The Importance of Bronchial and Bronchiolar Physiology and Pathophysiologic Changes in the Management of Chest Injuries*

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Introduction

Pulmonary problems remain high on the list of important and even lethal complications following both surgical and accidental thoracic trauma. These problems include infection, bronchorrhea, wheezing, cough, dyspnea and all types and degrees of decreased pulmonary ventilation. It is the purpose of this paper to review those normal bronchial and bronchiolar mural and mucosal changes which are capable of causing the symptoms and signs of airway obstruction and to consider the relation of these changes to pulmonary complications following trauma. Emphasis will be placed on the similarity between physiologic and pathologic alterations, on the importance of these alterations when they antedate the injury and upon methods of predicting, detecting and treating these changes and the hypoventilatory states which they tend to produce.

Normal-Abnormal Mural Dynamics of Bronchi

The mechanisms of wheezing, cough and airway obstruction in diseased bronchi are fundamentally the same as those occurring in normal states. The differences mostly lie in severity, duration and in mural stiffening. For example, cough, bronchorrhea, vascular engorgement and even smooth muscle spasm occur due to many stimuli including, but not limited to, disease processes. Normal reflex responses become abnormal states when thresholds are sufficiently lowered, when vascular engorgement persists and when lymph stasis, leukocyte infiltration and fibroplasia ensue. The attendant loss of elasticity of the entire pulmonary lobule or unit which is affected compounds the problem by further limiting bronchial-bronchiolar-alveolar ventilation. Removal of bronchial secretions and cellular debris is thus impaired and stagnation and secondary infection result. Infection in turn results in further engorgement, lymph stasis, cellular infiltrate and, if prolonged, fibrosis. At both ends of this normal-abnormal spectrum the importance of the factors is measured by the degree to which the normal flow of tracheal-bronchial-bronchiolar-alveolar air is impaired.**

The mural and mucosal changes and reactions which are common to all degrees of both normal and abnormal states may be listed as follows:

(a) Serous and mucoid secretion, intraluminal; (b) Alterations in sensitivity (threshold) of cough and spasm reflexes; (c) Increases in thickness of walls due to vascular engorgement, lymph stasis, leukocyte infiltration and fibrosis and the resultant decreases in lumen to wall ratios and lung elasticity.

Perhaps retained secretions and decreases in lumen to wall ratio give the answer, in terms of size alone; perhaps not. It is true, however, that small bronchial and bronchiolar obstruction interferes more with ventilation than does large bronchial obstruction. A more basic reason may well be that in most, if not all, cases of small bronchial obstruction post trauma one can find definite evidence that important inflammatory changes found in the bronchi were present before the injury. Also, such inflammatory changes tend to be very widespread, sparing little or none of either lung, although varying in severity from part to part.


**Gas exchange between lung and blood is not within the scope of this paper.
part. It becomes important, therefore, to detect and evaluate any bronchitis or bronchiolitis the patient may have had prior to injury. It should be stressed that while any or all of these changes can occur due to acute, subacute or chronic infections, they also can and do occur in the absence of any proof that infection is primary. On the contrary, infection, when present, often plays a secondary role and is itself brought about by the bronchial obstruction incident to the above described mechanisms.

**The Detection of Pre-Existing Bronchitis in an Injured Patient**

If the injured patient is capable of answering questions intelligently, it may prove very helpful to elicit a history of any of the following, all of which strongly suggest the presence of bronchitis or bronchiolitis before the injury:

(a) Frequent respiratory infection (clinical diagnosis); (b) persistent cough following colds; (c) wheezing during or following respiratory infections; (d) chronic cough, early morning cough or “cigarette cough”; (e) “sinus trouble”; (f) specific pulmonary diagnoses, such as emphysema, bronchitis, bronchiectasis, and (g) unexplained dyspnea and especially progressive dyspnea.

Physical examination should include a search for signs of inflammation, acute or chronic, in any portion of the respiratory system or its appendages. Occult bronchitis often accompanies less subtle signs of inflammation elsewhere in the respiratory system. Any of the following physical signs should alert the examiner to suspect bronchitis:

(a) Nasal edema, obstruction or secretion; (b) lymphoid hypertrophy in the pharynx; (c) laryngitis, deep voice, hoarseness; (d) sensitive cough reflex; (e) patchy differences in the quality of breath sounds upon auscultation of the lungs; (f) rhonchi, wheezes, rales, and (g) tachypnea or any of the other objective physical signs of hypoventilation.

