Medical Education Systems, Inc.

Course 109

RESPIRATORY DISEASES

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RESPIRATORY DISEASES

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RESPIRATORY DISEASES

Part I. AUTHOR'S COMMENTS AND UPDATE

Chronic respiratory disease has emerged as a major nationwide health problem. Chronic obstructive pulmonary diseases have increased to epidemic proportions and are currently significant causes of morbidity and mortality in this country. These conditions have revolutionized the practice of pulmonary medicine and have promoted the emergence of the allied health specialties, such as respiratory therapy and pulmonary nursing.

There has also developed increasing recognition of the dangers of occupational lung diseases and much has been done to prevent them and promote early diagnosis. Despite these new developments, tuberculosis remains a significant worldwide health problem. Many advances, however, have made treatment much easier (more available drugs and shorter treatment programs).

Lung disease is not restricted to any age group. Each year tens of thousands of children under the age of five die from various pulmonary causes and even more adult deaths occur annually due to chronic lung disease.

As is true in many other organ diseases, prevention and early diagnosis are the keys to ultimate control and cure. Respiratory therapists can play a very important role in assisting physicians in gathering clues in the clinical history, examination and physiologic measurements that may lead to early detection and treatment. To accomplish this end, respiratory therapists must learn about lung disease. Learning involves not only reading and studying didactic information, but devoting time to clinical rounds with a teacher well versed in the spectrum of lung disease.

Respiratory Diseases

Sources: Co-Lead Agencies: Centers for Disease Control and Prevention
National Institutes of Health

[Note: This update consists of the **Healthy People 2010 Information Access Project** which provides dynamic, pre-formulated PubMed searches for selected objectives in this focus area so that current information and evidence-based strategies related to these objectives are easier to find. The National Library of Medicine has also provided PubMed links to available references that appear at the end of this focus area document. This update is intended to augment the information provided in this course which follows. To make the best use of the course, review the material presented in this brief update, and as you finish each section, move to the corresponding section in the course.]

Goal

Promote respiratory health through better prevention, detection, treatment, and education efforts.

Overview

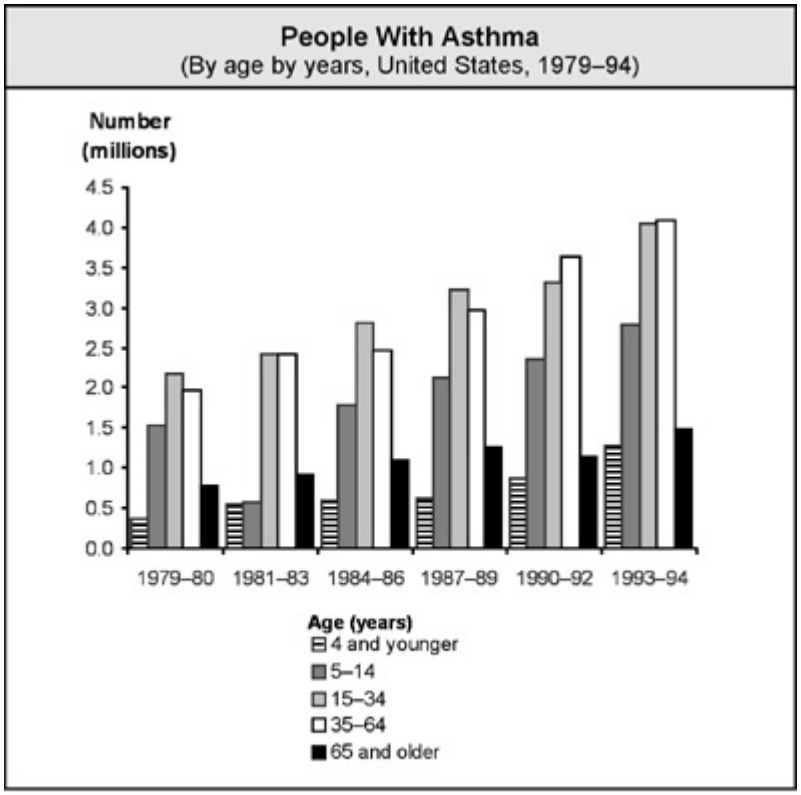
Asthma, chronic obstructive pulmonary disease (COPD), and obstructive sleep apnea (OSA) are a significant public health burden to the United States.[1] Specific methods of detection, intervention, and treatment exist that may reduce this burden. Several behaviors and diseases that affect the respiratory system, such as tuberculosis, lung cancer, acquired immunodeficiency syndrome (AIDS), pneumonia, occupational lung disease, and smoking, are covered in other chapters. (See Focus Area 3. Cancer, Focus Area 13. HIV, Focus Area 14. Immunization and Infectious Diseases, Focus Area 20. Occupational Safety and Health, Focus Area 25. Sexually Transmitted Diseases, and Focus Area 27. Tobacco Use.) Certain other important respiratory diseases, such as respiratory distress syndromes, sarcoidosis, and chronic sinusitis, which are difficult to define, detect, prevent, or treat, are not discussed in this chapter. Their omission, however, is not a reflection on the magnitude of the health problems associated with them.

Asthma and COPD are among the 10 leading chronic conditions causing restricted activity. After chronic sinusitis, is the most common cause of chronic illness in children.[2] Methods are available to treat these respiratory diseases and promote respiratory health.

Issues and Trends

Asthma is a serious and growing health problem. An estimated 14.9 million persons in the United States have asthma .[3] The number of people with asthma increased by 102 percent between 1979–80 and 1993–94.[4]

Asthma is responsible for about 500,000 hospitalizations,[3] 5,000 deaths,[3] and 134 million days[4] of restricted activity a year. Yet most of the problems caused by asthma could be averted if persons with asthma, along with their health care providers, managed the disease according to established **guidelines.** Effective management of comprises four major components: controlling exposure to factors that trigger episodes, adequately managing with medicine, monitoring the disease by using objective measures of lung function, and educating patients to become partners in their own care.[2, 5] Such prevention efforts are essential to interrupt the progression from disease to functional limitation and disability and to improve the quality of life for persons with asthma .



Source: CDC, NCHS. Surveillance for Asthma—United States, 1960–1995. *Morbidity and Mortality Weekly Report* 47(SS-1);1-28, 1998.

In 1996, was the 10th most common principal diagnosis in emergency department (ED) visits.[3] Among diseases commonly seen in outpatient departments, asthma was the ninth most frequent diagnosis in 1996.[6] In 1995, some 9 million physician office visits were made for asthma .[6] From 1990 to 1992, persons with spent an estimated 64 million days in bed because of asthma , ranking as the fourth highest chronic health condition.[4] The proportion of people with asthma who are limited in activity increased slightly from 19.4 percent in 1986–88 to 19.6 percent in 1994–96.[7]

Direct medical expenditures for asthma amounted to $3.64 billion in 1990, and indirect economic losses accounted for an additional $2.6 billion.[8] Of direct medical care costs, approximately 57 percent was spent on hospitalizations ($1.6 billion), outpatient hospital visits ($190 million), and ED visits ($295 million). Physician-related services accounted for 14 percent of the total expenditures, including $347 million for outpatient services. Prescription medications represented 30 percent of direct medical costs. Such facts highlight the significant cost of hospital care for asthma , compared to the more frequently used and less costly outpatient and pharmaceutical services.

Indirect costs—nonmedical economic losses such as days missed from work or school, caregiver costs, travel and waiting time, early retirement due to disability, and premature death—account for slightly less than 50 percent of the total costs of asthma . Data suggest that the uneven distribution of costs of asthma relates to nonscheduled acute or emergency care, indicating poor management and suboptimal outcomes.[9], [10]

Environmental and occupational factors contribute to illness and disability from asthma . Decreases in lung function and a worsening of asthma have been associated with exposure to allergens, indoor pollutants (for example, tobacco smoke), and ambient air pollutants (for example, ozone, sulfur dioxide, nitrogen dioxide, acid aerosols, and particulate matter).[11], [12] Approximately 25 percent of children in the United States live in areas that exceed the Federal **Government's standard for** ozone.[13] Occupational factors cause or trigger asthma episodes in 5 to 30 percent of adults with the disease.[14] Environmental factors are associated with upper respiratory infections that contribute to illness and disability in children and adults.[15]

Disparities

Within the U.S. population, the health, economic, and social burdens of asthma vary. Disproportionate rates of death, hospitalization, ED use, and disability from asthma occur in certain age, gender, racial, and ethnic groups.

While the number of adults with asthma is greater than the number of children with asthma , the rate is rising more rapidly in preschool-aged children than in any other group.[1] In 1995, the rate of self-reported among children and adolescents under age 18 years was 7.5 percent, compared to 5.7 percent among the general population. The rates were higher in boys under age 18 years than in girls in the same age group. The rates of self-reported asthma attacks were higher for women (6.7 percent) than men (5.2 percent) and higher for African Americans (6.7 percent) than whites (5.6 percent).[1] Among adults, women of all races have higher rates of illness and death from asthma than men.[16]

Death from asthma is two to six times more likely to occur among African Americans and Hispanics than among whites.[1] Although the number of deaths annually from asthma is low compared to other chronic diseases, the death rate for children aged 5 to 14 years and young adults aged 15 to 34 years doubled from 1979–80 to 1993–95 (from 1.5 to 3.7 deaths per million children aged 5 to 14 years and 2.8 to 6.3 deaths per million persons aged 15 to 34 years).[1] In 1993–95,death rates are slightly higher overall in women than in men.[1]

Rates of hospitalization for asthma demonstrate similar variations. Rates for African Americans are almost triple those for whites. Rates are higher among women than among men.[1] Asthma hospitalization rates have increased dramatically among children under age 5 years. From 1980 to 1993, the rate increased from 36 to 65 children hospitalized per 10,000 children under age 1 year. Some of this increase may be related to changes in diagnostic practices and changes in coding and reimbursement, but a large portion represents a true increase in illness and disability.

