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May 29, 1981

Raymond L.H. Murphy, M.D.
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Dear Ray:

You are a leading member of a distinguished ACCP section (committee). I was delighted to hear from my good friend, Bernie Gee, in his capacity as Chairman of the ACCP Section of Environmental Health. Bernie transmitted to me the original draft of the report "Environmental Pulmonary Carcinogens." I know that this is being reviewed by members of your committee and that revised versions are being contemplated. Permit me to share with you some early observations.

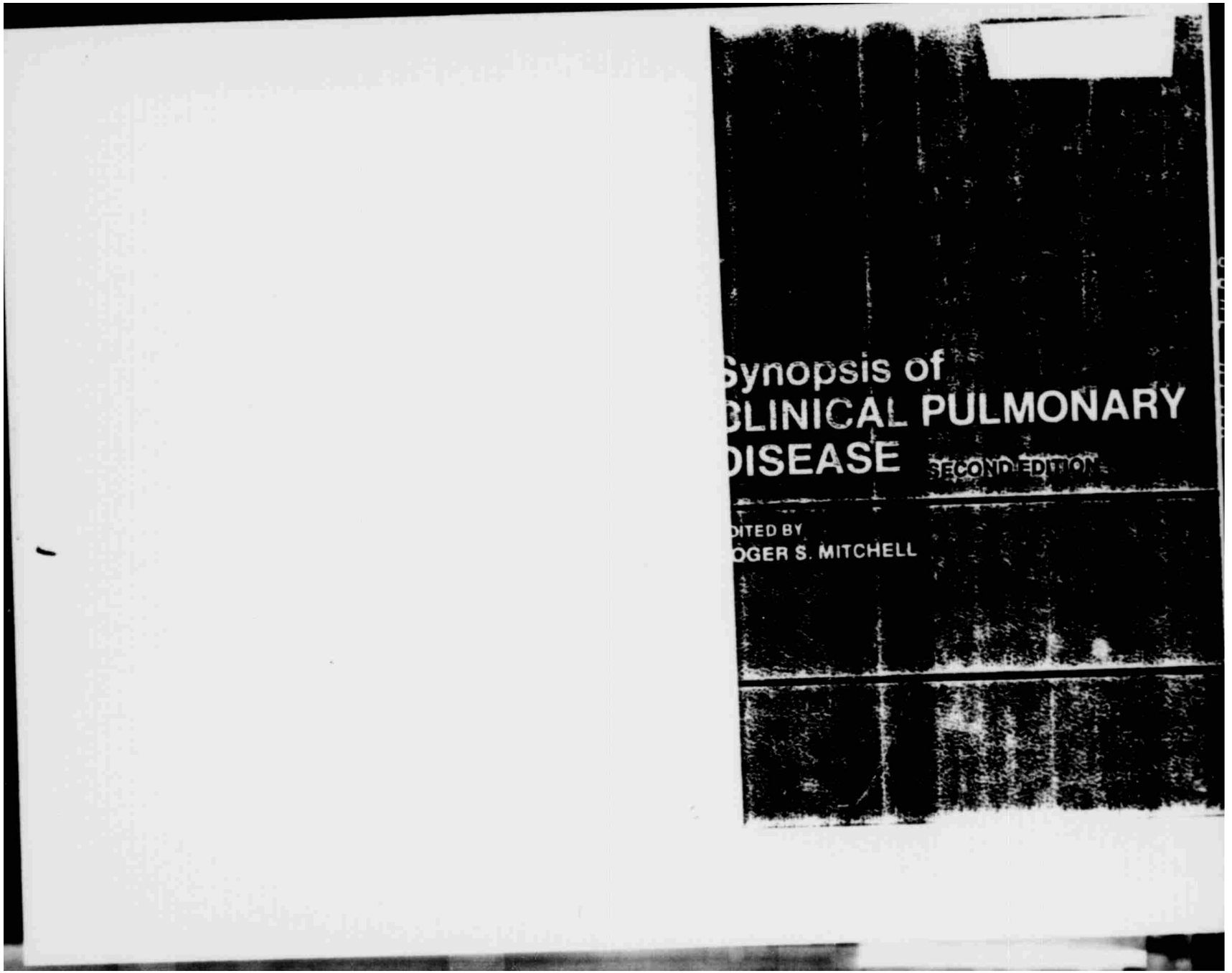
The 1981 policy concerning publications is to publish "state of the art" reports in CARDIOPULMONARY MEDICINE, the bulletin of ACCP. We contemplate publication of national guidelines in either CARDIOPULMONARY MEDICINE or CHEST. The latter, -- however, would have to be uniquely new authoritative guidelines rather than review material. Would it be accurate to say that this report is intended as a current review more than the establishment of new guidelines? The report is single-spaced and I assume that the final version would be double-spaced. The length is very worrisome in view of our current critical shortage of page space. Would revision include a more concise report?

Please know that this letter is intended as a plea for more information. In no way do I wish to be arbitrary since I respect your position as a consultant and author and I am desperately eager to assist the Committee on Environmental Health. I hope that this opens a dialogue which will result in a superb published communication.

Sincerely yours,

Alfred Soffer, M.D., F.C.C.P.
Editor-in-Chief
Professor of Medicine

cc: Bernie Gee, M.D.





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# Foreword

Pulmonary medicine has come a long way in the past 20 years! It seems incredible that exactly 2 decades ago as a senior medical student I chose an elective in pulmonary medicine with Dr. Mitchell. Very clearly, as I look back, this was the major force that kindled my interest in the fascinating field of pulmonary medicine. Since that time many medical students have sought elective training in pulmonary medicine at the University of Colorado Medical Center. Currently over 50% of our graduating class and many students from other medical schools choose our elective in the senior year. Hopefully they all become "turned on" to the delights of the practice and science of pulmonary disease!

Thus, members of the Division have been particularly dedicated to the task of revising chapters from the first edition of this synopsis, or writing new chapters, with hopes that collectively we may be able to spark the imagination of many more undergraduate physicians, house officers, and our colleagues in the allied health professions who will be tomorrow's servants for patients with diseases of the respiratory system.

I personally thank my former chief for his continued efforts in medical education in our field and particularly for the effort of this synopsis and all it means.

**Thomas L. Petty**




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# Preface

The first edition of this introduction to the intricacies of chest medicine published in 1974 was sufficiently successful to prompt an update. In response to suggestions, the present volume goes into more detail, adds many illustrations, including both chest films and tabular material, and updates and expands the bibliographies.

As before, the intended audience for this pocket-sized volume includes both undergraduate and postgraduate medical students and practitioners other than the pulmonary specialist.

All but one of the contributors are members of the faculty of the University of Colorado School of Medicine, and all but one, a surgeon, are members of the Division of Pulmonary Sciences, Department of Medicine, to which all proceeds derived from the sale of this volume will again be donated.

Roger S. Mitchell



*Cardiopulmonary Journal Correspondence on the ACCP Report on Environmental Pulmonary Carcinogens (1981)*
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# 8

# Respiratory failure

Thomas M. Hyers

Respiratory failure occurs when the respiratory system cannot supply adequate oxygen to maintain metabolism and/or cannot eliminate sufficient carbon dioxide to avoid respiratory acidosis. This definition emphasizes that respiratory failure always causes other organ dysfunction as well. Clinically, respiratory failure is recognized when the patient's $Pa_{O_2}$ is less than 50 mm Hg or when the $Pa_{CO_2}$ is greater than 50 mm Hg with associated symptomatic abnormalities.

## Pathophysiology

Alveolar gas pressure or tension is formed by the sum of the partial pressures of nitrogen, oxygen, carbon dioxide, and water vapor. Since total alveolar gas pressure and the partial pressures of nitrogen and water vapor are fixed by the surrounding atmosphere, the alveolar partial pressures of oxygen and carbon dioxide tend to vary inversely. This simplified version of the alveolar gas equation shows the inverse relationship:

$$PA_{O_2} = \text{inspired } P_{O_2} - \frac{PA_{CO_2}}{\text{respiratory quotient}}$$

The respiratory quotient is the ratio of $CO_2$ production divided by $O_2$ uptake and is determined by tissue metabolism in the steady state. The ratio usually takes a value of approximately 0.8, which means that an increase in $P_{CO_2}$ will result in a slightly smaller decrease in $P_{O_2}$. Since carbon dioxide is highly soluble in liquids and freely diffusible, alveolar and arterial tensions of this gas are nearly always equal. Therefore, when $Pa_{CO_2}$ rises, the cause is a widespread inability to ventilate alveoli. This state is called *diffuse alveolar hypoventilation* and is the only important cause of an elevated $Pa_{CO_2}$.