**The Clinical Detection of Hypoventilation**

Inadequate pulmonary ventilation may be obvious in the presence of labored breathing, pallor, cyanosis or if the patient can report severe dyspnea to the examiner. Occult ventilatory inadequacy can also be detected. Any of the following should suggest its presence:

(a) Broken sentence speech due to pauses for breath; (b) apprehension, obstreperousness or delirium; (c) tachypnea and tachycardia, and (d) arterial hypertension.

CO₂ retention probably outweighs hypoxia in producing the above signs, but both are factors, along with the metabolic acidosis which results from asphyxia.

**Objective Pulmonary Ventilation Testing**

The measurement of pulmonary ventilation (MBC and FEV) in an injured patient is not usually possible. A simple test such as having the patient blow on the palm of the examiner's hand may be much more helpful in evaluating his functioning ventilatory capacity for survival than the objective tests. The circumstances dictate that an evaluation must be made by clinical means, which surely may include measurements when applicable, but which must also be capable of working without them when necessary.

**Anatomic Alterations Associated with Hypoventilation**

Important hypoventilatory states may be divided arbitrarily into two groups:

1. Those that threaten life.
2. Those that delay recovery; and either may include the following:

(a) Laryngeal or major tracheobronchial obstruction; (b) massive, bilateral and/or tension pneumothorax; (c) respiratory muscle paralysis; (d) multiple rib and cartilage and/or sternal fractures resulting in large flail chest wall segments; (e) displacement—compression of lung tissue by large volumes of fluid or herniated viscera,
and (f) widespread bronchiolar obstruction with or without attendant interstitial pneumonitis.

For the purposes of this discussion, only the last will be considered. As a matter of fact, if there is a total absence of bronchiolar obstruction, severe degrees of any of the others may be tolerated surprisingly well. The converse is also true. Proper attention to this matter would appear to go along the following lines:

1. Suspicion, from history, of pre-existing bronchitis.
2. Detection, from physical examination.
3. Control, from therapy.

The same thinking may well be applied to management after the emergency in all stages of the therapy. The most important observation of all is the detection of hypo-ventilation, in any degree. Secondly, the examiner must give attention to those alterations which constitute signs of the tendency towards bronchiolar changes, as they may occur or worsen at any time during the course of therapy, and thus endanger the patient's life.

In a case reported elsewhere, the authors described a patient with multiple injuries, none severe enough to cause death, but in whom death ensued, due to widespread small bronchial obstruction. The clinical course, the mode of death and the pathologic findings at necropsy were very similar in that case to those of a known asthmatic who underwent successful mitral valve surgery only to expire in the postoperative period from pulmonary insufficiency due to small bronchial (bronchiolar) obstruction. The following summary includes only the most pertinent of the salient features of this case:

Mrs. R. D. was readmitted to the Baylor Medical Center in Dallas in February, 1962 for mitral valvuloplasty. Previous admissions had established the diagnosis of mitral stenosis, bronchial asthma and pulmonary insufficiency. Respiratory difficulties including wheezing, dyspnea, pallor, cyanosis, restlessness, apprehension, hypotension and excessive bronchial secretions characterized her entire postoperative course. She expired on March 14, 1962, one month following the mitral valve repair, of inadequate pulmonary ventilation. Pathologic examination revealed gross and microscopic evidence of severe pneumonitis in the right upper and left lower lobes. No areas of lung were completely free of the characteristic bronchial changes alluded to earlier in this paper. Figure 1 is a photomicrograph which shows hyperemia, edema, fibrosis, leukocyte infiltration and marked thickening of submucosa, muscular and adventitial portions of bronchiolar wall. All of these findings denote chronicity and represent considerably more time than the one month following the surgery. The bronchiolar lumen is noted to be occluded by mucus and detritus. Figure 2 shows alveolar septal disruption representing emphysema, plus severe thickening and presumed stiffening in the wall of a tiny bronchiole due to fibrosis and cellular infiltrate. The lumen to wall ratio is strikingly decreased. This section was taken from the least involved, or most nearly normal lung area. These findings represent conclusive proof that the bronchial and bronchiolar changes which proved uncontrollable and led to death in the postoperative period long antedated the surgery. They also point up the vulnerability of a patient who sustains surgical or accidental trauma superimposed upon pre-existing bronchial disease. This, of course, represents an extreme case; however, it is the opinion of the authors that the same mechanisms obtain.
in less severe states and that the same principles of suspicion, diagnosis and treatment should prevail. Along this line it would seem appropriate to mention the factor of the body's response to atmospheric temperature and humidity.