In the inner city, patients frequently use EDs for asthma care. In 1993 and 1994, African Americans were four times more likely than whites to visit an ED because of asthma. Asthma patients in general and high-risk inner-city patients—in particular, those with a history of severe who were hospitalized or visited the ED for within the previous 2 years—need to be able to recognize the signs and symptoms of uncontrolled asthma and know how to respond appropriately.

The economic burden of asthma disproportionately affects patients with severe disease. Socioeconomic status, particularly poverty, appears to be an important contributing factor to asthma illness, disability, and death. In the United States, the rate of asthma cases for nonwhites is only slightly higher than for whites, yet the death, hospitalization, and ED-visit rates for nonwhites are more than twice those for whites.[1] Although reasons for these differences are unclear, they likely result from multiple factors: high levels of exposure to environmental tobacco smoke, pollutants, and environmental allergens (for example, house dust mites, cockroach particles, cat and dog dander, and possibly rodent dander and mold); a lack of access to quality medical care; and a lack of financial resources and social support to manage the disease effectively on a long-term basis.[17] Research into the role of socioeconomic factors is needed to identify additional prevention opportunities.

Opportunities

Scientific research has led to greater asthma control than was available in the early 1980s.[5] Effective management of includes four components: avoiding or controlling the factors that may make worse (for example, environmental and occupational allergens and irritants), taking appropriate medications tailored to the severity of the disease, objective monitoring of the disease by the patient and the health care professional, and actively involving the patient in managing the disease.[5] Effective asthma management reduces the need for hospitalizations and urgent care visits (in either an ED or physician's office) and enables patients to enjoy normal activities.[18], [19]

Advances in human genetics related to asthma are expected to provide better information about the contribution of genetic variation to the development of disease when people are exposed to certain environmental factors and variation in individual response to therapy. The use of this genetic information will improve targeted disease prevention and health management strategies for respiratory diseases.

Patient education is one of four components of effective asthma management.[5] Patients who are taught self-management skills are able to manage and control their disease better than patients who do not receive education.[5] Patients need to learn to work with health care providers to optimize asthma care. Thus, both patients and health care providers need to be trained and educated on effective asthma management. Health outcomes for —illness, disability, quality of life, and death—are related directly to the actions of health care professionals and patients. The National Asthma Education and Prevention Program (NAEPP) provides guidelines for diagnosis and management that should be incorporated into the curricula of health professional schools.[5, 20] Currently, there are no national data systems for tracking the training of health care providers in asthma management. Therefore, the issue is not covered in this focus area's objectives. It represents an important research and data collection agenda for the coming decade. In addition, research to identify the primary causes of development of asthma is a high priority. Such research can provide a scientific basis for efforts to prevent the development of asthma.

To control asthma effectively, asthma patients, particularly those on daily medication, need an action plan developed under their physician's guidance. The plan spells out when and how to take medicines

correctly, as well as what to do when asthma worsens. The treatment of persistent asthma emphasizes daily long-term therapy aimed at the underlying inflammation and preventing symptoms, rather than relying solely on treating symptoms with short-acting inhaled medication, such as a beta agonist medication. Use of more than one canister of the short-acting inhaled beta agonist medication per month is an indication of uncontrolled asthma and the need to start or increase long-term preventive therapy. Patients also need to work with health care providers during follow-up visits, particularly after being hospitalized, to make sure they understand and are able to follow the long-term management plan.

Working with local community groups to mobilize community resources for a comprehensive, culturally and linguistically competent approach to controlling asthma among high-risk populations is a priority. From a community-based perspective, States need to track occupational and environmental factors that cause or trigger asthma episodes. Such surveillance efforts should include collecting State-based data on the proportion of the population with asthma and monitoring occupational and environmental exposures and their impact on illness and disability related to . Efforts directed to improving the environmental management of asthma also include reducing exposure to allergens and irritants, such as environmental tobacco smoke, and outdoor air pollution from ozone, sulfur dioxide, and particulate diesel matter.

Professional organizations, lay volunteer groups, Federal agencies, and the private sector have worked together and with NAEPP to implement a spectrum of asthma programs at national and local community levels. For example, numerous publications, media campaigns, and conferences target various audiences. Intensified efforts are planned to reach primary care providers, patients, and school personnel.[16, 20] A high-level work group convened by the U.S. Department of Health and Human Services in 1997 assessed the most urgent needs for tackling the growing problem of . The work group's departmentwide strategic plan, Action Against Asthma, identified opportunities and presented a coordinated approach for improving prevention and management.[16]

Chronic Obstructive Pulmonary Disease

Issues and Trends

COPD includes chronic bronchitis and emphysema—both of which are characterized by irreversible airflow obstruction and often exist together. Similar to , COPD may be accompanied by an airway hyperresponsiveness. Most patients with COPD have a history of cigarette smoking. COPD worsens over time with continued exposure to a causative agent—usually tobacco smoke or sometimes a substance in the workplace or environment.

COPD occurs most often in older people. As much as 10 percent of the population aged 65 years and older is estimated to have COPD.[2] COPD has a major impact on health care, illness, disability, and death in the older population, and the magnitude of the problem is growing. Since 1980, the prevalence and age-adjusted death rate for COPD increased more than 30 percent.[2, [21], [22]] Most of the increase occurred in people over age 65 years. Taking into account the expected aging of the U.S. population over the next 10 to 30 years as well as the improved management of other smoking-related diseases, any decline in the proportion of persons with COPD is unlikely without substantial changes in risk factors, mainly reductions in cigarette smoking. This is important for both men and women, given the modest decline in cigarette smoking rates from 1990 to 1995.[22]

Between 80 and 90 percent of COPD is attributable to cigarette smoking. However, not all smokers develop COPD, and not all patients with COPD are smokers or have smoked in the past.[23], [24] Individual susceptibility to the adverse health effects of cigarette smoke on the lung appears to vary within the general population. Some 10 to 15 percent of smokers show a rate of decline in lung function that will result in COPD with severe disability. Smoking cessation is the only treatment that slows the decline. Susceptible smokers who stop smoking do not regain lost lung function,[25] but the rate of loss will return to what is normal for a nonsmoker.

How cigarette smoking causes COPD is an active area of research. The development of COPD—in particular, emphysema—is thought to be due to a chemical imbalance in the lungs caused by cigarette smoke.[26] In some individuals, emphysema occurs because of a genetic deficiency. Emphysema due to genetic deficiency, called familial emphysema, occurs even in nonsmokers, **but smoking hastens its** occurrence.

Familial emphysema probably accounts for less than 5 percent of all cases of COPD.[27]

Smoking and occupational exposures together cause respiratory diseases and lung cancer.[28], [29] Miners, firefighters, metal workers, grain handlers, cotton workers, paper mill workers, agricultural workers, construction workers who handle cement, and others employed in occupations associated with prolonged exposure to dusts, fumes, or gases develop significant airflow obstruction, coughing, phlegm, dyspnea, wheezing, and reduced lung function.[27, 29, 30]

Population studies have shown that chronic exposure to air pollution has an independent adverse effect on lung function.[31], [32] A multiyear study of the respiratory effects of long-term exposure to environmental tobacco smoke and air pollution reported that both long-term ozone and childhood exposure to maternal tobacco smoke were associated with diminished lung function in college students.[33] Viral infections also may contribute to susceptibility to COPD, and they are considered to play a role in the onset of airflow obstruction.

The direct costs of health care services and indirect costs through loss of productivity related to COPD amounted to $26 billion in 1998.[26] About 14 million persons in the United States have COPD—about 12.5 million have chronic bronchitis and 1.9 million have emphysema.[27] Emphysema has not increased, but since 1980, cases of chronic bronchitis increased 75 percent.[27]

Because national data systems will not be available in the first half of the decade for tracking progress, two subjects of interest concerning respiratory diseases are not addressed in this focus area's objectives. The first topic addresses increasing the proportion of primary care providers who are trained to provide culturally competent health services to racial and ethnic groups seeking care for chronic obstructive pulmonary disease. The second involves increasing the proportion of primary care providers who are trained to use appropriate lung function tests to **recognize the early signs of chronic obstructive pulmonary disease before the disease becomes serious and disabling.**

Disparities

Reliable statistics are not as available for COPD total cases, illness, disability, or death in African Americans, Hispanics, and other ethnic groups as for whites.[34], [35] From 1982 to 1984, the proportion of adults with COPD was 6.2 percent among whites and 3.2 percent among African Americans. In 1982, the age-adjusted COPD death rate for whites was 16.6 deaths per 1,000 population and 12.8 deaths per 1,000 for African Americans.

Among the Hispanic groups studied, Puerto Ricans demonstrated a higher proportion of chronic bronchitis (2.9 percent) than Mexican Americans (1.7 percent) or Cuban Americans (1.7 percent).[34, 35]

In 1995, the proportion of the population with COPD was 5 percent in men aged 45 to 64 years and 11 percent in men aged 65 years and older. The proportion was 10 percent in women aged 45 to 64 years and 9 percent in women aged 65 to 74 years.