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Diffuse alveolar hypoventilation can occur in intrinsic lung diseases (usually COPD or chronic bronchitis and emphysema), in disorders of the neuromusculature and chest wall, and with abnormalities of central ventilatory drive as described below. From the alveolar gas equation it is clear that when $P_{CO_2}$ rises, $P_{O_2}$ must fall unless oxygen is added to the inspired air.

The transport of oxygen is more complicated than carbon dioxide elimination. Oxygen is both less soluble in liquids and less diffusible than carbon dioxide, which means that $Pa_{O_2}$ will always be 10 to 20 mm Hg less than $PA_{O_2}$ even in normal lungs. In abnormal lungs the *gradient* between $PA_{O_2}$ and $Pa_{O_2}$ can be much larger. The most common disorder that enlarges this gradient is seen in COPD where regions of decreased alveolar ventilation do not keep pace with capillary blood flow (ventilation-perfusion disturbance or mismatch). The extreme example of ventilation-perfusion mismatch is *shunt*, which is a region of no alveolar ventilation without impairment of blood flow. Shunt is seen often in pneumonia and pulmonary embolism. The third disorder that can cause an enlarged gradient and hypoxemia is impaired *diffusion*, which results when the alveolar-capillary distance is widened. This problem is often encountered in the interstitial lung diseases. Thus, hypoxemia may occur in ventilation-perfusion mismatch, shunt, impaired diffusion, and, of course, in diffuse alveolar hypoventilation.

Minute ventilation (tidal volume × respiratory rate) is regulated by neurologic, metabolic, and chemoreceptor input. Of these factors, chemoreceptor input is best understood and is comprised of a $Pa_{O_2}$ sensor in the carotid body and $Pa_{CO_2}$ sensors in the carotid body and the brainstem. Either a fall in $Pa_{O_2}$ or a rise in $Pa_{CO_2}$ can stimulate ventilation in the normal individual. In a few people, abnormalities in this central ventilatory drive mechanism may lead to diffuse alveolar hypoventilation, with both elevated $Pa_{CO_2}$ and decreased $Pa_{O_2}$.

Low oxygen tension or elevated carbon dioxide tension may cause increased pulmonary artery pressure by a direct constriction of pulmonary arterioles. The utility of this reflex in adult life is unclear, and it may simply be a vestige of a fetal reflex. Chronic elevation of pulmonary artery pressure can eventually cause right ventricular failure analogous to the way systemic hypertension causes left



**CAUSES OF RESPIRATORY FAILURE**

A. Intrinsic lung diseases
   1. COPD (e.g., chronic bronchitis and emphysema)
   2. Interstitial lung diseases
   3. Infections
   4. Inhalation of toxic gases
   5. Drowning
   6. Chest trauma
B. Neuromuscular and chest wall
   1. Neuromuscular blocking agents (e.g., curare)
   2. Cervical spinal cord injuries
   3. Diseases (e.g., myasthenia gravis, amyotrophic lateral sclerosis, myotonic dystrophy, Guillain-Barré syndrome, poliomyelitis, botulism)
   4. Kyphoscoliosis
C. Central ventilatory drive
   1. Drug overdose (e.g., opiates, barbiturates, tranquilizers, alcohol)
   2. Obesity-related hypoventilation (pickwickian syndrome)
   3. Myxedema
   4. Stroke, brain trauma
D. Other causes
   1. Carbon monoxide inhalation
   2. Upper airways obstruction (e.g., foreign body, tumor, micrognathia)

ventricular failure. This state is called *cor pulmonale*, that is, heart failure caused by lung disease.

### Causes

Intrinsic diseases of the lung, disturbances of the chest wall or neuromusculature, and disorders of the central ventilatory drive can all lead to respiratory failure (see the above outline).

Of the intrinsic lung diseases, COPD is easily the most frequent cause of respiratory failure. Severe viral and bacterial pneumonias, interstitial lung diseases, drowning, and inhalation of toxic substances such as $NO_2$ can also be causative. In this setting the chest wall and central ventilatory drive are usually normal; the lung


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architecture itself is the deterrent to effective oxygen transport (see preceding chapter). Some of these causes of respiratory failure can be either acute or chronic, and the degree of *renal* bicarbonate retention in compensation for any respiratory acidosis present ($Pa_{CO_2}$ elevation) often furnishes the clue to chronicity (Table 8-1).

Distubances of the chest wall or neuromusculature are less frequent than intrinsic lung diseases, but important to recognize. Acute causes include neuromuscular blocking agents used in anesthesia; more chronic causes are cervical spinal cord injuries, myasthenia gravis, Guillain-Barré syndrome, amyotrophic lateral sclerosis, myotonic dystrophy, and, rarely, poliomyelitis and botulism.

Central ventilatory drive can be suppressed acutely by overdoses of opiates, barbiturates, or tranquilizers. Stroke and brain trauma can also cause respiratory failure by suppressing central ventilatory drive, and some individuals seem to have a genetic insensitivity to elevated $Pa_{CO_2}$ and lowered $Pa_{O_2}$. Obesity-related hypoventilation is a well-known cause of chronic respiratory failure in this group.

Inhalation of carbon monoxide causes a unique form of respiratory failure by inhibiting the access of oxygen to hemoglobin. Since only the binding of oxygen to hemoglobin is affected, the $Pa_{O_2}$ can be normal even with massive carbon monoxide inhalation. Thus, the problem can be difficult to recognize and must be suspected in any type of smoke inhalation. The treatment is high-flow oxygen supplementation that speeds the clearance of carbon monoxide from the blood.

**Table 8-1.** Blood gas patterns in respiratory failure

| | $Pa_{O_2}$ | $Pa_{CO_2}$ | pH | $HCO_3$ (meq/liter) |
|---|---|---|---|---|
| Acute hypoxemia | ≤50 | ≤40 | 7.44* | 24 |
| Acute hypoxemia and hypercapnia | ≤50 | ≥50 | ≤7.30 | 24 |
| Chronic hypoxemia and hypercapnia, with renal compensation | ≤50 | ≥50 | 7.38 | ≥30 |

*Approximate



## Diagnosis

No matter what the cause, respiratory failure can be difficult to recognize. Symptoms include altered mentation, malaise, headache, weakness, weight loss or gain, palpitations, breathlessness, and cough—all deceptively nonspecific. Cardiovascular manifestations include tachycardia, arrhythmias, cyanosis, ascites, and edema. Central nervous system manifestations vary from mild personality changes to stupor and coma. When respiratory failure is suspected, arterial blood gases and pH must be measured immediately to confirm the diagnosis.

## Treatment

Acute respiratory failure can often be a medical emergency and two priorities of therapy must be committed to memory. Adequate oxygenation is always the first priority. Oxygen can be administered by nasal prongs or by a tight-fitting mask with attached reservoir bag if manual assistance in breathing is required. These methods are usually adequate until the arrival of someone trained to perform endotracheal intubation.

The second priority is reversal of respiratory acidosis. Carbon dioxide retention, signifying diffuse alveolar hypoventilation, frequently accompanies acute respiratory failure and is quickly corrected by increasing alveolar ventilation. Rarely is administration of intravenous $NaHCO_3$ necessary to correct respiratory acidosis, and in some instances $NaHCO_3$ may be detrimental by overloading an already compromised cardiovascular system. Thus, the proper initial therapy of acute respiratory failure is maintenance of an adequate airway, supplemental oxygen, and assisted ventilation as necessary to correct $Pa_{O_2}$ and acid-base abnormalities.

Proper treatment ultimately depends on correctly identifying the underlying cause. With this knowledge in mind, the clinician selectively treats infection if present, administers antidotes as available to counteract ingested drugs or toxins, initiates controlled oxygen therapy, and begins chest physiotherapy to assist in the removal of obstructing secretions.

The use of intermittent positive pressure breathing (IPPB) lacks proven benefit in acute respiratory failure. In general, inhaled drugs can be nebulized as effectively with powered but nonpressurized


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nebulizers. Furthermore, IPPB frequently can be tiring to a patient and can lead to pneumothorax and air swallowing with massive gastric distension.

The decision to perform endotracheal intubation and initiate mechanical ventilation is based both on clinical evaluation and on blood gas abnormalities. Many individuals with respiratory failure caused by COPD may be treated with judicious oxygen therapy, chest physiotherapy, bronchodilators, and antibiotics without resorting to intubation and mechanical ventilation, although arterial blood gases interpreted alone might dictate otherwise. In contrast, some individuals with massive pneumonia or drug overdose require immediate mechanical ventilation. General indications for endotracheal intubation and mechanical ventilation in acute respiratory failure include (1) unconsciousness, (2) copious secretions that cannot be cleared by coughing, and (3) severe hypoxemia or acidosis that does not respond promptly to lesser measures.