**Chilling and the Physio-pathology of the Bronchi**

Peterson in 1934, McCord in 1941, Ralston and Kerr in 1945, Mathov in 1950, Goldstein in 1951, Waddy in 1952 and others have made reference to climatic and thermal effects upon patients and their respiratory systems. While none of these has completely demonstrated the pathways through which respiratory membranes respond to thermal stimuli, they all agree that there is a response. Both in the available literature and in the authors' own experience, the response to heat and cold can be fairly well observed in terms of the following mucous membrane and submucous parameters:

(a) Reflexes for sneezing and cough can be stimulated and their thresholds can be changed; (b) mucosal and submucosal vascular engorgement occurs and recedes; (c) serous and mucoid secretion appears, and (d) tone and spasm of smooth muscle is altered.

These are quite evident in the nasal mucosa and it is the opinion of the authors that the same changes can occur in the bronchi due to thermal stimuli. It is noteworthy that these are the same physiologic-pathologic changes in bronchi which were described earlier in this paper. Because the responses represent the effector limb of some kind of reflex system, anything which lowers the threshold of the reflex can be of clinical importance. It would seem a valid assumption that inflammatory bronchial states, whether due to infectious, non-infectious or combined causes, would tend to lower the reflex thresholds and thus predispose to secretion, engorgement, muscular spasm and ultimately bronchiolar obstruction. This assumption is based upon the widely held physiologic aphorism that inflammation near nerve endings characteristically lowers the threshold of a reflex arc.

One of the main theses of this paper is that any degree of chilling of the body, including exposure to the usual 72° to 76° F. work area room temperatures in hospitals, could possibly result in cough, sneezing, wheezing and engorged bronchial and bronchiolar mucous membranes, plus excesses of bronchial and bronchiolar secretions. Further, since some reflex mechanism is presumed, it is felt that pre-existing bronchial inflammation will render the patients' membranes more sensitive and the response more severe. The ultimate result of swollen bronchioles, thickened and more copious secretions and smooth muscle spasm is faulty pulmonary ventilation. While we can offer no proof, our experience strongly suggests that the prevention of chilling is one of the most important ways of maintaining adequate pulmonary ventilation in post-injury and postoperative patients.

**Practical Methods of Preventing Chilling and Thus Preventing Hypoventilation in Injured Patients**

1. **Encase the patient's entire body and limbs in light weight cotton clothing.** Bed covers and sheets will not offer the same protection as will garments. Only the hands and the head and neck may be left out. The simple, sure routine includes long sleeve, long leg cotton pajamas, light cotton undershirt and sox, with the addition of covers as needed.

2. **These garments must be changed as often as necessary to keep the patient dry.**

3. **After exposure to room temperature for necessary examinations and treatment clothing must be reapplied immediately.**

4. **Room atmospheric control at constant temperature and humidity is very desirable.**

This routine generally serves exceedingly well as prophylaxis; however, inevitably, patients will be seen in whom this prophylactic program was not used or was ineffective. In these, therapy to restore adequate ventilation must be instituted at once.
RESTORATION OF ADEQUATE PULMONARY VENTILATION

Objectives of Treatment
1. Reduce bronchial and peribronchial edema.
2. Remove retained endobronchial secretions.
3. Treat infection as specifically as possible.

Methods of Treatment
1. The prophylactic clothing discipline as outlined above can be life-saving as a part of the therapy. In the authors' opinion, the application of multiple layers of light weight cotton garments can be an excellent "bronchodilator."19
2. Retained endobronchial secretions in the larger bronchi can be removed readily by the use of the naso-bronchial catheter suction method described by Haighta and Haight and Ransona.19 Removal of the mucus and detritus plugging in the bronchioles usually requires the reduction of edema and the control of spasm by inserting and then suctioning. Heated aerosol as used by the authors and described by Cushing and Miller19 is recommended to provide the essential to adequate airway humidification, which is enough water in a vapor state in the inspired air to reach the terminal bronchioles. Wetting agents and chemical bronchodilators both are value. However, their worth is greatly enhanced if the deranged physiology is corrected, too. Likewise, intermittent positive pressure respiratory assistance is a good means for delivering aerosols and water vapor to the smaller bronchi, but its chief usefulness is as a ventilatory assist, rather than as a bronchodilator. Here too, any good that comes from the use of IPPB will be greatly augmented if the physiologic derangements are corrected simultaneously.
3. Whenever possible, antibiotic therapy of bronchial infection should be based on cultures and sensitivity studies. In addition, the reduction of edema and removal of bronchial and bronchiolar secretions may remove most of the actual cause of pulmonary infections (bronchial obstruction) in post-traumatic patients.
4. Adequate oxygenation is rarely enough to keep an injured patient in the range of safety. Adequate pulmonary ventilation is also required. The success of all attempts at oxygenation and ventilation is directly dependent upon airway patency and the functional integrity of the chest wall, diaphragm and the lungs themselves. During a period of emergency, assisted breathing with almost any intermittent positive pressure apparatus may be lifesaving by providing precious time to get under way the various other measures which will correct the altered physiology and restore the anatomic integrity of the respiratory organs.

