In the last of a three-part series on pulmonary physiology, the author focuses on the anesthetic management of the patient who has primary pulmonary disease and is unable to absorb certain respiratory stresses during the surgical procedure. Attention is given to the physiologic and pathologic changes that can compromise the function of an abnormal lung.

In the first article in this AANA Journal series (February, 1980), the alveolus was discussed as the physiologic and anatomic unit of pulmonary gas exchange with the blood stream. Attention was focused on this physiologic unit and on the physiologic changes that can occur in transient ventilation/perfusion imbalances.

In Part II (April, 1980), respiratory insufficiency frequently found in the postoperative patient who may have had normal lungs prior to the operation was addressed. Clinical observation of the patient and the frequent postoperative pulmonary problems that are seen by the anesthetist in the immediate postoperative period were also discussed. The patient with normal lungs is susceptible to pathophysiologic changes in ventilation/perfusion when anesthesia is administered. Early recognition and prevention of these postoperative pulmonary problems is stressed so that the patient’s postoperative morbidity can be minimized.

In this article, we will focus on those patients who have primary pulmonary problems that cannot be prevented. They are admitted with the pulmonary disease which must be taken as a given in their clinical status. As noted in Part I of this series, physiologic imbalances in ventilation/perfusion can occur in normal lungs during the course of normal function. These physiologic imbalances can become pathologic in the patient who is administered an anesthetic, even if he has no primary lung disease.

Both physiologic and pathologic changes can be superimposed on the compromised function of an abnormal lung. Management of these patients requires modification in technique and extreme care in assuring delivery of oxygenated blood for aerobic metabolism in cellular respiration.

The concept of pulmonary reserves will be a useful one to remember, since the healthy individual not undergoing an operation generally has a large pulmonary reserve that can compensate for transient changes in ventilation or perfusion. The patient with normal lungs, undergoing an anesthetic for purposes of an elective operation, can generally absorb certain respiratory stresses if compensatory mechanisms are facilitated, or at least, not interfered with by the intervention of anesthetic techniques and surgical procedures.

However, for the patient with compromised pulmonary reserves due to a primary pulmonary illness, the lungs are far less forgiving so to speak.
The amount of insult they can endure from further surgical or anesthetic interference in the new steady state brought about by the extra-compensatory efforts is much less. Compromise of pulmonary function with limited pulmonary reserves jeopardizes the patient, and produces a real deficit in the delivery of oxygenated blood to the periphery for aerobic metabolism upon which life depends. Further compensation is not possible if all pulmonary reserves have been exhausted, and often other organ systems cannot take up the slack.

We frequently attribute one organ failure to a preceding failure in another organ system. For example, we say that the heart failed because the lungs were bad (cor pulmonale) or, the kidneys failed because the liver had no further reserves (hepatorenal syndrome). Any primary organ failure puts a severe compensatory stress on other organ systems that must make up for the deficit.

If these other organ systems are stressed beyond their capacity to compensate with their reserves, multiple sequential organ system failures ensue, and the life steady state that is comprised of the functions of organ systems quickly collapses. Since all human life functions are critically dependent upon aerobic metabolism, for which adequate respiration is a precondition, management of the patient with compromised pulmonary reserves is a critical challenge. This is the case particularly when the patient must undergo the stress of an operation, superimposing further the risks of the postoperative pulmonary problems found in the individual with healthy lungs.

Acute changes superimposed on chronic illness

The patient with chronic lung disease is at least as susceptible to the acute respiratory insufficiencies discussed in Parts I and II as the individual with normal lungs. However, the consequences of even a minimal pulmonary compromise may tip the patient over into frank decompensation. It is for this reason that prophylaxis for respiratory infections (with the use of some antibiotics in peak seasons for pneumonia or the administration of influenza vaccines) is indicated for patients who have chronic pulmonary disease.

Acute catastrophies can occur in the patient with compromised pulmonary function and underlying anatomic derangement in the lungs can occur with therapeutic manipulation. An example of this is the patient who pops an emphysematous bleb during "ambu" bag ventilation and sustains a pneumothorax. This closed pneumothorax under tension quickly compromises the rest of the lung.