Death from COPD is more common in men than in women, and the death rate increases steeply with age.[27, 36] Men and women have similar COPD death rates before age 55 years, but the rate for men rises thereafter. At age 70 years, the rate for men is more than double that for women, and at age 85 years and older, the COPD death rate for men is 3.5 times that for women.[37] The proportion was 8 percent for whites aged 45 to 64 years and 10 percent for whites aged 65 years and older. The proportion of African Americans with COPD was 6 percent for those aged 45 to 64 years and 8 percent for those aged 65 years and older.[2] COPD death rates were lower in the Hispanic groups than in non-Hispanic whites; however, these rates have been increasing for Hispanics.[33]

Women might be more susceptible than men to developing COPD when exposed to risk factors such as tobacco smoke.[38] The beneficial effects of stopping smoking on the rate of lung function decline may be greater for women than men.[39]

Opportunities

Primary care physicians are in a key position to provide optimal care to patients with COPD and to provide counseling during clinical or health center visits to patients who smoke. Effective tests are available to screen patients for COPD, and primary care physicians need to be trained in the latest methods to detect and treat the disease.

Obstructive Sleep Apnea

Issues and Trends

Some 18 million persons in the United States were estimated to have OSA in 1993.[40] OSA affects all races, ages, and socioeconomic and ethnic groups.[41] Because OSA causes serious disturbances in normal sleep patterns, patients experience excessive daytime sleepiness and impaired performance. Common consequences of OSA range from personality changes and sexual dysfunction to falling asleep at work or while driving.[40, 41]

OSA symptoms include many repeated involuntary breathing pauses during sleep. The breathing pauses often are accompanied by choking sensations that may wake the patient. Other symptoms include intermittent snoring, awakening from sleep (poor sleep), early morning headaches, and excessive daytime sleepiness.

OSA can increase the seriousness of other lung diseases that decrease airflow, such as and COPD. Cardiovascular deaths alone due to OSA have been estimated at 38,000 a year.[40] Individuals with OSA often do not recognize reductions in alertness, diminished productivity, and discord in interpersonal relationships as part of the syndrome. Persons affected by OSA, for example, are seven times more likely to be involved in multiple vehicular crashes.[42] In children, OSA can disrupt sleep. OSA also may cause daytime behavioral problems that affect workplace performance and affect their learning ability in school. Infants with siblings or parents who have OSA inherit an increased risk of sudden

infant death syndrome (SIDS).[43] This tragic sleep-related breathing disorder takes the lives of more infants than all other causes combined.

Disparities

OSA is prevalent particularly in men over age 50 years and in postmenopausal women, when hormonal changes appear to increase risk. The risk of OSA also is increased in certain racial and ethnic groups. Among young African Americans, the likelihood of experiencing OSA symptoms is twice that of young whites.[44] Nearly 50 percent of OSA patients have high blood pressure.[45], [46], [47]

Opportunities

A major factor in the pervasiveness of OSA's effects on health and society has been the failure to educate people—and especially health care practitioners—about the disorder. A wide range of behavioral, mechanical, and surgical treatments can be used to manage OSA symptoms. Providing persons at risk with culturally and linguistically appropriate information about OSA could enable them to prevent or lessen the effects of OSA. Improved awareness of OSA symptoms represents a major public health challenge.

Primary care providers are an important barometer of OSA awareness because they are a first stop for patients who are seeking appropriate diagnosis and treatment. However, only 79 cases of sleep disorder were diagnosed in a sample of 10 million patient records from 1989 and 1990.[40] In 1990 about a third of the medical schools in the United States offered no training in sleep medicine, and another third provided less than 2 hours on average for all sleep topics.[48] Data systems to track the training of health care providers in OSA over the decade are not currently available, and therefore the issue is not addressed in this focus area's objectives. However, it represents an important research and data collection agenda. In the absence of strong educational models for physicians, the risk remains high that OSA will be misdiagnosed and mismanaged.

The National Commission on Sleep Disorders Research[40] was established by the U.S. Congress in 1988 to assess the societal and economic impact of sleep disorders and the resources available to promote the prevention, diagnosis, and treatment of such disorders. In a 1994 report to Congress, the Commission concluded that even though the science of sleep disorders is not fully developed, such disorders can be prevented. The commission recommends that research on the natural history of sleep disorders be made an urgent national concern. Epidemiologic studies must be conducted to evaluate risk factors that lead to sleep disorders and to determine which sleep disorders lead to other serious health problems.

Interim Progress Toward Year 2000 Objectives

For the three objectives specific to in Healthy People 2000, available data indicate movement away from the targets as the rate of hospitalizations and activity limitation increase and movement toward the target for increasing the proportion of persons with who receive patient education. There were no objectives in Healthy People 2000 for COPD and OSA.

Note: Unless otherwise noted, data are from the Centers for Disease Control and Prevention, National Center for Health Statistics, *Healthy People 2000 Review, 1998–99.*

Healthy People 2010—Summary of Objectives

Respiratory Diseases

Goal: Promote respiratory health through better prevention, detection, treatment, and education efforts.

Number	Objective Short Title
24-1	Deaths from asthma
24-2	Hospitalizations for asthma
24-3	Hospital emergency department visits for asthma
24-4	Activity limitations
24-5	School or work days lost
24-6	Patient education
24-7	Appropriate care
24-8	Surveillance systems
Chronic Obstructive Pulmonary Disease (COPD)	
24-9	Activity limitations due to chronic lung and breathing problems
24-10	Deaths from COPD
Obstructive Sleep Apnea (OSA)	
24-11	Medical evaluation and follow-up
24-12	Vehicular crashes related to excessive sleepiness

24-1. Reduce deaths.

Target and baseline:

Objective	Age Group	1998 Baseline	2010 Target
		Rate per Million	
24-1a.	Children under age 5 years	2.1	1.0
24-1b.	Children aged 5 to 14 years	3.3	1.0
24-1c.	Adolescents and adults aged 15 to 34 years	5.0	2.0
24-1d.	Adults aged 35 to 64 years	17.8	9.0
24-1e.	Adults aged 65 years and older	86.3	60.0

Target setting method: Better than the best.

Data source: National Vital Statistics System (NVSS), CDC, NCHS.

Select Age Groups, 1998	Asthma Deaths				
	24-1a. Children Under Age 5 Years	**24-1b. Children Aged 5 to 14 Years**	**24-1c. Adoles-cents and Adults Aged 15 to 34 Years**	**24-1d. Adults Aged 35 to 64 Years**	**24-1e. Adults Aged 65 Years and Older**
	Rate per Million				
TOTAL	2.1	3.3	5.0	17.8	86.3
Race and ethnicity					
American Indian or Alaska Native	DSU	DSU	DSU	DSU	DSU
Asian or Pacific Islander	DSU	DSU	DSU	12.8	136.9
Asian	DNC	DNC	DNC	DNC	DNC
Native Hawaiian and other Pacific Islander	DNC	DNC	DNC	DNC	DNC
Black or African American	8.1	9.7	16.6	52.3	130.4
White	DSU	2.0	3.0	13.3	81.1
Hispanic or Latino	DSU	DSU	3.7	16.4	84.5

Select Age Groups, 1998	Asthma Deaths				
	24-1a. Children Under Age 5 Years	**24-1b. Children Aged 5 to 14 Years**	**24-1c. Adoles-cents and Adults Aged 15 to 34 Years**	**24-1d. Adults Aged 35 to 64 Years**	**24-1e. Adults Aged 65 Years and Older**
	Rate per Million				
Not Hispanic or Latino	2.1	3.5	6.9	17.8	86.2
Black or African American	8.3	10.3	21.9	54.0	133.5
White	DSU	2.0	3.9	12.8	80.6
Gender					
Female	1.4	2.7	4.3	22.3	99.1
Male	2.8	4.0	5.7	13.0	68.1
Education (aged 25 to 64 years)					
Less than high school	NA	NA	10.0*	31.0	NA
High school graduate	NA	NA	11.4*	22.9	NA
At least some college	NA	NA	3.9*	9.3	NA

DNA = Data have not been analyzed. DNC = Data are not collected. DSU = Data are statistically unreliable. NA = Not applicable.
*Data are for persons aged 25 to 34 years.

24-2 Reduce hospitalizations for asthma.

Target and baseline:

Objective	Age Group	1998 Baseline	2010 Target
		Rate per 10,000	
24-2a.	Children under age 5 years	45.6	25
24-2b.	Children and adults aged 5 to 64 years*	12.5	7.7
24-2c.	Adults aged 65 years and older*	17.7	11

*Age adjusted to the year 2000 standard population.

Target setting method: Better than the best.

Data source: National Hospital Discharge Survey (NHDS), CDC, NCHS.