A Look at Some Common Practices
Proper, physiologic management of injured patients with any respiratory problem should avoid at all costs:
---heavy doses of narcotics
---nakedness
---refrigerated, dehumidified oxygen tents
Lasagna and Beecher18 and probably others have pointed out the fact that large doses of narcotics are unsafe and smaller doses may be just as effective. In patients with chest injuries, restlessness, apprehension and even delirium, stupor or coma may be due to faulty pulmonary ventilation, in which narcotics could be lethal.

Nakedness causes and perpetuates the bronchial changes which lead to hypoventilation. Patients should be fully clothed at all times except when exposure is essential for examination or treatment.

Refrigerated oxygen tents dehumidify and cool the inspired air and the skin. While this may initially be welcomed by the patient as an aid to his comfort, it can result in bronchial edema and secretions leading to plugging of small bronchioles. The net result is hypoventilation.

BRONCHITIS AND BRONCHIOLITIS
In this presentation, primary emphasis has been placed on functional and struc-
tural changes in the bronchi and the profound effect such changes can have on pulmonary ventilation. While it is agreed that these changes cause entirely different effects when they take place in the large bronchi than when they occur in the bronchioles and smaller bronchi, due to the smaller lumen size, it is nevertheless a fact that structurally, functionally and pathologically the changes are identical. Simply stated, bronchitis may differ from bronchiolitis clinically, but pathologically, they are the same, and further, careful study might reveal that one does not even exist without the other, although the one or the other may be responsible for the predominant clinical manifestations.

**Summary**

In this presentation attention to the bronchi has been called in an attempt toward better understanding of pulmonary complications following trauma. Emphasis has been placed upon the similarity between abnormal bronchial states and normal dynamics of bronchial physiology. Many of the more severe post traumatic and postoperative pulmonary problems have as their underlying basis bronchitis which antedated the injury. Methods of detecting occult hypoventilatory states and objectives and methods of treatment and control were specified.

**Resumen**

En este trabajo se llama la atención sobre los bronquios, intentando lograr una comprensión mejor de las complicaciones pulmonares después de traumas. Se recalca la similaridad entre los estados anormales de los bronquios y la dinámica bronquial de la fisiología. Muchos de los problemas más graves post-traumáticos y postoperatorios tienen una base subyacente de bronquitis que precedió a la lesión. Los métodos para descubrir los estados de hipoventilación y el modo de tratarlos se detallan.

**Résumé**

Dans cette communication, l'auteur attire l'attention sur les bronches, pour essayer de mieux comprendre les complications pulmonaires qui suivent un traumatisme. Il met l'accent sur la coexistence d'états bronchiques anormaux et de constantes normales de la physiologie bronchique. Beaucoup des atteintes post-traumatiques les plus graves et des problèmes pulmonaires post-opératoires ont pour base une bronchite antérieure au traumatisme. L'auteur précise les méthodes qui permettent de détecter les états hypoventilatoires occultes et les objectifs et méthodes de traitement et de contrôle.

**Zusammenfassung**

Bei dieser Darstellung wurde die Aufmerksamkeit auf die Bronchien gerichtet mit dem Ziel eines besseren Verständnisses pulmonaler Komplikationen, die sich nach einem Trauma entwickeln. Die Bedeutung wurde auf die Ähnlichkeit zwischen abnormen Verhältnissen an den Bronchien und normalen Zuständen der Bronchialphysiologie gelegt. Viele der schwereren postoperativen und posttraumatischen pulmonalen Probleme haben als Grundlage eine Bronchitis, die dem Trauma vorausgegangen ist. Methoden zur Entwicklung verborgener hypoventilatorischer Zustände und Maßstäbe und Methoden ihrer Behandlung und Beherrschung werden im einzelnen erläutert.

**References**


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