Unless immediate recognition of the tension pneumothorax is followed quickly by evacuation of air from around the compromised lung, such a problem is lethal.

Similar disruption of the normal perfusion through a lung that is compromised in its capacity for ventilation can be similarly disastrous. For instance, a pulmonary embolism might be survivable in the patient with normal lung form and function, but for the patient who has stretched cardiac reserves to the limit because of pulmonary dysfunction, such a stress cannot be tolerated and cardiac decompensation develops.

Chronic pulmonary disease

Acute and chronic lung problems are often demarcated by their reversibility. Their reversibility depends upon their etiology. If a pulmonary process is inflammatory, often inflammation might subside, such as in pneumonia. The late effect of inflammation is to heal by scarring and few pulmonary fibroses are reversible. A third etiologic compromise is neoplasia. Many of the neoplasms that occur in the lung respond poorly to treatment.

Reversibility is also determined by the pathophysiology of the process. Obstructive lesions can diminish airflow or blood flow through the lung, or alveolar-capillary blocks can develop in chronic pulmonary fibrosis. Even normal ventilation and normal perfusion are ineffective in allowing gas exchange through the impermeable alveolar-capillary barrier after it has undergone the pathologic changes that resist gas exchange.

Many chronic lung diseases are given different names because of the morphologic differences or etiologies that bring them to the stage of pulmonary insufficiency. Functionally, however, they are much the same in the end-stage when the lung, for any of a variety of reasons, no longer functions effectively to oxygenate blood. The lungs does not have a wide repertoire of responses against a host of environmental influences and inner organ failures.

As chronic lung disease progresses, the pulmonary reserves are used up until an end-stage pulmonary insufficiency develops, analogous to the end-stage renal failure that is even more prevalent. End-stage renal failure patients can persist in surviving because there is an extra-renal substitute for at least some excretory functions available. However, since there are currently no effective means of extra-pulmonary respiration, end-stage pulmonary disease is a lethal condition that merely awaits the fatal further compromise.
The list of chronic pulmonary diseases outlined in Table I is largely based on the morphologic abnormalities found in examination of the lung, directly by histologic observation or indirectly through radiographic or clinical criteria. Early in the course of these illnesses, differential diagnosis is possible and specific methods of management may be employed, which are modified for each condition. Later, however, these different entities, morphologically or etiologically, become functionally similar as they blur into end-stage pulmonary insufficiency in their ultimate progression.

**Table 1**

<table>
<thead>
<tr>
<th>Chronic Pulmonary Disease</th>
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<tbody>
<tr>
<td>Bronchitis</td>
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<tr>
<td>Asthma</td>
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<tr>
<td>Emphysema</td>
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<td>Bronchiectasis</td>
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<td>Pneumoconiosis</td>
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Broad categories of chronic lung disease can be distinguished morphologically, radiographically or clinically; but functionally, they blur into end-stage pulmonary insufficiency in their ultimate progression.

**Bronchitis**

Bronchitis is an inflammatory process of the lower air passages, primarily, the segmental bronchi. Prolonged irritation of bronchial mucosa, secondary to an infectious organism or inhaled pollutants, stimulates overproduction of mucus from the mucosal goblet cells.

The patient with bronchitis has difficulty clearing this sputum because the mucociliary "escalator" that normally clears particles from the lesser air passages is undergoing gradual epithelial metaplasia. The stagnant secretions in the lower air passages allow bacterial superinfection, and long-standing recurrent infections can destroy the structural support of the lower bronchiolar passages, leading to bronchiectasis. Functional studies show expiratory air flow obstruction.

It is difficult to treat the underlying inflammatory process of chronic bronchitis. Consequently, the anesthetist, who must insure adequate ventilation for such patients undergoing surgery, has to focus particular attention on the prevention of further acute inflammatory infection and on assisting in the clearance of sputum. Further thickening and stagnation of the excessive mucus production by drying agents should be avoided.