Select Age Groups, 1998	**Hospitalizations**		
	24-2a. Children Under Age 5 Years	**24-2b. Children and Adults Aged 5 to 64 Years***	**24-2c. Adults Aged 65 Years and Older***
	Rate per 10,000		
TOTAL	45.6	12.5	17.7
Race and ethnicity			
American Indian or Alaska Native	DSU	DSU	DSU
Asian or Pacific Islander	DSU	DSU	DSU

Select Age Groups, 1998	Hospitalizations		
	24-2a. Children Under Age 5 Years	24-2b. Children and Adults Aged 5 to 64 Years*	24-2c. Adults Aged 65 Years and Older*
	Rate per 10,000		
Asian	DNC	DNC	DNC
Native Hawaiian and other Pacific Islander	DNC	DNC	DNC
Black or African American	82.4	28.4	27.3
White	29.5	7.8	12.4
Hispanic or Latino	DSU	DSU	DSU
Not Hispanic or Latino	DSU	DSU	DSU
Black or African American	DSU	DSU	DSU
White	DSU	DSU	DSU
Gender			
Female	33.1	15.9	24.6
Male	57.6	9.0	8.5

DNA = Data have not been analyzed. DNC = Data are not collected. DSU = Data are statistically unreliable.
*Age adjusted to the year 2000 standard population.

24-3. Reduce hospital emergency department visits for asthma .

Target and baseline:

Objective	Age Group	1995–97 Baseline	2010 Target
		Rate per 10,000	
24-3a.	Children under age 5 years	150.0	80
24-3b.	Children and adults aged 5 to 64 years	71.1	50
24-3c.	Adults aged 65 years and older	29.5	15

Target setting method: Better than the best.

Data source: National Hospital Ambulatory Medical Care Survey (NHAMCS), CDC, NCHS.

Select Age Groups, 1995–97	**Hospital Emergency Department Visits for Asthma**		
	24-3a. Children Under Age 5 Years	**24-3b. Children and Adults Aged 5 to 64 Years**	**24-3c. Adults Aged 65 Years and Older**
	Rate per 10,000		
TOTAL	150.0	71.1	29.5
Race and ethnicity			
American Indian or Alaska Native	DSU	DSU	DSU
Asian or Pacific Islander	DSU	DSU	DSU

Select Age Groups, 1995–97	Hospital Emergency Department Visits for Asthma		
	24-3a. Children Under Age 5 Years	24-3b. Children and Adults Aged 5 to 64 Years	24-3c. Adults Aged 65 Years and Older
	Rate per 10,000		
Asian	DNC	DNC	DNC
Native Hawaiian and other Pacific Islander	DNC	DNC	DNC
Black or African American	407.2	191.7	90.8
White	101.7	53.4	23.1
Hispanic or Latino	DSU	DSU	DSU
Not Hispanic or Latino	DSU	DSU	DSU
Black or African American	DSU	DSU	DSU
White	DSU	DSU	DSU
Gender			
Female	103.0	83.6	37.8
Male	195.5	57.9	17.9

DNA = Data have not been analyzed. DNC = Data are not collected. DSU = Data are statistically unreliable.

24-4. Reduce activity limitations among persons with asthma .

Target: 10 percent.

Baseline: 20 percent of persons with asthma experienced activity limitations in activity in 1994–96 (age adjusted to the year 2000 standard population).

Target setting method: Better than the best.

Data source: National Health Interview Survey (NHIS), CDC, NCHS.

Persons With Asthma , 1994–96	Experienced Activity Limitations
	Percent
TOTAL	20
Race and ethnicity	
American Indian or Alaska Native	DSU
Asian or Pacific Islander	DSU
Asian	DSU
Native Hawaiian and other Pacific Islander	DSU
Black or African American	26
White	18
Hispanic or Latino	22
Not Hispanic or Latino	19

Persons With Asthma , 1994–96	Experienced Activity Limitations
	Percent
Black or African American	26
White	18
Gender	
Female	21
Male	17
Family income level	
Poor	28
Near poor	20
Middle/high income	16

DNA = Data have not been analyzed. DNC = Data are not collected. DSU = Data are statistically unreliable.
Note: Age adjusted to the year 2000 standard population.

24-5 (Developmental) Reduce the number of school or work days missed by persons with due to asthma.

Potential data source: National Health Interview Survey (NHIS), CDC, NCHS.

24-6. Increase the proportion of persons with asthma who receive formal patient education, including information about community and self-help resources, as an essential part of the management of their condition.

Target: 30 percent.

Baseline: 8.4 percent of persons with asthma received formal patient education in 1998 (age adjusted to the year 2000 standard population).

Target setting method: Better than the best.

Data source: National Health Interview Survey (NHIS), CDC, NCHS.

Persons With Asthma , 1998	Received Patient Education
	Percent
TOTAL	8.4
Race and ethnicity	
American Indian or Alaska Native	DSU
Asian or Pacific Islander	DSU
Asian	DSU
Native Hawaiian and other Pacific Islander	DSU
Black or African American	11.2
White	7.8
Hispanic or Latino	7.8
Not Hispanic or Latino	8.4
Black or African American	11.3
White	7.9
Gender	

Persons With Asthma , 1998	Received Patient Education
	Percent
Female	9.1
Male	7.1
Family income level	
Poor	7.2
Near poor	10.3
Middle/high income	8.4

DNA = Data have not been analyzed. DNC = Data are not collected. DSU = Data are statistically unreliable.
Note: Age adjusted to the year 2000 standard population.

24-7. (Developmental) Increase the proportion of persons with asthma who receive appropriate care according to the NAEPP Guidelines.

24-7a. Persons with asthma who receive written management plans from their health care provider.

24-7b. Persons with asthma with prescribed inhalers who receive instruction on how to use them properly.

24-7c. Persons with asthma who receive education about recognizing early signs and symptoms of episodes and how to respond appropriately, including instruction on peak flow monitoring for those who use daily therapy.

24-7d. Persons asthma with who receive medication regimens that prevent the need for more than one canister of short-acting inhaled beta agonists per month for relief of symptoms.

24-7e. Persons with who receive follow-up medical care for long-term management of after any hospitalization due to .

24-7f. Persons with asthma who receive assistance with assessing and reducing exposure to environmental risk factors in their home, school, and work environments.

Potential data source: National Health Interview Survey (NHIS), CDC, NCHS.

24-8. (Developmental) Establish in at least 25 States a surveillance system for tracking asthma death, illness, disability, impact of occupational and environmental factors on , access to medical care, and management.

Potential data sources: Periodic surveys, Council of State and Territorial Epidemiologists and Public Health Foundation; Association of Schools of Public Health.

Chronic Obstructive Pulmonary Disease

24-9. Reduce the proportion of adults whose activity is limited due to chronic lung and breathing problems.

Target: 1.5 percent.

Baseline: 2.2 percent of adults aged 45 years and older experienced activity limitations due to chronic lung and breathing problems in 1997 (age adjusted to the year 2000 standard population).

Target setting method: Better than the best.

Data source: National Health Interview Survey (NHIS), CDC, NCHS.

Adults Aged 45 Years and Older, 1997	Experienced Activity Limitations Due to Chronic Lung and Breathing Problems
	Percent

Adults Aged 45 Years and Older, 1997	Experienced Activity Limitations Due to Chronic Lung and Breathing Problems
	Percent
TOTAL	2.2
Race and ethnicity	
American Indian or Alaska Native	DSU
Asian or Pacific Islander	DSU
Asian	DSU
Native Hawaiian and other Pacific Islander	DSU
Black or African American	2.3
White	2.3
Hispanic or Latino	1.7
Not Hispanic or Latino	2.3
Black or African American	2.2
White	2.3
Gender	
Female	2.1

Adults Aged 45 Years and Older, 1997	Experienced Activity Limitations Due to Chronic Lung and Breathing Problems
	Percent
Aged 45 to 64 years	1.6
Aged 65 years and older	3.0
Male	2.5
Aged 45 to 64 years	1.6
Aged 65 years and older	4.1
Family income level	
Poor	5.2
Near poor	4.0
Middle/high income	1.8

DNA = Data have not been analyzed. DNC = Data are not collected. DSU = Data are statistically unreliable.
Note: Age adjusted to the year 2000 standard population.

24-10. Reduce deaths from chronic obstructive pulmonary disease (COPD) among adults.

Target: 60 deaths per 100,000 adults.

Baseline: 119.4 deaths from COPD per 100,000 persons aged 45 years and older occurred in 1998 (age adjusted to the year 2000 standard population).

Target setting method: 50 percent improvement.

Data source: National Vital Statistics System (NVSS), CDC, NCHS.

Adults Aged 45 Years and Older, 1998	Chronic Obstructive Pulmonary Disease Deaths
	Rate per 100,000
TOTAL	119.4
Race and ethnicity	
American Indian or Alaska Native	79.6
Asian or Pacific Islander	48.6
Asian	DNC
Native Hawaiian and other Pacific Islander	DNC
Black or African American	85.3
White	124.3
Hispanic or Latino	52.5
Not Hispanic or Latino	122.8
Black or African American	87.2
White	127.9
Gender	
Female	98.8

Adults Aged 45 Years and Older, 1998	Chronic Obstructive Pulmonary Disease Deaths
	Rate per 100,000
Male	153.7
Education (aged 25 to 64 years)	
Less than high school	19.7
High school graduate	12.5
At least some college	4.4

DNA = Data have not been analyzed. DNC = Data are not collected. DSU = Data are statistically unreliable.
Note: Age adjusted to the year 2000 standard population.

Obstructive Sleep Apnea

24-11. (Developmental) Increase the proportion of persons with symptoms of obstructive sleep apnea whose condition is medically managed.

24-11a. Persons with excessive daytime sleepiness, loud snoring, and other signs associated with obstructive sleep apnea who seek medical evaluation.

