospitals today.

Surgery on patients with respiratory tract infections (including even mild colds) are postponed until two weeks after the infection subsides, since infected material may be aspirated during anesthesia and may cause bronchial occlusion. Smoking is decreased to a minimum for a week before the operation. Narcotics are used cautiously because they may produce shallow respiration and suppress the cough reflex. Anesthesia is given at even rates. If spinal anesthesia at higher levels is used, carbon dioxide and oxygen inhalations are administered to insure adequate and complete expansion of the lung, in the event that the accessory respiration muscles become paralyzed.

Postoperatively, the patient is aroused as soon as possible, and his throat and trachea are thoroughly cleaned. A radical change of position hourly is also advocated. For gravity drainage of bronchial secretions and to prevent aspiration of regurgitated gastric contents, the Trendelenburg position may be used during and immediately after surgery. Chemotherapeutics are given prophylactically when necessary.

The use of physiotherapy and hyperventilation procedures have also been proposed for the prevention of atelectasis. Thorén,\textsuperscript{1} in a study of 343 cholecystectomy cases, found postoperative pulmonary complications to be minimal when physiotherapy, in the form of respiration and coughing exercises and postural drainage, was used.

The effects of hyperventilation in the prevention of atelectasis were first demonstrated by Scott and Cutler,\textsuperscript{13} who had a patient rebreathe into a paper bag to accumulate carbon dioxide and stimulate greater
tial volumes. Because the face masks used in this method were often leaky and because the subsequently developed, contour masks proved bulky and expensive, Schwartz and Dale proposed the use of a rubber tube to extend the patient's normal respiratory dead space by a liter. By rebreathing in this manner for five minutes every one to two hours postoperatively, significant increases in alveolar pCO₂ and arterial pCO₂ are produced to stimulate the central nervous system, resulting in hyperventilation.

SUMMARY

Atelectasis is still an important postoperative pulmonary complication which threatens the patient who has undergone major surgery. It is generally agreed that the predominant causal factor is a complete bronchial occlusion by secretions, although reflex nervous stimuli have also been implicated to contribute greatly to the occlusion. The role of postoperative hypoventilation in the etiology of atelectasis remains a controversial one.

The diagnosis of atelectasis depends chiefly upon a study of the signs and symptoms, but is aided by the findings of physical examination, X-ray films, and occasionally, bronchoscopy. Treatment and prevention of atelectasis are simple and effective, consisting chiefly of measures to remove the obstruction, such as, coughing, postural drainage, or bronchial catheterization.

RESUMEN

La atelectasia es aún una complicación pulmonar postoperatoria importante que amenaza a los enfermos que han pasado por la cirugía mayor. Se admite generalmente que el factor causal predominante es la oclusión de los bronquios con secreciones, aunque los estímulos de los reflejos nerviosos también pueden estar implicado para contribuir grandemente a la obstrucción.

El papel de la hipoventilación postoperatoria en la etiología de la atelectasia es aún discutido.

El diagnóstico de atelectasia depende en especial del estudio de los signos y síntomas pero es auxiliado por los hallazgos del examen físico, las películas de rayos X y ocasionalmente, la broncoscopia. El tratamiento y la prevención de la atelectasia son simples y efectivos consistiendo principalmente en la eliminación de la obstrucción por la tos, canalización postural o cateterización bronquial.

RESUMÉ

L’atelectasie reste encore une complication pulmonaire postopératoire importante, qui menace le malade qui a subi une sérieuse intervention chirurgicale. Il est admis généralement que le facteur causal prédominant est une occlusion complète des bronches par les sécrétions, bien qu’un réflexe nerveux ait aussi été également considéré comme contribuant fortement à l’occlusion. Le rôle de l’hypoventilation postopératoire dans l’étiologie de l’atelectasie reste controversé.

Le diagnostic d’atélectasie dépend principalement d’une étude des signes et des symptômes, mais il est aidé par les constatations de l’examen physique, de la radiologie et éventuellement de la bronchoscopie. Le traitement et la prévention de l’atélectasie sont simples et efficaces. Ils consistent principalement à prendre des mesures pour éliminer l’obstruction, telles que la provocation de la toux, le drainage de posture et le cathétérisme bronchique.

ZUSAMMENFASSUNG

Die Atelektase bedeutet auch heute noch eine wichtige postoperative pulmonale Komplikation, die die Kranken bedroht, bei denen größere Operationen vorgenommen worden sind. Es besteh ne allgemeine Übereinstimmung daruber, dass der predominierende ursächliche Faktor hierfür ein kompletter Bronchialverschluss durch Sekretmassen ist, obwohl reflektorische, nervöse Reflaxe ebenfalls in beträchtlichem Maße am Zustandekommen des Verschlusses beteiligt sind. Die Rolle einer postoperativen Hypoventilation für die Ätiologie der Atelektase bleibt umstritten.

Die Diagnose der Atelektase hängt hauptsächlich von einer Beobachtung der Anzeichen und Symptome ab; sie wird aber erleichtert durch die Befunde der physischen Untersuchung, Röntgenuntersuchung und gelegentlich der Bronchoskopie. Behandlung und Verhütung der Atelektase sind einfach und wirksam; sie bestehen hauptsächlich in Maßnahmen zur Behebung der Obstruktion, sowie Husten, Tieflagerung oder Bronchialkatheterisierung.
REFERENCES


ESOPHAGEAL MONILIASIS

Moniliasis is common in the chronically ill. Severe esophagitis may occur either by extension from the oral cavity or as an isolated infection. The disease may be uncovered early in its course by the striking radiographic findings following a barium swallow. The involved portion of the esophagus shows irritability and spasm. The mucosa has a peculiar granular, cobblestone appearance which may be limited to a segment of the esophagus or extend throughout its length. This appearance is due to a pseudomembranous lining, consisting of Monilia and debris. Hyperemia, inflammation and edema of the deeper layers are associated with the mucosal ulceration. Detected and treated early, esophageal moniliasis is relatively innocuous and the symptoms are quickly relieved. Undetected, it progresses and may contribute to the patient's death. It is incumbent upon the radiologist to consider monilia infection of the esophagus in debilitated patients with sudden onset of dysphagia and pain on swallowing, since specific and effective treatment is readily available.


ANTICOAGULANTS IN CORONARY DISEASE

In acute myocardial infarction, the consensus is that all cases should receive anticoagulant therapy. Carefully compiled studies by many independent investigators give statistics which warrant an expected improvement in the mortality rate of from 35 to 50 per cent in such therapy.

In long-term therapy, after a single myocardial infarction our results show the mortality rate to be less than one half that of the controls five years after the attack (44.2 per cent mortality in the control and 18.3 per cent in the treated). These findings generally agree with those in the literature. It is to be noted that especially in the group with recurrent attacks which in the control series has an increased mortality rate, the use of anticoagulant therapy would seem to be even more beneficial.