**Asthma**

Asthma is a bronchiolar constriction caused by the hypersensitive smooth muscle around segmental bronchi that spasm, restricting the diameter of the airway. This bronchospasm does not inhibit inhalation as much as it interferes with exhalation, since positive intrapleural pressure on the narrowed airway lumen results in bronchiolar collapse. This is the phenomenon accountable for the air trapping that is worsened during attacks in the chronic asthmatic.

A severe attack, in which it is difficult to relax the bronchiolar smooth muscle as the excited patient traps more and more air in the distal airways, is called *status asthmaticus*. This is a life-threatening emergency, and the anesthetist may be called upon to administer bronchodilators, wetting agents, anti-inflammatory agents and supplemental oxygen to make up for the decreased ventilation through the constricted air passages. However, this must be done without causing any further direct bronchial irritation which may trigger more bronchospasms. This compounds the asthmatic state and reinforces the feedback cycle, characterized by hypoxemia and further excitation, as is observed in the panicky patient who is susceptible to these asthmatic attacks.

**Emphysema**

Emphysema is a structural change in the lung, characterized by an irreversible breakdown of alveoli and loss of lung elasticity. As with asthma, there is a collapse of the airways during the expiratory phase of respiration. This air trapping gradually leads to carbon dioxide retention and hypoxia, with a hyperexpanded "barrel chest."

Emphysema may have specific causes in certain enzyme deficiency states; and it may be congenital. In most individuals, however, it is an acquired disease occurring after prolonged exposure to cigarette smoke or other environmental pollutants. It may be idiopathic in its origin as well.

A chest x-ray of the emphysematous patient (Figure 1) may show an increase in the capacity of the chest, with flattened diaphragms and hyper-inflated lung fields. Some bullae or blebs may be seen in areas of consolidated airway destruction. These are weak points in the lung, highly susceptible to "blow outs" during the hyperinflation of ventilator management.

The emphysematous patient is susceptible to the life-threatening crisis of tension pneumothorax in the course of anesthetic ventilation if one of the structurally weakened areas in the lung blows out.
When this occurs, air enters the pleural space on each inspiration but does not re-enter the tracheobronchial tree on exhalation because of the collapse of the small perforation in the bleb. This trapped intrapleural air can expand with each inspiration and does not decrease with each expiration. Gradually, the minimal function in that lung is further compromised by this space-occupying air under pressure in the pleura. The mediastinum gradually shifts to the opposite side, also compromising the function of the less involved lung. Unless this problem is recognized and treated immediately, tension pneumothorax will be a lethal complication of chronic pulmonary emphysema.

The anesthetist must manage the patient with emphysema very carefully to minimize inspiratory pressure on the airway. Postural drainage, humidified air, and bronchodilators are preferable to intermittent positive pressure in maintaining pulmonary hygiene in these individuals.

**Bronchiectasis**

The bronchiectasis that is seen on the bronchogram (illustrated in Figure 2) is either a congenital abnormality or one acquired after longstanding chronic bronchitis when the inflammatory process invades through the epithelium to destroy the structural cartilagenous support of the segmental bronchi.

Lesser airways can also be affected in a variant of this problem known as bronchiolectasis. Foul sputum and fetid breath are characteristic of the patient with bronchiectasis since the stagnant secretions that build up in the dilated air passages are often superinfected. Antibiotics are indicated for these patients, but postural drainage and chest physiotherapy techniques are very important in clearing the airway.

Patients with bronchiectasis occasionally need thoracotomies for resection of severely destroyed areas of the lung to eliminate a focus of chronic infection. The anesthetic management of such patients must be carefully planned in advance to preserve the remaining pulmonary function, and to assist in clearing the air passages above the bronchiecatic areas.

**Pneumoconiosis**

A number of environmental or occupational exposures can lead to a reaction by the lung to irritants or toxins that are inhaled, either from the general atmosphere or pulled through a personal atmospheric contaminated conduit such as a cigarette. These toxins initially irritate the lung,
and it sets up an inflammatory reaction to them. Over a prolonged period of exposure, chronic scar formation and pulmonary fibrosis results, analogous to the irritant which produces pearls in oysters. However, the “pearl” in the lung is a structural change that prevents adequate gas exchange in the affected airways.