A hospital in which endotracheal intubation and mechanical ventilation are performed must have an adequately staffed intensive care unit, a physician skilled in the care of the critically ill and in the use of mechanical ventilators, and a laboratory capable of accurate and immediate arterial blood gas analysis. Endotracheal intubation must be performed by an experienced physician and the position of the tube checked with a portable chest radiograph. The end of the tube should be 3 to 4 cm above the carina. A prestretched, high compliance cuff is important in avoiding extensive ischemia to the tracheal mucosa and cartilaginous rings. The internal diameter of the tube must be large enough (≥7½ mm) to allow easy passage of a suction catheter. In general, endotracheal tubes are left in place from 3 to 10 days, after which a tracheostomy is performed if intubation is still necessary.

*Mechanical ventilators* used today are mostly of the *volume* delivery type, that is, they will deliver a preset volume of gas limited only by a very high airway pressure (60 to 100 cm $H_2O$) that, upon being reached, opens a safety valve. Some ventilators are *pressure* regulated and deliver gas only until a preselected pressure of 20 to 30 cm $H_2O$ is reached. Pressure-regulated ventilators are subject to the vagaries of pulmonary compliance in that as lung compliance decreases because of pulmonary edema, pneumonia, or fibrosis,



**CHECKLIST FOR ENDOTRACHEAL INTUBATION AND MECHANICAL VENTILATOR THERAPY**

A. Endotracheal tube
   1. Type—7½ mm or greater internal diameter with prestretched high compliance cuff (cuff pressure < 30 mm Hg)
   2. Position—check with portable chest radiograph
B. Mechanical ventilator
   1. Tidal volume—12 to 15 ml/kg body weight
   2. Rate—as set by the patient or to maintain the $Pa_{O_2}$ and pH listed below (usually a minute ventilation of 5 to 10 liters is sufficient)
   3. Alarm—in-line, pressure or flow sensitive, continuous use
C. Patient
   1. Arterial blood gases—$Pa_{O_2}$: 60 to 90 mm Hg; pH: 7.35 to 7.50
   2. Vital signs—frequent monitoring aimed at normalizing (fast pulse and low blood pressure can be caused by a pneumothorax or a large tidal volume interfering with venous return)
   3. Cardiac rhythm—should be continuously monitored
   4. End organ function—especially mentation, urine output

delivered tidal volume also decreases. All ventilators must be used with an in-line audible alarm system sensitive to changes in airway pressure or flow. Large tidal volumes are used (12 to 15 ml/kg body weight) to help prevent atelectasis, and when possible the machine is adjusted so that the patient's breathing effort will trigger the machine. When a patient is apneic, the rate is set to achieve a $Pa_{O_2}$ between 60 and 90 mm Hg and a pH between 7.35 and 7.50. A minute ventilation between 5 and 10 liters will usually approach this pH and oxygen tension. Because of the physical characteristics of oxygen-hemoglobin affinity, a $Pa_{O_2}$ higher than 90 mm Hg does not result in an appreciable increase in blood oxygen content but can cause lung damage from hyperoxia. In all instances, oxygen tension and acid-base status must be monitored by repeated arterial blood gas determinations. Other maneuvers may be necessary. These include sedation, the addition of respiratory dead space if the patient is markedly hyperventilating, and the use of PEEP in certain types of ARDS when oxygenation is difficult (see preceding chapter).


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Adequate oxygen supply to tissues is the goal, remembering that tissue oxygenation is dependent on both blood oxygen content and cardiac output. Therefore, careful attention must be paid to pulse, blood pressure, and cardiac rhythm during mechanical ventilation. In some instances determination of cardiac output is necessary.

Nosocomial (hospital-acquired) pneumonia is the primary complication of acute respiratory failure. This problem is caused not only by the patient's weakened condition, but also by the common use of antibiotics in high dosages in the intensive care unit.

The direct complications of intubation and mechanical ventilation are iatrogenic and can be lethal. These include inadvertent intubation of the right mainstem bronchus; respiratory acidosis or alkalosis caused by improper use of the ventilator; pneumothorax; and tracheal damage and stenosis.

### Weaning procedures

Endotracheal intubation and mechanical ventilation cannot be viewed as specific therapy for respiratory failure but rather as support until reversible abnormalities, e.g., pneumonia, bronchitis, pulmonary edema, are resolved. Once the patient appears to be responding to treatment for the underlying cause of respiratory failure, the following criteria should be met before discontinuing ventilatory support: (1) adequate arterial oxygen (a $Pa_{O_2}$ above 60 mm Hg with an $FI_{O_2}$ of 0.4 or less), (2) a spontaneous minute ventilation less that 10 liters as measured with a Wright respirometer, (3) a maximal voluntary ventilation at least twice the resting minute ventilation, and (4) a vital capacity at least 2 to 3 times the resting tidal volume. When these criteria are met, the ventilator may be removed and spontaneous breathing allowed through the endotracheal tube. The $FI_{O_2}$ should be the same or slightly higher than that delivered with the ventilator and adequate humidity must be added to the inspired air. The patient must remain in the intensive care unit for close observation. If alveolar ventilation decreases (i.e., $Pa_{CO_2}$ rises) or other signs of respiratory failure appear (restlessness, tachycardia, diaphoresis, confusion), ventilation is started again and weaning postponed. If the patient remains stable and arterial blood gases remain satisfactory for more than 30 minutes, the endotracheal tube can usually be safely removed.



### Chronic respiratory failure

COPD is also the principal cause of chronic respiratory failure. Less common causes include neuromuscular disorders and obesity-related dysfunction of central ventilatory drive. In chronic respiratory failure, the work of breathing can become a significant part of total body metabolism because of inefficiency of the respiratory system. The patient tends to respond to this marginal situation either by decreasing ventilation relative to metabolic demand to allow a higher blood $Pa_{CO_2}$ and a lower $Pa_{O_2}$ or by shedding lean body mass. Both responses can be considered compensatory because each leads to decreased work of breathing relative to the metabolic demands of the body. The former compensation is seen typically in the so-called "blue bloater" and the latter in the "pink puffer" COPD patient. The basis for selection of one of these responses is not clear but may be dependent in part on a genetic predisposition. Neither response is completely compensatory and each leads to further problems. The "blue bloater" hypoventilates with a resultant respiratory acidosis and more hypoxemia, both of which increase pulmonary hypertension hastening the onset of cor pulmonale. The "pink puffer" frequently develops cachexia severe enough to suggest neoplastic disease.

The administration of supplemental oxygen has potential hazard in the individual with chronic respiratory failure and elevated $Pa_{CO_2}$. The chemoreceptor drive to breathe is directed by hypoxemia here, and the sudden introduction of high-flow oxygen can cause increased hypoventilation or even apnea by suppressing the hypoxemic drive. In practice this is rarely seen with nasal oxygen flows of 3 liters/minute or less, and these flow rates are usually sufficient to raise the $Pa_{O_2}$ to an acceptable level. When someone with chronic respiratory failure is given ventilatory therapy one must avoid the temptation to correct an elevated $Pa_{CO_2}$ when the pH is nearly normal. In these patients the pH approaches normal because of previous renal conservation of bicarbonate ion, and a rapid lowering of $Pa_{CO_2}$ can lead to severe metabolic alkalosis with seizures, hypotension, and arrhythmias. In fact, it is usually better to ventilate to the $Pa_{CO_2}$ to which the patient has previously compensated the pH with bicarbonate ion retention.

In patients with chronic respiratory failure and cor pulmonale,


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the administration of continuous low-flow (1 to 2 liters/minute) nasal oxygen can frequently correct or alleviate pulmonary artery hypertension, correct or prevent congestive right heart failure, and return the patient to a more functional life.

## SUGGESTED READINGS

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# 10

# Chronic obstructive pulmonary disease

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## Definitions

*Chronic obstructive pulmonary disease* (COPD) is a clinical state of dyspnea on exertion with objective evidence of reduced airflow, not explained by specific or infiltrative lung or heart disease. Reduced airflow is detected on physical examination by diminution in the sounds of air movement, especially on expiration, throughout both lungs. Reduced airflow may be measured by the standard ventilation tests (MVV, $FEV_1$, MMEF) or by measuring increased airways resistance (in the body box). Synonyms include chronic obstructive pulmonary emphysema (COPE), chronic obstructive lung disease (COLD), chronic airways obstruction (CAO), diffuse obstructive pulmonary syndrome (DOPS), emphysema, chronic bronchitis, and others.