24-11b. Persons with excessive daytime sleepiness, loud snoring, and other signs associated with obstructive sleep apnea who receive follow-up medical care for long-term management of their condition.

Potential data source: National Health Interview Survey (NHIS), CDC, NCHS.

24-12. (Developmental) Reduce the proportion of vehicular crashes caused by persons with excessive sleepiness.

Potential data source: National Health Interview Survey (NHIS), CDC, NCHS. Fatality Analysis Reporting System (FARS), U.S. Department of Transportation, National Highway Traffic Safety Administration.

Related Objectives From Other Focus Areas

1. Access to Quality Health Services

1-10. Delay or difficulty in getting emergency care

7. Educational and Community-Based Programs

7-8. Satisfaction with patient education

7-11. Culturally appropriate and linguistically competent community health promotion programs

8. Environmental Health

8-1. Harmful air pollutants

8-2. Alternative modes of transportation

8-3. Cleaner alternative fuels

8-4 Airborne toxins

8-14 Toxic pollutants

8-16. Indoor allergens

8-17. Office building air quality

8-20. School policies to protect against environmental hazards

8-23. Substandard housing

8-26. Information systems used for environmental health

8-27. Monitoring environmentally related diseases

8-28. Local agencies using surveillance data for vector control

11. Health Communication

11-6. Satisfaction with health care providers' communication skills

15. Injury and Violence Prevention

15-15. Deaths from motor vehicle crashes

15-17. Nonfatal motor vehicle injuries

20. Occupational Safety and Health

20-1. Work-related injury deaths

20-2. Work-related injuries

20-4. Pneumoconiosis deaths

22. Physical Activity and Fitness

22-6. Moderate physical activity in adolescents

22-7. Vigorous physical activity in adolescents

23. Public Health Infrastructure

23-2. Public access to information and surveillance data

23-4. Data for all population groups

23-6. National tracking of Healthy People 2010 objectives

23-7. Timely release of data on objectives

23-10. Continuing education and training by public health agencies

23-16. Data on public health expenditures

23-17. Population-based prevention research

27. Tobacco Use

27-1. Adult tobacco use

27-2. Adolescent tobacco use

27-3. Initiation of tobacco use

27-4. Age at first tobacco use

27-5. Smoking cessation by adults

27-6. Smoking cessation during pregnancy

27-7. Smoking cessation by adolescents

27-8. Insurance coverage of cessation treatment

27-9. Exposure to tobacco smoke at home among children

27-10. Exposure to environmental tobacco smoke

27-11. Smoke-free and tobacco-free schools

27-12. Worksite smoking policies

27-13. Smoke-free indoor air laws

27-14. Enforcement of illegal tobacco sales to minors laws

27-15. Retail license suspension for sales to minors

27-16. Tobacco advertising and promotion targeting adolescents and young adults

27-17. Adolescent disapproval of smoking

27-18. Tobacco control programs

27-19. Preemptive tobacco control laws

27-20. Tobacco product regulation

27-21. Tobacco tax

Terminology

(A listing of abbreviations and acronyms used in this publication appears in Appendix H.)

Ambulatory care: Medical care provided at hospital emergency and outpatient departments.

Asthma: A lung disease characterized by airway constriction, mucus secretion, and chronic inflammation, resulting in reduced airflow and wheezing, coughing, chest tightness, and difficulty breathing.

Chronic bronchitis: A lung disease characterized by the presence of chronic productive cough most days for 3 months in each of 2 successive years.

Chronic obstructive pulmonary disease (COPD): A lung disease characterized by airflow obstruction due to chronic bronchitis and emphysema, two diseases that often occur together. COPD is one of the most common respiratory conditions among adults worldwide and is the fourth leading cause of death in the United States.

Dyspnea: Shortness of breath.

Emphysema: Abnormal permanent enlargement of the airspaces in the lungs accompanied by coughing and difficulty breathing.

Epidemiologic studies: Studies of disease occurrence.

Obstructive sleep apnea (OSA): An illness characterized by snoring, partial or complete cessation of breathing during sleep, reductions in blood oxygen levels, severe sleep disruptions, and excessive daytime sleepiness. OSA is a chronic breathing problem with serious effects on individual health and productivity, including an inheritable risk of sudden infant deaths, behavior and learning disturbances, injury from accidents, and reduced quality of life.

Rate: The basic measure of disease occurrence that most clearly expresses the probability of risk of disease in a defined population over a specified period of time. A rate is defined as:

$$\frac{\text{Number of events}}{\text{Population at risk}}$$

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RESPIRATORY DISEASES

PART II. LEARNING OBJECTIVES

Upon completion of this module, the student will:

1. Differentiate between the various chronic obstructive pulmonary diseases.
2. Differentiate between the various restrictive pulmonary diseases.
3. Describe the pathophysiology of the infectious respiratory disease processes.
4. Discuss the pathophysiology of pulmonary embolic disease.
5. Summarize the effects of pulmonary pathology due to trauma.

PART III. ASSIGNED READING

As there are no clinical procedures to perform for this module, the student is expected to spend more time on assigned readings. Because of the wide array of disease states the therapist encounters, familiarity obtained by these readings will aid in proper diagnosis and treatment.

For your own benefit, we highly recommend reviewing the supplemental reading, however it is not required.

Burton and Hodgkin, Respiratory Care - A Guide to Clinical Practice. **Philadelphia: J.B. Lippincott Company.**

RESPIRATORY DISEASES

PART IV. CONTENT OUTLINE

Unit 1. Introduction

Unit 2. Chronic Obstructive Pulmonary Disease:

Chronic Bronchitis

Pulmonary Emphysema

Bronchiectasis

Asthma

Small Airways Disease

Cor Pulmonale

Cystic Fibrosis

Unit 3. Restrictive Pulmonary Disease:

Interstitial Pneumonia

Pneumoconiosis

Sarcoidosis

Thoracoskeletal Disease of the Chest Wall

Neuromuscular Disorders

Pickwickian Syndrome

Pneumothorax

Pleural Effusion

ARDS

Pulmonary Edema

Unit 4. Infectious Pulmonary Disease:

Pneumonia

Pulmonary Fungal Infection

Pulmonary Tuberculosis

Unit 5. **Pulmonary Embolic Disease**

Unit 6. **Pulmonary Pathology Due to Trauma:**

Burns/Smoke Inhalation

Penetrating Chest Wounds

Flail Chest/Rib Fractures

Gastric Aspiration

Near Drowning

Hypothermia

RESPIRATORY DISEASES

PART V. INSTRUCTIONAL CONTENT

Unit 1. Introduction:

To cover the entire topic of pulmonary diseases in a single module is a difficult if not impossible task. In order to provide the respiratory therapy student with a sound general concept of the pathophysiology of the majority of pulmonary disease, this module has been broken down into several major categories:

1. Chronic obstructive pulmonary disease
2. Restrictive pulmonary disease
3. Infectious pulmonary disease
4. Pulmonary embolic disease
5. Pulmonary pathology due to trauma

Before beginning the discussion of the individual diseases, it is important for the student to be able to differentiate between obstructive and restrictive disease and acute and chronic disease states. Obstructive pulmonary disease is generally defined as a state in which there is an obstruction to expiratory air flow. This disorder can be determined by simple spirometry. On the other hand, restrictive pulmonary disease is defined as disease processes in which there is a restriction to inspiratory air flow. Both obstructive and restrictive diseases can occur acutely and chronically.

Acute pulmonary disease is defined as one in which the onset of symptoms is rapid and severe. An acute disease usually will follow a shorter course than chronic disease states. Chronic disease is defined as any disease with a long, drawn-out course. Acute and chronic are antonyms.

Each adult pulmonary disease will be further discussed according to certain major characteristics. The following terms will be used in the discussion:

1. Etiology: The cause of the disease.
2. Incidence: The frequency of occurrence of the disease in relation to the population.
3. Pathogenesis: The origin and development of the disease.
4. Pathology: The conditions produced by the disease.
5. Clinical Manifestations: Those manifestations which can be clinically observed by actual patient observation.
6. Radiographic Examination: Data obtained by x-ray.
7. Sputum Examination: Data obtained from sputum collection.
8. Laboratory Findings: Data obtained from laboratory tests.
9. Pulmonary Function Studies: Data obtained from PFT's.
10. Diagnosis: Establishing the name of the disease by evaluation of the above data.
11. Management: How the disease is treated.
12. Disease Course: The progression of the disease.
13. Prognosis: Prediction of the final outcome of the disease.

An attempt will be made to cover each of these aspects of the individual pulmonary diseases. A great deal of additional information may be obtained from the resource readings and it will be most important for the student to study these sources to obtain in-depth knowledge of pulmonary pathology.

Unit 2. Chronic Obstructive Pulmonary Disease:

Chronic obstructive pulmonary disease (COPD) is a title given to a number of disease entities that are all similar and often difficult to distinguish from one another. This is due in part to the fact that they often overlap; the patient may well have more than one of the disease entities.

Generally, COPD includes chronic bronchitis and pulmonary emphysema. Closely associated with these diseases are bronchiectasis, cystic fibrosis, bronchiolitis and small airway disease. Bronchiolitis and cystic fibrosis are predominantly pediatric disorders and should be covered at length in a pediatric module.

The chronic obstructive pulmonary diseases have much in common. They tend to be chronic illnesses and exhibit an obstructive component in pulmonary function studies. Because of these similarities, treatment is often much the same for each of the COPD states.