The irreversible structural changes that have already occurred are not the focus of the anesthetist’s attention, since it is too late to prevent these changes. Instead, the anesthetist’s effort is directed at preventing further inflammatory reaction on the part of the lung to any agents that may be introduced into the airway during anesthesia, and that may further tip the balance away from adequate compensated gas exchange.

The appearance of a lung affected by anthracosis—a form of pneumoconiosis in which carbon particles are trapped in the lung, sometimes mixed with silicates—is noted in Figure 3. This macroscopic view of such a lung makes it easy to understand how it would be compromised in its ability to oxygenate blood.

**Figure 3.**
*Anthracosis (black lung) is a chronic form of pneumoconiosis—fibrotic scarring of the lung induced by inhaled carbon and silicate particles.*

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**Malignant lung disease**

Malignant lung diseases share many of the physiologic abnormalities found in chronic benign lung diseases (which have a similar poor prognosis despite the misleading term *benign*), but they are often considered differently by the therapist when taking care of such patients. The focus has ordinarily been on the tumor and its eradication. Although concentration on curative eradication of tumor is an appropriate initial perspective, the majority of cancers in the lung are not “cured” so to speak. That is to say, the patient who has lung cancer will eventually go on to die with this disease.

It is not often that the death can be directly attributable to the lung cancer, however. It is often due to an acute inflammatory intercurrent illness. Pneumonia remains the leading cause of death among patients with lung cancer, both because of their debility, and because of the obstruction of the airway that prevents normal clearance mechanisms that prevent pneumonitis.

Another factor that enters into these cases is treatment. “Treatability”, as such, may be modified from the current perspective—distinguish a *disease* that cannot be treated from a *patient* who cannot be treated. All patients should have the benefit of treatment to relieve suffering and to improve the functional quality of their lives, even if such treatment is not directed at eradicating the malignant disease, but at maximizing their functional reserves despite the presence of the cancer. It is for this reason that a distinction is made between *operability* and *resectability*.

Some tumors are potentially resectable, but may reside within an “inoperable” patient. Such a patient may have severe limitations on his pulmonary reserve, in that individual eradication of the tumor would be ill-advised. The attention of the health care team should be directed at the patient around the tumor, and what the

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**Table 2**
*Tolerance for exertion—a simple clinical indicator of pulmonary reserve*

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<tr>
<th>Tolerance</th>
<th>Resection</th>
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<tbody>
<tr>
<td>1 Flight</td>
<td>Segmental</td>
</tr>
<tr>
<td>2 Flights</td>
<td>Lobar</td>
</tr>
<tr>
<td>3 Flights</td>
<td>Pneumonectomy</td>
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Tolerance of exertion is a rough indicator of pulmonary reserve, and may lead to an initial determination that though a lung tumor may be “resectable,” the patient is so compromised as to be “inoperable.”
Tolerance of exertion is a good indicator of pulmonary reserve for evaluating whether such a cancer should be resected (Table 2). A patient cured of cancer who becomes a pulmonary cripple has not had much of a life saved. Before the resection of the lung tissue designed to remove the cancer is undertaken, the question must primarily be addressed as to whether there will be any possibility for the patient to survive with the compromised pulmonary reserve that remains.

Treatment of the patient with primary pulmonary disease and compromised pulmonary reserves

The anesthetist’s attention in the management of patients with primary pulmonary disease is to very carefully assure adequate oxygenation and the delivery of arterialized blood, yet doing so with minimal effect on the injured lung itself. Generally, most anesthetic management of illness or injury in other body parts involves “resting the part.” In the exception of pulmonary disease, the injured organ itself is stressed in the treatment.

The role of the anesthetist remains preventive, but the patient’s primary pulmonary disease (as a preoperative status) is not preventable when presented to the anesthetist as a given. Rather, the focus of preventive management involves the prevention of progression of acute to chronic disease, or the superimposition of acute inflammatory pulmonary problems on the chronic underlying disease that may have already severely limited pulmonary reserves.