*Emphysema* is defined morphologically as a state of the lungs in which the air spaces distal to the terminal bronchioles are abnormally increased in size and undergo destructive changes in their walls. The Ciba Symposium in 1958 took the position that increase in the size of the air spaces may result either from dilatation or destruction; in other words, in their view overinflation alone (i.e., dilatation without destruction as in one lung after removal of the other) may be regarded as emphysema. The view accepted by most observers, however, is that overinflation alone should be labeled simply as overinflation and not as emphysema.

*Chronic bronchitis* is defined clinically: it is characterized by excessive mucus secretion in the bronchial tree, with chronic or recurrent productive cough on most days for a minimum of 3 months




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a year for not less than 2 successive years. The diagnosis can only be made after excluding the presence of specific diseases of the lungs or bronchi such as tuberculosis, abscess, tumor, bronchiectasis, and others that may cause identical symptoms.

### Pathology and physiology

*COPD* is almost always a mixture of emphysema and chronic bronchitis, plus, at times, elements of bronchiolitis and asthma. *Emphysema* has been classified as *centrilobular* (CLE) or *centriacinar* involving initially the respiratory bronchioles, *panlobular* (PLE) or *panacinar* involving the entire acinus, *paraseptal*, and *irregular* or "scar" emphysema. As CLE progresses, it becomes increasingly difficult to distinguish it from PLE and it may be labeled *mixed* or *endstage*. CLE is predominantly a disease of bronchitic patients who smoke cigarettes, whereas PLE, the type associated with $\alpha_1$-antitrypsin deficiency (zz type), is also often found in bronchitic patients who smoke cigarettes. The airway tissues, especially the cartilaginous tissue, are often atrophic, resulting in flabbiness and premature collapse on expiration. Physiologically, advanced emphysema is most commonly identifiable by loss of elastic recoil, increased total lung capacity, and impaired $O_2$ diffusion. The impaired diffusion tends to be balanced by the destruction of the capillary bed so that ventilation-perfusion (V/Q) imbalance is slight or absent.

*Chronic bronchitis* is characterized morphologically by hypertrophy and hyperplasia of the mucous glands lining the bronchi, chronic leucocytic and lymphocytic infiltration of the bronchial walls, increase in goblet cells, loss of cilia, and squamous metaplasia. These changes often extend to the noncartilage-bearing bronchioles that may be almost or totally occluded by inflammation, exudate, or dried mucus plugs (as in bronchiolitis and asthma).

In normal lungs most of the resistance to airflow lies in airways more than 2 mm in diameter, because the total cross sectional diameter of the smaller ones is so much greater than those larger than 2 mm in diameter. Thus, involvement of the small airways must be extensive before conventional tests of expiratory airflow (MVV, $FEV_1$, MMEF) will be abnormal. As chronic bronchitis progresses to severe obstruction, ventilation becomes severely impaired while



perfusion is not altered very much; this produces a V/Q imbalance with hypercapnia and especially hypoxemia. The latter then gradually produces pulmonary hypertension, cor pulmonale, and episodes of right heart failure.

COPD in its advanced stages sometimes presents as "pink puffers"—intense dyspnea, marked weight loss, hyperlucent lungs, small heart, and a tendency to normal blood gases—and "blue bloaters"—less dyspnea, no weight loss, large hearts, episodes of right heart failure, erythremia, hypercapnia, and hypoxemia. The majority of cases tend to fall between these two extremes of the clinical spectrum. Untreated, the "pink puffer" tends to show severe emphysema and relatively little airway disease; the "blue bloater" tends to show severe airway diseases and less emphysema.

COPD is much more common in men than in women and in smokers than in nonsmokers. It is also more common in urban than in rural dwellers and in the socioeconomically disadvantaged than in the economically privileged classes. Cigarette smoking is probably the most important known factor in the pathogenesis of COPD, whether it is manifested by emphysema, chronic bronchitis, or both. Cigarette smoke contains numerous irritants that stimulate mucus production, impair and ultimately destroy ciliary function, induce cough, and ultimately cause alveolar wall damage. Persons who regularly smoke cigarettes are also more susceptible to recurrent deep respiratory infections, which appear to play an important contributory role in the evolution of COPD. Cigarette smoke contains a high concentration of carbon monoxide, which can aggravate the hypoxemia that is a frequent complicating problem late in the course of the disease, especially after the appearance of congestive right heart failure.

Urban air pollution plays a role in COPD similar to that of cigarette smoke but to a less striking degree. Severe chronic cough may cause further damage to already partially damaged bronchial, bronchiolar, and alveolar walls. This opinion is supported by the clinical observation that progression of disability tends to slow down or ceases when coughing is controlled in the not too severely ill patient. Microscopic pulmonary thromboemboli, possibly induced by nicotine, carbon monoxide, or other ingredients of cigarette smoke, may play a role in causing alveolar wall damage.


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Finally, certain tissue defects and increased susceptibility to injury appear to be involved in the pathogenesis of COPD. How else can it be explained that some people can live in an industrial and heavily polluted city and inhale cigarette smoke regularly all their lives and still not develop the severe symptomatic disease? These defects may be acquired or genetically determined. A small number with deficiency of $\alpha_1$-antitrypsin (zz type) tend to have early onset panlobular emphysema. Patients with heterozygous deficiency who also smoke cigarettes may have a higher than expected risk of emphysema and COPD. The mechanism of this form of alveolar damage appears to be a failure of protection against protein digestion from enzymes released from disintegrating macrophages and leukocytes. A forme fruste of mucoviscidosis in the adult has been proposed as an underlying tissue defect by some observers. Airway clearance becomes severely impaired in advanced COPD; healthy young persons and persons with mild disease have a spectrum of airway clearance from rapid to slow; it is thus suggested that impaired clearance, genetic or acquired, may also be an important predilection factor.

The mechanisms of airways obstruction are somewhat better understood than the etiology. These mechanisms include inflammatory swelling of the bronchial and bronchiolar walls causing them to narrow; the presence of excessive and viscid mucus obstructing airflow, especially in the bronchioles; atrophy of airway walls, presumably a complication of prior inflammation, together with loss of the normal supporting elastic structure and elastic recoil (i.e., emphysema) causing them to kink and collapse prematurely on expiration; and many bronchioles are simply no longer patent because of prior obliterative inflammation.

### Clinical picture

COPD at times appears to have its origin in repeated chest infections in childhood. About two thirds of cases however, begin in individuals in their 30s or 40s with chronic productive cough and repeated colds or deep chest infections. Approximately 15% of patients give a history of episodic, noneffort-dependent, wheezing dyspnea (i.e., true bronchial asthma) generally during childhood, before the onset of effort-dependent dyspnea. Some 20% of patients



insist that dyspnea on exertion was their first symptom, but when closely questioned, most of these add that chronic cough started at the same time or soon thereafter. Some 5% of patients deny the presence of chronic cough at any time throughout the course of the disease. A few patients begin with a sudden awareness of dyspnea and cough; others report a sudden and persistent worsening of their dyspnea at some time during the course of the disease. These episodes, sometimes devastating to exercise tolerance, are often labeled *pneumonia* by the attending physician. They often are no doubt episodes of pneumonia, but some may be episodes of acute bronchiolar inflammation, which may become chronic or result in irreversible obliteration of many bronchioles; some may be episodes of extensive microscopic thromboembolism. Most such episodes probably represent an acute awareness of a little more damage to an already-damaged respiratory system. It has been suggested that progressive alveolar destruction provides collateral ventilation distal to simultaneously occurring bronchiolar obliteration, in other words, a temporary advantage from some emphysema. A considerable amount of disease may be present with little subjective dyspnea and no or only mild airway obstruction so that a superimposed deep respiratory infection may be enough to make a subject abruptly and permanently symptomatic.

The appearance of the sputum is important in the evaluation of patients with COPD. When it is purulent, a readily treatable and potentially reversible aspect of the disease has been identified.

The physical findings vary with the type and severity of the obstruction. A relatively immobile chest with shoulders carried high, suppressed breath sounds especially on expiration, and decreased cardiac dullness are common findings in all severe cases. An apparent increase in the anteroposterior diameter, scattered rhonchi, occasionally a few fine basal rales, accentuated second heart sound in the pulmonic area, palpable $P_2$ in the second left intercostal space, gallop rhythm, right ventricular lift, cyanosis, and dependent edema may all be observed.

The chest radiograph may be helpful when properly interpreted (Figs. 10-1 to 10-4). Radiographic signs suggesting emphysema are flattening of the hemidiaphragms, enlargement and avascularity of the lungs, presence of bullae, and increased retro-

*Text continued on p. 110.*


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Fig. 10-1. COPD with diffuse emphysema, severe. **A**, Posteroanterior: note right hemidiaphragm at seventh anterior *rib*. *No* pneumothorax is present despite absence of lung markings. **B**, Lateral. (Courtesy Medical Illustration Service, Veterans Administration Hospital, Denver, Colorado.)