Chronic Bronchitis:

Chronic bronchitis is best defined as a chronic productive cough for at least three months of the year for two successive years. The patient with "asthmatic bronchitis" suffers from chronic bronchitis accompanied by frequent bouts of bronchospasm.

Incidence

Chronic bronchitis is found in 10 to 20% of the adult population. It is more common in men than women (a 10:1 ratio).

Etiology

The key factor in the etiology of chronic bronchitis is cigarette smoking. There is also an increased incidence of the disease in areas associated with air pollution.

Infection can cause exacerbation of the bronchitis into an acute state. Anything causing prolonged irritation of the bronchial mucosa can cause chronic bronchitis.

Pathology and Pathogenesis

The primary pathological feature of chronic bronchitis is inflammation of the airways. This inflammation leads to hypertrophy of the bronchial mucosal glands and an increase in the number of goblet cells. Increased amounts of mucus are produced. The bronchial mucosa becomes edematous.

The increased mucus production, mucus gland hypertrophy and edematous tissues cause increased airway resistance. This is the cause of the obstructive component of chronic bronchitis.

The above changes are accompanied by mucociliary impairment and some actual loss of cilia. Phagocytic activity is decreased by the increase in mucus production and decreased mucus infection.

Often, chronic bronchitis is accompanied by vascular changes due to the hypoxia that it causes. Pulmonary hypertension and an increase in pulmonary vascular resistance may occur and lead to right ventricular hypertrophy or cor pulmonale.

Clinical Manifestations

The major symptom of chronic bronchitis is the cough accompanied by excessive expectoration. The cough is usually loose and "rattling," worsening in the morning and at night. Sputum is mucoid and difficult to expectorate. It varies in color from white to yellow or gray.

Respiratory difficulties often appear at about thirty years after the onset of lung injury. Shortness of breath is not marked, however, unless CHF is involved.

The chronic bronchitis patient often suffers frequent attacks of acute bronchitis in periods of damp or cold weather.

The physical examination reveals either a normal or "barrel" chest. Rhonchi and wheezes may be heard throughout the lung fields and expiration time is often prolonged. In extreme cases, cyanosis may present, along with clubbing of the digits. In the later stages, pulmonary hypertension may cause neck vein distention and peripheral edema.

Laboratory Findings

Pulmonary function tests indicate obstructive airway disease. The flow rate measurements (including FEV_1, FEV_1/FVC and FEF_{25-75}) are decreased. The degree of abnormality determines whether the disease state is mild, moderate or severe.

Arterial blood gases may exhibit a carbon dioxide retention accompanied by hypoxemia. A second polycythemia may also be present due to the chronic hypoxemia.

Roentgenographic Findings

The chest x-ray is normal in 20 to 40% of the cases of chronic bronchitis. This usually will occur if mainly the larger airways are affected. If the more peripheral bronchi are involved, hyperinflation and a flattened diaphragm may be observed. Increased bronchovascular markings may be present and, on bronchography, dilated bronchial glands may be seen.

Treatment

The first step in the treatment of chronic bronchitis is to have the patient stop smoking. Air pollution and occupational hazards should also be avoided.

The patient should be taught to maintain good bronchial hygiene. Effective cough methods should be taught, along with postural drainage.

A high fluid intake should be encouraged to promote hydration of secretions. If the patient develops an upper respiratory infection, early antibiotic therapy should be instituted.

Bronchodilator therapy and expectorants may be helpful. In the patient with "tic bronchitis," steroid therapy is often used.

Pulmonary Emphysema:

Emphysema is a pathological process characterized by an abnormal increase of the respiratory portion of the lung beyond the terminal bronchioles with attenuation and loss of pulmonary septal tissues. It is characterized by dilation and destruction of all or part of the acinus, the tissue distal to the terminal bronchioles.

There are may different types of emphysema. The following types will be discussed:

1. Centrilobular emphysema
2. Panlobular emphysema
3. Focal dust emphysema
4. Paraseptal emphysema
5. Alveolar-duct emphysema
6. Paracitatrical (scar) emphysema
7. Unilateral (Swyer-James' syndrome, hyperlucent lung syndrome and MacLeod's syndrome) emphysema

In addition, Alpha-1 antitrypsin deficiency will be discussed.

Emphysema is considered to be an obstructive disorder not because the airways are narrowed, but because there is premature airway closure on expiration due to disruption of the alveolar septum and a subsequent loss of lung elasticity.

Centrilobular emphysema primarily affects the middle of the acinus and the respiratory bronchioles. The centrilobular type of emphysema is often found in patients with chronic bronchitis. It is common for patients with this variety of emphysema to retain secretions and develop respiratory infections. This type of emphysema occurs primarily in the upper two-thirds of the lung fields and is seldom seen in nonsmoking patients.

Panlobular (panacinar) emphysema involves the entire acinus. The alveoli tend to enlarge and appear very similar to the alveolar duct. This type of emphysema is found mainly in the lower lobes. Panlobular emphysema often occurs in the elderly and, like centrilobular emphysema, is more often found in males than in females. However, females with emphysema are more likely to have panlobular emphysema than centrilobular emphysema.

Focal dust emphysema is characterized by inhaled dust in the respiratory and terminal bronchioles accompanied by dilation of the bronchioles. There is little, if any, impairment of respiratory function.

Paraseptal (lineal) emphysema is usually a localized, asymptomatic form of emphysema and is thought to be a major cause of spontaneous pneumonthorax. The lung periphery and the pleura are primarily involved. Bullae may develop from disrupted alveoli and it is thought that this type of emphysema may be an element of bullous emphysema.

Alveolar-duct emphysema is commonly found in elderly patients and is characterized by an increase in the diameter of the alveolar ducts. The patient with alveolar-duct emphysema is rarely symptomatic. The cause of the disease is most likely due to airway dilation as a direct result of the aging process.

Paracitatrical (scar) emphysema occurs in areas of pulmonary scarring and is characterized by small areas of alveolar destruction and distention.

Unilateral emphysema is also known as MacLeod's syndrome, hyperlucent lung and Swyer-James' syndrome. This disease is characterized by one lung being more radiolucent than the other.

The affected lung is often afflicted with bronchitis, bronchiolitis and air trapping. Patients with this disease, however, are often asymptomatic. The hyperlucency is most likely due to decreased vascular markings and widespread emphysema in that lung. The etiology in the majority of cases is most likely infection.

Alpha-1 (α-1) antitripysin deficiency is a genetic abnormality resulting in emphysematous lung tissue. Alpha-1 antitrypsin is the major proteinase inhibitor in normal adult plasma.
When a deficiency of this substance is present, the lung parenchyma is destroyed by leukocyte proteases. Panlobular emphysema is usually seen and affects mainly the lower lobes.

Those with severe deficiency are usually homozygous (possess like pairs of genes with the genetic abnormality) and often develop the disease at age thirty five or older. Those with a milder deficiency are usually heterozygous and become symptomatic at approximately fifty years or older.

Alpha-1 antitrypsin deficiency is responsible for less than 5% of the reported cases of emphysema. One-third of the patients thirty five years old or less exhibit an alpha-1 antitrypsin deficiency. Genetic counseling should be given to patients with this disorder to prevent reoccurrence.

Etiology

The etiology of emphysema is questionable, but cigarette smoking and environmental pollutants are definitely key causative factors. Infection, retained secretions in the small airways and subsequent air trapping can all cause destruction and distention of the alveolar tissue. As mentioned earlier in the various types of emphysema, old age, repeated respiratory infections, heredity and congenital anomalies can also be etiologic factors.

Pathology and Pathogenesis

Emphysema begins with small holes or fenestrations in the alveolar membranes. Eventually as these fenestrations increase in number, they become larger; breaking, tearing and rupturing the alveolar membrane. Septal destruction also appears due to frequent coughing, hyperinflation and air trapping.

Unfortunately, this alveolar destruction occurs long before the patient becomes symptomatic. As the alveoli undergo these changes, there is a subsequent loss of surface area available for diffusion of gases.
When this occurs, arterial blood gas values become abnormal and hypoxemia and carbon dioxide retention may present. Diffusing capacities decrease and compliance increases as elasticity decreases. The lung tends to over-expand and become larger as the loss of elasticity continues. A "barrel" shaped appearance of the chest results. The ribs have a tendency to separate and the diaphragm flattens to allow for the increased lung expansion.

Clinical Manifestations

Shortness of breath on exertion is the earliest clinical sign of emphysema. Work of breathing is greatly increased as more and more effort is required to exhale, due to airway collapse and air trapping. Cough and expectoration may be present, but are generally not severe in the early stages of emphysema. Eventually, the patient will experience generalized symptoms of orthopnea, weight loss, loss of appetite, inactivity and depression. On physical examination, a prolonged expiratory phase is encountered. End-expiratory wheezing may be present and breath sounds may be decreased to absent. There is hyper-resonance to percussion and a decrease in tactile fremitus. Chest expansion decreases, A-P diameter increases, work of breathing increases and there is often a "barrel" chest appearance.

Roentgenographic Findings

The chest x-ray shows a flattened diaphragm, overall hyperinflation, a vertical heart and a possible decrease in pulmonary vascular markings.

There is an overall hyperlucency of the lung fields. The chest x-ray is not a good tool in the diagnosis of emphysema, as these symptoms are also seen in other pulmonary diseases.