Other members of the health care team should also focus on ways to avoid producing a chronic lung “cripple” by the treatment method of the acute disease. With this focus of attention, the management of the patient with chronic pulmonary disease is not much different from the treatment of the patient with normal lung. Adequate oxygenation must be ensured with minimal damage to the lungs as the primary goal of the anesthetist. However, this preventive approach is desirable for the patient with normal lungs who undergoes an anesthetic; it is critical for the patient with compromised pulmonary reserve who is similarly undergoing treatment.

Acute catastrophies in patients with limited reserves

Patients with limited pulmonary reserves are more susceptible to acute changes in ventilation than patients who have normal lung anatomies. For example, the major disruption in ventilation often seen in patients with chronic obstructive pulmonary disease is pneumothorax. Pneumothorax can come from an external penetration of the chest in the instance of trauma. This type of pneumothorax, which is open and from which a sucking noise may be heard, is often survivable for the patient with normal lung function.

However, the lethal form of pneumothorax is most frequently seen in the patient with chronic lung disease. This is the tension pneumothorax that may occur from an internal leak of air from one of the weakened areas in the lung, particularly in a “blown out” bleb in the emphysematous lung. This allows air into the chest on inspiration, but collapses in expiration. The air is not evacuated from the chest, and breath to breath, the intrapleural space is filled with extrapulmonary air which shifts the mediastinum, compromising the function of the opposite lung.

The important differences between an open sucking pneumothorax and a closed tension pneumothorax are outlined elsewhere. However, the basic point for the anesthetist to consider while ventilating the patient with chronic lung disease is that the patient most likely to suffer the tension pneumothorax is the one who is least able to afford this further pulmonary compromise. If the inductive suspicion is high enough, even before the diagnosis is made in certainty (and, surely before the delay required for x-ray confirmation), the chest should be entered, converting a lethal tension pneumothorax into an open pneumothorax which is survivable.

The best technique to judge that in the operating room setting is to insert a large gauge needle in each chest cavity hooked to a syringe of approximately 30-50 cc capacity. If the plunger jumps from the barrel of the syringe on the affected side, the diagnosis of tension pneumothorax has been made simultaneously with its decompression.

An example of a catastrophe on the perfusion side is the pulmonary embolism which may decompensate the patient, not as a result of the disruption in ventilation, but because of an acute shift in perfusion pressure. The patient who suffers a pulmonary thromboembolism dies a cardiac death because of the rapid shift in hemodynamics. The pulmonary arterial hypertension that follows an abrupt occlusion of the perfusion in the lesser circulation causes right heart strain and right heart failure. This frequently leads to fibrillation and cardiac arrest.

The important feature to consider in pulmonary thromboembolism is not its ease of diag-
nosis, nor its effective therapy, since both are complex, costly and largely ineffective. The real virtue of preventive medicine is found in thromboembolism where prophylactic measures have proven effective, less costly, and life saving as shown in several large clinical trials.

Summary

Subtle and transient changes in ventilation/perfusion occur normally and are easily compensated for by physiologic maneuvers which help the patient expand ventilation with minimal disruption of perfusion. These normal compensatory mechanisms are suppressed in the patient with normal lungs undergoing anesthesia, and for that reason postoperative pulmonary complications are frequently seen. Prevention is the primary focus in the management of these postoperative pulmonary problems that occur in patients with normal lungs.

But there are certain pulmonary problems that are not preventable because the patient presents an already well established chronic lung disease. This chronic lung disease severely limits the pulmonary reserves, and the management of such patients is critical because further insult often puts them into frank pulmonary insufficiency, incompatible with life. The anesthetist’s management of such patients is geared to the prevention of acute disease progressing to chronic disease. Careful technique is used to minimize the introduction of acute inflammatory pulmonary problems over the baseline of chronically compromised pulmonary reserves.

With both chronic benign diseases and malignant lung disease, the focus should be on the patient and the pulmonary function that is necessary to secure vital functions, rather than on the eradication of the disease. Management of such patients should concentrate on the residual lung function that remains after the impact of our treatment, and on the careful avoidance of chronic crippling lung limitations as the result of the treatment employed for acute inflammatory pneumonitis or malignant lung disease. All patients can be treated to maximize their function in their chronic pulmonary insufficiency states, even if some pulmonary diseases are not treatable.

REFERENCES


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