Fig. 10-1, cont'd. For legend see opposite page.



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Fig. 10-2. COPD with predominant chronic bronchitis and cor pulmonale. A, Posteroanterior. Note huge pulmonary arteries. B, Lateral. (Courtesy Medical Illustration Service, Veterans Administration Hospital, Denver, Colorado.)





Fig. 10-2, cont'd. For legend see opposite page.


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Fig. 10-3. Bronchiolitis manifested by scattered, nonuniform, fine, "soft" nodulation. A complication of severe COPD and asthma. (Courtesy Medical Illustration Service, Veterans Administration Hospital, Denver, Colorado.)



Fig. 10-4. Bullous emphysema, bilateral, left more than right. (Courtesy Medical Illustration Service, Veterans Administration Hospital, Denver, Colorado.)



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sternal space. Indications of chronic bronchitis include increased bronchovascular markings, increase in the size of the main and branch pulmonary arteries, and an enlarged heart. Chest fluoroscopy may be useful in estimating the speed of expiratory airflow; this dynamic measure of air trapping cannot be reliably predicted from an inspiratory film, not even when accompanied by an expira[illegible] film since some patients can empty their lungs if given enough t[illegible] Complicating bronchiolitis may be manifest radiographically by a fine stippling in both midlung fields (Fig. 10-3). A bronchogram, if performed, may reveal bits of contrast medium in dilated bronchial mucous gland ducts, especially under the left main bronchus, as well as a failure of the bronchi to taper peripherally. In centrilobular emphysema, contrast medium may pool peripherally and look like scattered "leaves on a winter tree," a finding that has been labeled *bronchiolectasis* (Fig. 10-5). The bronchogram should be used with great care however (i.e., including one side at a time) in patients with severe airway obstruction; the information provided is of academic interest rather than practical value.

Bronchoscopy in severe chronic airway obstruction will show easy collapsibility of all the visible major airways including the trachea. In chronic bronchitis, the mucosa has a red velvety appearance and secretions may be seen bubbling up from the involved airways.

The electrocardiogram is useful in detecting the presence of cor pulmonale and pulmonary hypertension. Various findings are associated with right ventricular enlargement as follows:

1. Right axis deviation (most common)
2. Terminal forces rightward, inferiorly and anteriorly directed (most reliable)
3. Tall 0.04 sec R in $V_3R$ or $V_1$
4. R/S ratio $V_1$ = >1; R/S ratio $V_6$ = <1
5. rsR' or rSR', in $V_3R$ with R' >5 mm or >½ of S
6. RAD of terminal forces with shallow notched $sV_1$ or $rSr' \cdot V_1$ and prominent $SV_{5,6}$ (early RVH)
7. $S_1$, $S_2$, $S_3$ syndrome
8. R in a VR >5 mm
9. With RBBB
   a. RAD of unblocked QRS forces
   b. R'$V_1$ >15 mm





**Fig. 10-5. Bronchiolectasis—"leaves on a winter tree"—a bronchographic indication of centrilobular emphysema. (Courtesy Medical Illustration Service, Veterans Administration Hospital, Denver, Colorado.)**

An elevated hematocrit is useful as an indicator of secondary erythremia and chronic hypoxemia.

The course of COPD is punctuated by exacerbations, usually caused by infections, also by noxious fumes and trauma, but sometimes apparently unrelated to any identifiable event. When an exacerbation is complicated by right heart failure, the outlook is likely thereafter to be poor. Progressive weight loss is also a poor prognostic sign. The causes of death in COPD are respiratory failure, congestive heart failure secondary to cor pulmonale, pneumonia, bronchiolitis, pulmonary thromboembolism, perforation or hem-


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orrhage of a peptic ulcer, and spontaneous pneumothorax, in approximately that order of frequency.

### Differential diagnosis

The differential diagnosis of COPD should include other causes of congestive heart failure, other causes of erythremia, and the various specific causes of chronic cough and expectoration such as tuberculosis, bronchiectasis, and fungus diseases.

One unusual cause of chronic cough and, at times, of recurrent patchy pneumonia, is reflux of usually acidic stomach contents into an esophagus that has impaired motility with resultant recurrent aspiration of irritant material, usually at night. That postpoliomyelitic or encephalitic impairment of pharyngeal constrictors also plays a role in this syndrome has been suspected (personal observations).

### Treatment*

The backbone of the treatment of COPD is the regular daily use of bronchial hygiene. This consists of the inhalation of nebulized (heated or room temperature) water or steam, preceded by the inhalation of a nebulized bronchodilator drug (e.g., isoproterenol 1:200 dilution, racemic epinephrine, metaproterenol, or isoetharine, diluted approximately 2:1 with water) and followed by postural drainage, usually aided by an assistant (e.g., the spouse) thumping the chest with the cupped hands. One treatment should consist of approximately 5 drops of bronchodilator diluted with water, followed by 10 to 15 minutes of steam or nebulized water inhalation. Such a treatment should be taken from 1 to 3 times a day. The bronchodilator may be aerosolized with a hand-bulb nebulizer, with a nebulizer powered by an electric pump, by air or oxygen under pressure, with a pump, or with a gas-activated intermittent positive pressure device.

These patients should avoid persons with acute respiratory infections and should therefore avoid crowds, especially in winter. They should rest at home, stay away from work, and force fluids for even mild acute upper respiratory infections.

*See also Chapter 9.



Empiric antimicrobial therapy should be given as soon as a respiratory infection has descended to the chest, as evidenced by the production of yellow, green or gray (i.e., obviously purulent) sputum. Infectious flare-ups are caused in most cases by *Diplococcus pneumoniae* and/or *Hacmophilus influenzae*. Tetracycline, 2 gm daily for 2 days, then 1 gm daily for 4 or 5 days, or a combination of Trimethoprin-sulfamethoxazole is indicated initially. Ampicillin in double the tetracycline dosage is a useful alterative. Erythromycin may be used in patients with penicillin allergy in the dosage recommended for tetracycline. A trial of streptomycin, 0.5 to 1 gm intramuscularly per day, may also be indicated. Sputum culture is generally helpful only after failure of empiric treatment. Only one antimicrobial agent should be used at a time in order not to suppress normal flora and thus promote the overgrowth of drug-resistant bacteria, fungi, or molds.

Patients who improve on antimicrobial treatment and then promptly relapse will often benefit from the continuous daily preventive use of 0.5 gm tetracycline or erythromycin or 1 gm ampicillin for weeks or months at a time.

Polyvalent influenza vaccine should be given in fall and winter.

These patients should not smoke anything, especially cigarettes. If they cannot stop smoking, they should try not to inhale smoke. Some patients can be persuaded to stop smoking by switching to snuff, or chewing cigars or tobacco.

Voluntary effort to resist the desire to cough except when it is productive is important. Cough-suppressant drugs are seldom necessary or wise, but they may be used judiciously when all other measures have failed.

Nebulized detergents are not recommended. So-called expectorant drugs (e.g., potassium iodide) are seldom needed. Water, by mouth and by inhalation, is the best expectorant.

Oral bronchodilators such as capsules containing ephedrine, 24 mg, with phenobarbital, 8 mg 3 times a day, may be useful. Terbutaline sulfate is a recent addition to this group. Theophylline and its derivatives may also be useful by mouth.

The measured-dose aerosol cartridge may be used when patients are away from home. They should be cautioned not to use it more than a few times a day because of the risk of cardiac arrhythmia.


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Aminophylline, 500 mg in 500 ml of 5% dextrose given slowly intravenously (i.e., approximately 40 to 50 drops a minute), as often as every 4 hours if needed, is a useful bronchodilator for the acutely ill patient in the hospital. Aminophylline rectal suppositories or enemas may also be helpful in treating acute dyspnea; the $Pa_{O_2}$ should be followed closely as V/Q relationships change with bronchodilators and hypoxemia may be worsened.

Corticosteroid drugs (preferably prednisone by mouth or hydrocortisone by vein) are probably the most effective "bronchodilator" drugs available. Their action is somewhat delayed, which is thought to be caused by (1) relief of mucosal congestion, (2) production of thinner mucus, and (3) shrinking of mucous glands with lesser volumes of mucus produced. Patients who are believed to have a major reversible component to their airway obstruction or who have suggestive evidence of bronchiolitis (by clinical or x-ray criteria), and in addition, patients who fail to improve satisfactorily on otherwise good management, sooner or later deserve a trial of corticosteroid therapy. The increased risk of the peptic ulcer complications of hemorrhage and perforation should not be overlooked however. The usual adult dose of 30 to 60 mg prednisone daily or its equivalent is recommended for 1 to 3 weeks, followed by a gradual tapering to none or the lowest effective dose. Medication should be given in single daily doses to minimize corticotropin (ACTH) suppression. In chronic use, every other day therapy may be desirable.