Laboratory Findings

Pulmonary function studies, of course, show obstructive pulmonary disease. If an element of bronchitis is present, airway resistance will increase; DLCO decreases, TLC increases (as does FRC and RV) and VC may be normal or decreased. If pulmonary emphysema is the only disease present, there will generally be a poor response to bronchodilator therapy due to the nonreversible components of airway collapse. Helium and nitrogen washout tests show poor distribution of ventilation.

Arterial blood gases usually indicate hypoxemia and possible carbon dioxide retention. The degree of severity of these abnormalities is dependent upon the extensiveness of the emphysema, the presence of other diseases and the occurrence of exacerbations.

Treatment

Emphysema is an irreversible disease state, but with treatment the parenchymal damage may be prevented from going any further. Treatment includes avoidance of smoking and airway irritants. Good bronchial hygiene, nutrition and adequate fluid intake are encouraged. If there is a significant response to bronchodilator therapy, it should be instituted. A good pulmonary rehabilitation program is indicated in the patient with emphysema. If necessary, supplemental oxygen and respiratory therapy may be used in the patient's home.

Bronchiectasis:

Bronchiectasis is a disease characterized by permanent irreversible dilation of either the bronchi, bronchioles or both. It is diagnosed with bronchography. A cardinal symptom is expectoration of purulent sputum.

Incidence

Since antibiotic therapy has been introduced, bronchiectasis occurs much less frequently than in the past.

Etiology

Bronchiectasis can be caused by a number of pulmonary insults. As a rule, bronchiectasis occurs following a primary insult accompanied by pulmonary infection. The following processes have been implemented as predecessors to bronchiectasis: necrotizing pulmonary infections, repeated pneumonias, cystic fibrosis, immunologic deficiency status, pulmonary neoplasm, tuberculosis, Kartagner's disease (bronchiectasis and sinus inversus), foreign body aspiration and Agammoglo bulinema.

Pathology and pathogenesis

There are three types of bronchiectasis:

1. Cylindrical bronchiectasis
2. Saccular bronchiectasis
3. Mixed Bronchiectasis

In the cylindrical or tubular type of bronchiectasis, fusiform (tapered at the end) dilations occur within the airway. The dilated airways then fill with purulent secretions. This type of bronchiectasis often occurs as a result of severe bronchitis.

Saccular bronchiectasis is characterized by irregular "grape-like" dilations and narrowing in the airways. Again, these airways fill with pooled purulent secretions.

The third type of bronchiectasis is a mixture of the other two types and is rightfully termed mixed bronchiectasis.

The exact mechanisms causing bronchiectasis are unknown. It is thought that repeated attacks to the airway by various disease states cause damage to the epithelial tissue, inflammation, narrowing of the airway alternating with dilation of the airway and finally erosion of the bronchial wall.

Clinical Manifestations

The patient with bronchiectasis presents with a productive cough of large amounts of foul-smelling purulent sputum. Sputum production peaks in early morning and immediately after the patient rises. In 50% of the cases of bronchiectasis, hemoptysis is present. A characteristic of sputum in severe cases of bronciectasis is its quality of settling into three layers: cloudy on top, saliva in the middle and purulent mucus on the bottom.

Bronchiectasis may be present for a period of time with no symptoms noted. Dyspnea will not normally be present in the bronchiectasis patient. Often in more advanced cases, clubbing, malnutrition, sinusitis, cor pulmonale, rales and hypoxemia may be present.

Laboratory Findings

Pulmonary function studies are nonspecific in the diagnosis of bronchiectasis and usually indicate obstructive airway disease. Significant hypoxemia may be present due to a "shunt effect."

Roentgenographic Findings

Standard chest x-ray results are generally negative, although increased markings may be present within localized areas. The x-ray may show some cystic spaces with recognizable air-fluid levels and "honeycomb" appearance of fibrosis surrounding emphysematous areas.

The bronchogram, however, is a more definite test for a diagnosis of bronchiectasis. Bronchography is the process of instilling a radio-opaque substance into the lung which will illustrate the size and appearance of the tracheobronchial tree. In bronchiectasis, the narrowing and dilation of the airways can be seen along with pooling of secretions within these areas.

Treatment

The major component of a treatment regimen for bronchiectasis is a good bronchial hygiene program and chest physiotherapy to remove retained secretions. Appropriate antibiotic therapy should be institued to treat pulmonary infections. A good general rule in the treatment of bronchiectasis is to treat it much the same as chronic bronchitis.

Asthma:

More than twelve million Americans suffer from asthma, a chronic obstructive pulmonary disease that makes the lungs extremely sensitive. In response to allergens or exertion, the airway mucosa becomes inflamed, the smooth muscles spasm, and the secretory cells increase their production. The mechanism behind asthma is a normal reflex of the sentries that protect the human body against invasions by allergens or infection. It's simply a defense that has become too powerful for the body's own good. For some reason, the immune cells that guard an asthmatic's bronchial tubes are in a perpetual state of inflammation. Like jittery soldiers firing at a shadow, the immune cells compound the problem by overreacting to triggers.

Triggers:

1. Allergies
2. Infections
3. Inhalants
4. Emotions

Upon exposure to these perceived enemies, the cells fire so many antibodies that airways to the lungs become jammed with mucus and squeezed by bands of constricting muscle, triggering an episode.

Asthma can produce severe shortness of breath, inability to perform routine activities, and if improperly treated, can result in respiratory failure and death. Each individual suffers a different level of severity. Almost all patients with asthma do enjoy a reversal of symptoms until something triggers the next episode.
Estimates of direct and indirect costs incurred by asthmatics in 1990 totaled $6.2 billion. related morbidity and mortality have increased in recent years in the United States. The incidence of asthma is rising. Asthma kills fourteen Americans of all ages every day, and asthma is the number one chronic disease afflicting American children.

Between seven and twenty million Americans have asthma, and approximately 5% of all Emergency Room visits are due to asthma (National Asthma Education Program Expert Panel, 1999). Asthma affects 4% to 5% of the population. There are at least two genetic influences. One is associated with the capacity to develop allergies (atopy) and the second is associated with the tendency to develop hyper-responsiveness of the airways independent of atopy. Genetic researchers have recently identified a region (locus) on chromosome eleven that is associated with atopy. This locus may contain an abnormal gene that encodes a part of the IgE receptor. In asthma, the normal airway function designed to protect the lungs (muscle contraction, mucosal swelling and mucus formation), becomes excessive. For reasons not entirely known, the airways become abnormally sensitive to infection, weather, exercise, irritants and allergens. The muscles tighten (bronchospasam) and the mucosa begins to swell, which reduces the diameter of the airway. In addition, mucus production is increased, sometimes forming sticky plugs in the bronchial tubes.

Asthma is a common chronic lung disease that affects individuals of all ages. It is an inflammatory disease characterized by hyper-responsiveness of the airways and episodic periods of bronchospasm (spasm, or prolonged contractions, of the bronchial smooth muscle). Asthma is the Greek word for panting, imposing a heavy burden of misery, and danger. The disease is characterized by the presence of reversible airway obstruction, airway inflammation, and a variable degree of airway hyper-responsiveness.

Allergens, viral illnesses, exercise, cold, weather, certain medications, and stress are all factors that exacerbate intermittent episodes of bronchospasm. Biochemical, autonomic, immunologic, infectious, endocrine, and psychologic factors are involved in varying degrees. Most asthmatic episodes of bronchospasm are short-lived, with freedom from symptoms between episodes; although airway inflammation is present, even in asymptomatic individuals.

The symptoms of asthma arise when the "caliber" of the tiny air passages that conduct the air in and out of the lungs is reduced. These tubes, or bronchi and bronchioles, are narrowed by the contraction of the thin layer of muscles that sheathes each airway. In addition, the bronchial walls become inflamed or swollen from the secretion of thick, tenacious mucous in the bronchial tubes. The inflammation resulting in hyperresponsiveness of the airways is the major pathological feature of all types of. The release of inflammatory mediators produces bronchial smooth muscle congestion, microvascular congestion, increased tracheobronchial secretions, and muscosal edema.

Asthma is a unique combination of clinical and pathological findings. It combines bronchial hyperresponsiveness with a distinctive form of airway inflammation associated with increased mucous production. The bronchial biopsy of even the mild asymptomatic individual with demonstrates epithelial disruption, eosinophilic proliferation, actively degranulating mast cells, and an increase in activated T-lymphocytes.

The way the asthmatic inflammatory process manifests itself is disease specific with the eosinophil playing a major role and activated cytokines orchestrating the inflammatory component of this illness. Asthma may be clinically indistinguishable from bronchiolitis in infants and toddlers, becomes a more easily definable illness from childhood to middle age, and then blends with a component of chronic obstructive lung disease in older adults.

Allergy in is an immediate or type I hypersensitivity reaction that requires the presence of specific immunoglobulin E (IgE) class of antibodies. This type of reaction occurs in individuals who have atopy, which is a hypersensitivity state with a genetic predisposition characterized by the production of an excessive amount of IgE antibodies and against a variety of antigens.

About 10% to 20% of the general population are atopic and have the tendency to develop hay fever, eczema, and other IgE- mediated allergic reactions. These types of hypersensitivity reactions result from the interaction of antigens (allergens) with their specific IgE antibodies, which tend to attach to the mast cells, basophilic ganulocytes, and perhaps other cells. Mast cells have the highest concentration of IgE molecules on their surface.
The cross-linking of two IgE antibody molecules by specific antigen signals the initiation of a series of intracellular biochemical events resulting in the release of several mediators. Some of these mediators are preformed and stored at specially stainable granules, and many others, before being released, are rapidly synthesized as a result of the signal from the antigen-antibody interaction.