Patients regularly seem to benefit from *breathing training*, especially when their lungs have become greatly overinflated. As the lungs "trap" more and more air, the ribs assume a more and more horizontal position, and the diaphragms become lower and flatter and thus descend less and less when they contract. Generally, either the ribs or the diaphragm take over the principal function of breathing, whereas the other breathing muscles simply assume a holding position, lag passively, or may even move paradoxically. Patients are taught (1) to coordinate rib and diaphragm movement, (2) to make expiration about three times longer than inspiration and smooth and even, and (3) to breathe out against pursed lips, which seems to hold flabby airways open longer and permit more efficient gas exchange.



Sedatives and tranquilizers should not be used in COPD because of their depressing effect on ventilation. The restlessness and irritability for which they are generally prescribed are common symptoms of both hypercapnia and hypoxemia. Meprobamate, 200 mg, or chlordiazepoxide, 10 mg, may be used sparingly (i.e., not more than 2 or 3 times daily) for restlessness and sleeplessness in selected cases.

When secondary erythremia (a hematocrit of 55% or more at sea level; 60% or more at 5000 feet) is present, a phlebotomy of 250 to 500 ml may give considerable temporary symptomatic relief. Erythremia usually indicates the presence of chronic hypoxemia, which indicates the need for oxygen therapy. Replacement iron therapy must be given to patients who receive frequent phlebotomies.

Congestive right heart failure is a frequent complication, especially in patients with chronic hypoxemia and pulmonary hypertension. Digitalization should be used; it is not only beneficial in right heart failure, but may also be useful in the management of other coexisting diseases of the myocardium such as coronary atherosclerosis. The fact that acidemia increases the risk of digitalis toxicity should not be overlooked. Oral and parenteral diuretics and salt restriction are also helpful for these patients. (The serum potassium level should be watched carefully, since hypoxemia and acidosis potentiate the kaliuretic effect of most diuretics.)

Continuous low-flow oxygen (1 to 3 liters/min by nasal cannulas) has proved remarkably beneficial in patients with chronic hypoxemia and its complications (pulmonary hypertension, secondary erythremia, and congestive right heart failure) and may make the use of phlebotomy and even digitalis and diuretics unnecessary. Oxygen should not be started without careful initial monitoring of its effect on both $Pa_{O_2}$ and $Pa_{CO_2}$, which is usually done in the hospital. The risk of "$CO_2$ narcosis," resulting from removal of the hypoxemic drive to breathe in patients with chronic hypercapnia, appears to have been exaggerated; this potentially dangerous complication is caused mostly by the administration of oxygen in concentrations that are too high (more than 3 liters/min) especially by face mask and especially in patients with acute exacerbations of airways obstruction. Muscle strengthening exercises can be taken by patients while they are receiving oxygen. As muscle strength is built up, they may


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function better when not receiving oxygen. Patients are urged to engage in physical exercise as much as their breathlessness will permit.

### Complications and prognosis

The most common complication of COPD is respiratory failure. Myocardial infarction and stroke are common incorrect diagnoses made in patients with respiratory failure. Peptic ulcer and its complications of hemorrhage and perforation occur more commonly in COPD than in the general population. Spontaneous pneumothorax is an uncommon, but very dangerous event.

Prognosis depends on the severity of the obstruction, the presence of cor pulmonale, the history of congestive heart failure, and the degree of abnormality of arterial blood gases.

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# 24

# The pneumoconioses

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## Definition and etiology

A pneumoconiosis is any change in the lung caused by inhaled dusts.

Various occupations in which pulmonary lesions have been reported include the following:

*Aluminum pneumoconiosis*
- Ammunition maker
- Fireworks maker

*Asbestosis*
- Asbestos weaver
- Brake manufacturer
- Clutch maufacturer
- Filter maker
- Floor tile maker
- Insulator
- Lagger
- Mill worker
- Miner or miller
- Roofer
- Shipbuilder
- Steam fitter

*Baritosis (barium)*
- Barite miller and miner
- Ceramics worker
- Fluorescent lamp maker
- Glassmaker
- Metallurgist
- Missile worker
- Neon sign maker
- Nuclear energy worker
- Phosphor maker
- Propellant manufacturer
- Toxicologist
- X-ray tube maker

*Beryllium disease*
- Alloy maker
- Bronze maker
- Ceramics worker
- Demolition person
- Electronic tube maker
- Extraction worker
- Fettler
- Flame cutter
- Foundryman
- Grinder
- Metalworker
- Metallizer
- Polisher
- Scarfer

*Coal workers' pneumoconiosis*
- Coal miner




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Motorman
Roof bolter
Tipple worker
Trimmer

*Kaolinosis (clay)*
Brick maker
Ceramics worker
China maker
Miner
Potter

*Siderosis (iron)*
Ship breaker
Welder

*Silicosis*
Abrasives worker
Bentonite mill worker
Brick maker
Ceramics worker
Coal miner
Diatomite worker
Enameler
Fettler
Filter maker
Foundryman
Miner or miller
Motorman
Paint maker
Polisher
Quarry man
Sandblaster
Shotblaster
Stonecutter
Stone driller
Well driller

*Talcosis (certain talcs)*
Ceramics worker
Cosmetics worker
Miner or miller
Papermaker
Plastics worker
Rubber worker

The simple fact that even prolonged exposure to a dust can be documented does not necessarily mean that a discernible pulmonary disorder or disability has occurred. Any harmful effect resulting from dust exposure is determined by a knowledge of the nature, duration, and intensity of the exposure, the particle size of the potentially hazardous dust, and whether or not the individual also smokes cigarettes. *Individual susceptibility to pulmonary injury*, for example, differences in pulmonary clearance efficiency, may play an important role.

Standards of safe working conditions in most nations today are based on maximum allowable concentrations, permissible concentrations, or target levels. Over the last 20 to 30 years these levels have been steadily lowered because of the following considerations: (1) experience with illnesses, (2) deaths of workers, and (3) feasibility. The goal of standards listed in Table 24-1 is the level to which workers may be exposed for 40 hours a week for a working *lifetime* without appreciable harm.



**Table 24-1.** *Examples of currently acceptable threshold limits of dust*

| Substance | m.r.p.c.f.* |
|---|---|
| ***Silica*** | |
| Crystalline (quartz) | 5 |
| Amorphous, including natural diatomaceous earth | 20 |
| ***Silicates*** (<1% crystalline silica) | |
| Asbestos | 5 |
| Talc | 20 |
| Portland cement (inert) | 50 |
| Coal dust | 2 |

*Millions of respirable particles per cubic foot of air.

In determining whether an individual has a symptom or disability-producing occupational disease, a complete and detailed work history is essential. General job terminology is much less desirable than the worker's personal job description. It is not enough to know, for example, that the patient was a foundry worker; the physician must be told whether he or she operated a shake-out section or an iron torch in the casting-cleaning department of the foundry, and so forth. Such a distinction is significant because very different exposures are involved. The person who operates a shake-out section is often exposed to high concentrations of finely divided dusts containing fibrogenic free silica. The iron torch operator, on the other hand, is usually exposed only to high concentrations of benign iron oxide fumes. A patient may say he or she is a sandblaster, but what does he or she use as an abrasive? Sand that contains a high concentation of free crystalline silica? Steel shot? Silicon carbide? Aluminum oxide? Details about welding are also important for several reasons. Some metals are known to cause disease. Cadmium, for instance, may produce a chemical pneumonitis. Dangerous exposure to ozone or to silica may result from welding; for example, gas shielded arc welding can produce excessive concentrations of ozone, a chemical highly damaging to the lungs; some welding fumes contain silica, but iron oxide fumes are harmless.


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In many instances it is desirable for the physician to obtain specialized advice on the potential disease-producing hazards of a given occupation from the state department of industrial or occupational hygiene. At times a specific on-site investigation of the hazards of a specific task is desirable. An additional problem is the frequency with which people tend to change jobs.

The particle size is important because particles of more than 8 μm seldom penetrate to the alveoli. When they become submicronic (i. e., less than 0.5 μm) in size, on the other hand, some may enter the lungs and pass right out again like a gas. In short, particles larger than 1.0 μm in diameter are most likely to be deposited in the nasopharynx; those less than 5 μm are most likely to be deposited in the nonciliated pulmonary surfaces, that is, alveolar walls.

The following are various occupational dust diseases:

1. Inert pneumoconioses
   - a. Iron: siderosis
   - b. Tin: stannosis
   - c. Barium: baritosis
   - d. Cement
   - e. Fiberglass
   - f. Talc
2. Active pneumoconioses caused by silica or silica content (i.e., fibrosis and other pathology producing)
   - a. Silica
   - b. Silicates, including kaolin
   - c. Diatomaceous earth
   - d. Asbestos
   - e. Bauxite
3. Active pneumoconioses without silica
   - a. Aluminum and aluminum oxide
   - b. Coal*
   - c. Graphite
   - d. Nickel carbonyl
   - e. Tungsten carbide
   - f. Beryllium
   - g. Cadmium
   - h. Cerium
   - i. Hair spray (?)

## SILICOSIS

Silicosis was first suspected by Hippocrates. Agricola described the fatal effects of silica dust in 1556, but the subject was given scant attention until the late nineteenth century. About 3000 new cases per year are diagnosed in the United States at present. It occurs particularly in persons engaged in underground mining, foundry work, and sandblasting. The duration of exposure needed to produce silicosis varies from as little as 18 months to as long as 30 years, depending on the intensity of the exposure. The disease process, once started, continues to worsen for at least 1 to 2 years after exposure ends.

*Coal, especially anthracite, sometimes contains an appreciable amount of silica.



The inhaled free crystalline silica or silicon dioxide particles are ingested by macrophages, which are destroyed; some pass into the lymphatics, which drain into the hilar nodes. More macrophages are produced; they ingest the liberated silica particles and are in turn destroyed. Collagen formation and hyalinization ensue, producing silicotic nodules measuring 2 to 5 mm in diameter. These nodules microscopically show fibrous whorls, are avascular and laminated, and contain silica particles that can be seen with lateral illumination. As the process progresses, the nodules coalesce and, when large enough, may undergo aseptic necrosis (i.e., cavitate). They tend to be distributed in the peribronchial and perivascular spaces that have perilymphatic drainage. Bronchiolar smooth muscle atrophy is frequently seen.

Simple silicosis is essentially an x-ray change consisting of nodulation with no single nodule greater than 1 cm in diameter. Subjects with simple silicosis in the absence of other pulmonary diseases such as chronic bronchitis and emphysema do not have symptoms or functional impairment that can reliably be related to their silicosis.

Complicated silicosis is the advanced stage of the disease and is characteristized by the presence of lumpy densities, usually associated with parenchymal fibrosis. In complicated silicosis, cough, expectoration, repeated chest infections, nonpleuritic chest pain, wheezing, dyspnea on exertion, ventilatory restriction, and airways obstruction may all be present. With the passage of time, the small nodules are replaced by large, conglomerate masses more than 1 cm in diameter, known as *progressive massive fibrosis* (PMF) (Fig. 24-1). In the late stages, the lungs commonly become severely restricted and sometimes obstructed, diffusion is impaired, the hypoxemia supervenes, all of which results in respiratory insufficiency, pulmonary hypertension, cor pulmonale, congestive right heart failure, and death. The complication of superimposed tuberculosis greatly accelerates this late disabling phase of silicosis.


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Fig. 24-1. Progressive massive fibrosis (PMF): advanced silicosis with superimposed tuberculosis. Note very large cavity with air fluid level, left lung. Also note central masses and peripheral emphysema. (Courtesy Medical Illustration Service, Veterans Administration Hospital, Denver, Colorado.)



Fig. 24-2. Eggshell nodes, bilateral: hilar nodes calcified only in their periphery, characteristic of both advanced silicosis and coal workers' pneumoconiosis. (Courtesy Medical Illustration Service, Veterans Administration Hospital, Denver, Colorado.)



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A characteristic radiographic feature of some cases of silicosis is the finding of the so-called *eggshell nodes* at the hila: they are enlarged, numerous, and characterized by calcification only in their periphery—a virtually pathognomonic sign of silicosis, but seen in only approximately 5% of cases (Fig. 24-2).

The *treatment* of advanced, disabling silicosis is not very effective; it consists of relieving the hypoxemia with oxygen-enriched air and treating the congestive heart failure with digitalis, diuretics, and salt restriction. The inhalation of aluminum powder to neutralize the effects of inhaled silica was once recommended, but is of no demonstrable benefit.

By far the most important consideration is *prevention*, which includes the use of wet drilling methods to keep down inhalable dust concentrations, adequate ventilation, effective and tolerable masks, and regular monitoring of dust counts in the mines. Routine annual chest radiographic examinations and change of occupation when the presence of simple silicosis is well established are helpful in preventing the progression to PMF.

Silica and silicon dioxide react synergistically with mycobacteria; not only is their growth accelerated on culture media, but also host immunity to them is reduced. Fully developed silicotuberculosis is difficult to treat even with the most effective antituberculosis chemotherapy; furthermore, it is devastating to pulmonary function. Patients with silicosis who react to tuberculin (i.e., 10 mm induration or more to purified protein derivative intermediate) should be given isoniazid (INH) preventive therapy for 2 years after careful exclusion of the presence of active tuberculosis, especially by sputum microbiology.

## ASBESTOSIS

Asbestos, a commercial term, is a mixture of hydrous silicates of magnesium, sodium, and iron in various proportions and in fibrous form. There are two major types: the *amphiboles* (crocidolite, amosite, anthophyllite) and *chrysotile*. Crocidolite fibers, long, thin, and straight in comparison with the others, which are larger and serpentine, are more prone to cause harm possibly because macrophages are less able to engulf them. Asbestos is used industrially in many ways, including the manufacture of clutch facings, brake





**Fig. 24-3.** Asbestosis: bilateral parenchymal fibrosis, especially in lower lungs, with diaphragmatic calcifications, bilateral. (Courtesy Medical Illustration Service, Veterans Administration Hospital, Denver, Colorado.)

linings, auto undercoating, roofing, insulation, and fire resistant textiles. The heat generated by applying brakes converts a high percentage of the asbestos brake lining into forsterite, an amorphous and essentially benign form of asbestos. The risk of mining asbestos is reduced because the mining is mostly open pit and the crude ore comes out in large clumps. Certain procedures such as drilling and


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milling are quite hazardous unless wet drilling and proper masks are used. The asbestos fiber causes a physical type of tissue irritation, morphologically seen as a diffuse, nonnodular, pulmonary fibrosis involving the terminal airways, alveoli, and pleurae (Fig. 24-3). Asbestos bodies may be found in the sputum, rarely in pleural fluid. Symptoms, especially dyspnea and dry cough, tend to occur before



Fig. 24-4. Mesothelioma: left pleura, with hydropneumothorax and obliteration right costophrenic angle. (Courtesy Medical Illustration Service, Veterans Administration Hospital, Denver, Colorado.)



radiographic changes are clearly manifested. Changes are noted particularly in the parietal pleurae and in the lower lungs. Pleural effusions, including bilateral effusions, are common, but often overlooked. The process tends to progress after exposure has ended. Physiologic changes include restriction and impaired diffusion caused by widespread fibrosis. The major complications of asbestosis in addition to pleural effusions and fibrosis are bronchogenic carcinoma, pleural mesothelioma (Fig. 24-4), and calcified basal parietal pleurae. The risk of bronchogenic carcinoma is largely confined to cigarette smokers in whom the risk is many times greater than in those smokers without asbestosis.

## TALCOSIS

Talc (or soapstone) is a hydrated magnesium silicate widely mined and milled for use as a dusting powder, and as a constituent of paints, ceramics, asphalt, roofing materials, insecticides, and cosmetics. Whether talc per se (free of fibers) can cause disease is not known. It is generally agreed that the disease of talc workers is attributable to *tremolite fibers* and possibly to chrysotile and anthophyllite (i.e., asbestiform) as well. Exposure may produce rather massive fluffy densities, especially in the midlungs, without significant symptoms or physiologic disturbance. There is a distinct risk, however, of disabling or fatal progressive fibrosis. Inhaled in high concentrations, talc may produce an acute bronchoconstriction that may be fatal in children. Pathologically, the process is initially a granulomatous reaction containing doubly refractile particles engulfed in macrophages; this is followed by interstitial fibrosis and endarteritis. Corticosteroids may cause symtomatic relief in severe cases.

## COAL WORKERS' PNEUMOCONIOSIS

Coal workers' pneumoconiosis (CWP) is defined as the accumulation of coal dust in the lungs and the reaction of the tissues to its presence. It should not be confused with "black lung," which has been defined as any chronic respiratory disease in a coal miner. The reason for making this distinction is that black lung compensation benefits are now given to surface coal miners as well as underground coal miners with respiratory disability after 25 or 30 years of exposure


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without the necessity of confirmation of CWP by chest radiograph. This is a result at least in part of disagreements over the interpretation of chest radiographs of miners. Legislation under consideration in 1977 would grant compensation to coal miners simply because of their exposure without proof of respiratory disability *or* radiographic evidence of CWP.

While some *silica* is present in some coal dusts, especially the anthracite and higher ranks (grades) of coal, and the radiographic appearances of silicosis and CWP are indistinguishable, the *consensus of informed opinion* is that contaminating silica does not play a major role in the causation of simple CWP. This opinion is based on two facts: (1) *pure carbon exposure in humans can produce* a disease radiographically and pathologically similar to CWP, and (2) *the pathology of CWP is quite different from that of* silicosis.

The *initial* pulmonary reaction to coal dust *is the coal macule*, which consists of dust enmeshed in connective tissue or around respiratory bronchioles with accompanying *focal emphysema; these* changes are scattered widely but with a predilection for the upper lungs. This process is distinguishable from the centrilobular emphysema of cigarette smokers by the absence of an associated bronchiolitis. Reticulin accumulation, collagen formation, and fibrosis ensue. In time, the fibrous areas are replaced by PMF as in silicosis. When more than 13% (some say 18%) of the lung ash at autopsy proves to be silica, the case is diagnosed as silicosis. In addition, silica is believed to play a key role when simple CWP progresses to PMF.

CWP was first described in Great Britain in the 1830s although the mining of coal goes back another 1400 years or so. CWP is now found in from 6% to 10% of current U.S. miners, more in ex-miners. The frequency of CWP is falling off because of markedly improved dust control in the mines in recent years. In the past, it took 15 to 20 years of exposure to acquire the disease.

The diagnosis is based on a detailed and reliable history of exposure (including dust counts made in the involved mines and the nature of the miner's work (i.e., intensity of exposure, in the mines) *and* interpretation of a chest film of good quality showing at least some fine nodulation.



An elaborate system of reading the chest film for all pneumoconioses was developed by the UICC/UC* in 1970. Each lung is divided into three zones. Each film is read twice. The fine nodulation is classified as predominantly rounded or irregular and rated as 1, 2, or 3 according to the profusion or intensity of the shadows. The process progresses from simple to complicated when any opacity measures at least 1 cm in diameter (Category A), when one or more opacities combined measure more than 5 cm but less than one third of one lung (Category B), and when one or more large opacities added together have a diameter exceeding one third of one lung (Category C).

The onset of symptoms and physiologic deficits (especially *impairment* of gas tranfer, restriction, and airways obstruction) reliably attributable to the presence of coal dust is delayed until the appearance of complicated CWP, usually of Categories B and C. Coal workers with or without radiographic evidence of simple CWP *often have the symptoms, signs,* and physiologic findings of COPD, but careful studies of miners and controls indicate that the incidence *of COPD is related to cigarette smoking* and not to coal mining.

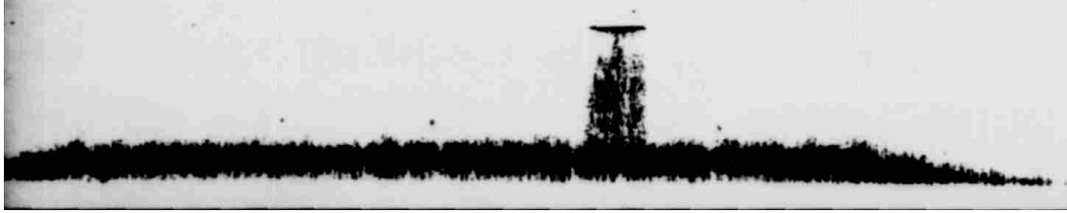
Death caused by CWP is invariably related to the development of restriction (i.e., fibrosis) usually with some degree of airways obstruction, hypoxemia, pulmonary hypertension, and cor pulmonale.

*Treatment* of CWP is essentially nonexistent. *Prevention* depends on dust monitoring and control, adequate ventilation of the mines, wet drilling, effective and tolerable masks, annual chest radiographic examination, and termination of employment of those with Category 2 or more CWP.

## OTHER PNEUMOCONIOSES INVOLVING SILICA

*Shaver's disease* is caused by the inhalation of fumes containing aluminum oxide and silica produced by fusing bauxite powder in iron pots at over 2000°C. It occurred almost exclusively in a small region near Niagara Falls in Canada. The course of the disease is rapid with severe dyspnea, spontaneous pneumothoraces, severe respiratory insufficiency, and death.

*International Union Against Cancer/University of Cincinnati.




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*Diatomite* or diatomaceous earth contains amorphous silicon dioxide and is mined in several parts of the world, especially in coastal California. It is used to manufacture abrasive polish, insulation, and cleaning materials. In the calcined or flux calcined form it can produce a severely disabling pneumoconiosis because of the contamination with free crystalline silica.

*Caplan's syndrome* was first described in Welsh coal miners with rheumatoid arthritis. It consists of a large "cannonball" type of infiltrate in both upper lungs, generally with no or mild pulmonary symptoms. The nodules are similar to rheumatoid nodules. These lesions have more recently been found in hard rock miners and even in a few non-miners with the rheumatoid diathesis in many other parts of the world (see Chapter 23).

## BERYLLIUM

Beryllium has become an important and useful metal because of its lightness and tensile strength. It has been used in the phosphors used in fluorescent lighting tubes, in the cement used to manufacture false teeth bridge work, in the solid fuels for space flight, and in ceramics and various alloys. Because of the hazards involved, it is currently used mostly in nuclear physics, since it reduces the speed of fission reactions, and as a window in x-ray tubes. The raw ore is not hazardous until it has been processed into the pure metal or one of its salts. Inhalation of purified beryllium may cause an acute rhinitis, tracheobronchitis, or pneumonitis, usually after a brief but heavy exposure. The interval between exposure and onset may vary up to several weeks. While some 80% recover completely, the resulting pneumonia or edema-like processes produce severe hypoxemia requiring supplementary oxygen and, at times, mechanical ventilation. Mortality is around 10% and some 10% progress to the chronic form.

Beryllium granulomas may occur in the skin.

Chronic *beryllium granulomatosis* is a systemic disease that produces granulomata throughout the body, especially in the lungs. It has occurred as long as 15 years after initial exposure. The exposure may be slight and even indirect, as for example, the wife of a beryllium worker exposed to beryllium oxide dust on his workclothes. There is a considerable variation in individual susceptibility, with only a small fraction of the population being highly susceptible.



It apparently occurs only after exposure to processed beryllium such as beryllium oxide and not on exposure to beryllium ores. The disease has an insidious onset characterized by persistent, severe, dry cough, severe weight loss, and severe dyspnea on exertion; other less common symptoms include a low-grade fever and anorexia. The chest radiographic findings resemble the miliary form of sarcoidosis, including hilar adenopathy. Severe, almost pure diffusion impairment with no ventilatory impairment or restriction is the striking physiologic picture. The morphology is almost indistinguishable from miliary sarcoidosis, except that the lesions and, in fact, many involved and uninvolved organs contain a significant amount of beryllium. Response to corticosteroids is often dramatically effective but only temporarily beneficial.

## OTHER MISCELLANEOUS PNEUMOCONIOSES

*Arc welder's siderosis* probably should not be classified as a disease, since symptoms, abnormal signs, and disability do not occur and autopsy shows no tissue changes. The chest radiograph shows a fine miliary stippling caused by the deposition of iron pigment. When the welding process includes minerals other than iron (e.g., silicon dioxide, silicates, aluminum), it becomes hazardous. Acute welder's lung is apparently a pneumonitis occurring in response to high concentrations of ozone, usually caused by the presence of aluminum.

*Tungsten carbide*, or hard metal, workers are exposed to a very fine powder and may develop interstitial fibrosis or airways obstruction possibly because of cobalt content.

*Nickel carbonyl*, emitted during nickel processing and utilized as a catalyst in petroleum, plastic, rubber, and nickel plating industries, may cause acute pneumonitis and/or bronchiolitis; it is also associated with an increased incidence of lung cancer.

*Thesaurosis* (storage disease) is a syndrome of mild cough, dyspnea, and miliary infiltrate occurring after exposure to hair sprays (polyvinylpyrrolidine). Results of biopsy sometimes show deposits of inert macromolecules. Since it has not been reproducible in animals, some biopsy results fail to reveal the inhaled material, and many who are exposed do not develop the condition, its existence as an entity remains questionable.

*Uranium* miners are unavoidably exposed to silica. Especially


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when they also smoke cigarettes, they have a very high risk of lung cancer, significantly higher than cigarette smoking alone would justify.

• • •

All physicians should watch for occupational or environmental causes of chest symptoms and radiographic changes. It is also possible that such exposures may precipitate pulmonary collagenoses such as lupus erythematosus and scleroderma.

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