Among the many chemical mediators identified thus far, important ones are: histamine, eosinophyil chemotactic factor of anaphylaxis (EFC-A), neutrophil chemotactic factor (NCF), leukotrienes (formerly known as slow-reacting substance of anaphyulaxis, or SRS-A), prostaglandins, and platelet-activating factor. In addition to mast cells, other cells, including macrophages, neutrophils, eosinophils, and endothelial cells, are known to produce many of these mediators as well as many cytokines. Both T lymphocytes and B lymphocytes are also active participants and interact with other cells in causing and maintaining airway inflammation and other heightened reactivity. Plasma cells, made from B-lymphocytes, are responsible from production of IgE antibodies.

The pathophysiologic changes during an asthmatic episode are based on the narrowing of the airways by contraction of smooth muscles, mucosal and submucosal edema, and increased secretions. Increased respiratory airway resistance results in decreased forced expiratory flow rates and hyperinflation. Increased respiratory work load is related to the increase in airway resistance. In addition to mechanical dysfunction, abnormal distribution of both ventilation and perfusion with their mismatching, results in alteration of the arterial blood gases, particularly hypoxemia. More severe and prolonged episodes may culminate in hyercapnia from worsening of ventilation-perfusion mismatching and ventilatory muscle fatigue.

Although the exact mechanism of airway hyper responsiveness is unknown, several factors, including genetic predisposition, autonomous nervous imbalance and the alteration of adrenergic receptors have been implicated in its development. Since everyone's asthma is different, medications come in different forms: liquids, pills, powders, vapors, and injections. Medications are given in different ways to different people.

Although drug companies sell medications under many brand names, there are only a few major types: inhaled bronchodilatiors, oral bronchodilators, corticosteroids, phosphodiesterace inhibitors, cholinergic blockers, anti-leukotrienes, and prophylactic bronchodilators to prevent the degranulization of the mast cell.

TRIGGERS AND TREATMENTS

Knowing what triggers the asthmatic episode, and following a proper medication plan, even on days when asthma isn't a problem, are an asthmatic's keys to maintaining a normal lifestyle.

MEDICATIONS:

RELIEVERS:

Reliever medications act quickly to relieve bronchoconstriction and its accompanying acute symptoms.

CONTROLLERS:

Controllers are medications taken daily on a long-term basis that are useful in getting and keeping persistent asthma under control.

RELIEVERS	CONTROLLERS
Short-acting Beta-2 agonists	Anti-inflammatory
Proventil, Ventolin, Maxiar,	Azmacort, Flovent Tilade,

Alupent, Brethaire

Anticholinergics

Atrovent

Short-acting Theophylline

Systemic Corticosteroids

Prednisone

Beclovent, Vanceril Aerobid, Intal

Long-acting Beta-2 agonists

Serevent

Anti-leukotrienes

Zyflo, Accolate

Long-acting Theophylline

Unidur, Theodur, Uniphyl

AEROSOLIZED MEDICATIONS

Alpha Agonist

Phenylephrine: NEO- SYNEPHRINE

Catecholamines

Epinephrine

Racemic Epinephrine: NEFRIN

Isoproterenol: ISUPREL

Isoetharine: BRONKOSOL

Beta Agonists

Bitolterol mesylate: TORNALATE

Resorcinols

Metaproterenol: ALUPENT, METAPREL

Fenoterol

Terbutaline: BRICANYL, BREATHAIRE

Saligenins

Salbutamol (Albuterols): VENTOLIN, PROVENTIL

Pirbuterol acetate: MAXAIR AUTOHALER

Sameterol Xinafoate: SEREVENT

Carbuterol

Cholinergic Blockers

Atropine

Ipratropium: ATROVENT

Prophylactic Bronchodilators

Cromolyn Sodium: INTAL

Corticosteriods

Beclomethasone Dipropionate: BECLOVENT, VANCERIL

Betamethasone

Dexamethasone: DECADRON, RESPIHALER

Flunisolide: AEROBID, AEROBID-M

Prednisone: PEDIAPRED

Triamcinolone: AZMACORT

Asthmatic episodes can be brief and infrequent, or long and debilitating. Most fatalities result from a condition termed “status asthmaticus” in which the airways become completely plugged. The diameter is reduced and the airway resistance is increased.

FOR SUFFERERS, KNOWLEDGE IS POWER

Asthma is now receiving the attention it deserves as a potentially life-threatening but controllable disease.

Asthma is a major public health concern in the United States. As indicated earlier in this monograph, morbidity and mortality has risen substantially over the past 10 to 15 years, as have costs associated with treatment. Considering the significant clinical and financial implications surrounding this disease, health providers need to recognize that patient education is one of the most powerful tools for helping patients gain more adequate control of their disease. A dedicated, collaborative effort on the part of various members of the health care team to educate patients, will ensure their compliance to self-management and allow for attainment of therapeutic goals.

Laboratory Findings

Arterial blood gases will show hypoxemia with severity dependent on the severity of the attack. Hypercapnia may also be present if the attack is severe. Sputum culture may show eosinophilia in asthma . If asthma is due to allergies, a skin test will be positive for specific antigens.

Pulmonary function studies during an attack will show decreased VC, FVC and flow rates; FRC, TLC and RV, however, will be increased due to air trapping.

Roentgenographic Findings

The chest x-ray will show hyperinflation and an increased A-P diameter. Areas of atelectasis may be present.

Treatment

Treatment of the asthma attack deals mainly with bronchodilation. This consists of a loading dose of aminophylline followed by an intravenous drip, aqueous epinephrine (adrenaline) injection and aerosolized bronchodialators (i.e., Albuterol).

In addition, corticosteroid therapy may be instituted for several days to reduce inflammation in the airways. If infection if present, antibiotic therapy is instituted. Fluid intake should be encouraged to promote secretion hydration.

Arterial blood should be analyzed and, if necessary, intubation and assisted ventilation should be instituted.

Long-term treatment of asthma includes avoidance of contact with specific allergens, inhaled irritants, extremes in humidity and temperature, emotional upset and several medications (aspirin, Inderol, Indocin and motrin).

Several medications may be used for long-term therapy including bronchodilators, expectorants, antibiotic, corticosteroids, cromolyn sodium, antihistamines and tranquilizers.

Small Airway Disease:

Small airways disease has also been referred to as early bronchitis or bronchiolitis. It is said to be "early" as there is a pulmonary abnormality present, but it cannot be detected by routine pulmonary function studies.

Etiology

The etiology of small airways disease would be the same as for bronchitis, including such factors as cigarette smoking, air pollution, recurrent infection, or similar conditions capable of causing prolonged irritation of the bronchial mucosa.

Pathology

The small airways less than 2 mm in diameter demonstrate early closure and are inflamed. This early airway closure is increased with inhaled irritants.

Clinical Manifestations

The patient with small airway disease may not even be aware of the problem. Minor symptoms, such as dyspnea on exertion, may be present, along with rales, expiratory wheezing and a prolonged expiration.

Laboratory Findings

Pulmonary function studies show restrictive flow patterns. The chest x-ray will likely be normal with the exception of those patients with small areas of atelectasis.

Treatment

The major treatment of small airway disease is removal of the causative agents, such as cigarettes or irritants in the air.

Cor Pulmonale:

Cor pulmonale is usually caused by chronic respiratory failure, as seen in the COPD patient. It is defined in the function and structure of the right ventricle of the heart resulting from lung disease.

Pathology and Pathogenesis

Hypoxemia and hypercarbia cause constriction of the pulmonary arterioles and an increased pulmonary vascular resistance. This causes an elevation of the pulmonary artery pressure. A chronic elevation causes a chronic stress to the right ventricle, which could result in hypertrophy due to overwork. Right heart failure ensues.

Clinical Manifestations

Pulmonary artery pressure will be high. Hypoxemia, tachypnea, orthopnea, dyspnea, thoracic pain, cyanosis, neck vein distention, a fourth heart sound and a right ventricular gallop (S-3) are found on clinical examination.

The patient will exhibit the clinical signs of severe dyspnea. Wheezing, prolonged expiration and rales will be heard with auscultation.

Laboratory Findings

The EKG is the most valuable diagnostic tool in cor pulmonale. The EKG may indicate right ventricular enlargement. Right heart catheterization will show increased pulmonary arterial pressures and a normal pulmonary capillary wedge pressure. Again, arterial blood gases will show severe hypoxemia and possible hypercapnia.

Roentgenographic Findings

The chest x-ray may be useful in indicating increased pulmonary vascular markings. If the cor pulmonale is long standing, right ventricular enlargement may be viewed.

Treatment

Treatment of cor pulmonale involves decreasing the workload of the right ventricle by decreasing pulmonary artery pressures. If the increased workload is due to hypoxemia and acidosis, these abnormalities should be treated. In other words, if the underlying cause is found and treated, the symptoms should resolve.

Cystic Fibrosis (mucoviscidosis):

Cystic fibrosis or mucoviscidosis is a hereditary disease characterized by dysfunction of exocrine glands and manifested by chronic pulmonary disease, pancreatic insufficiency, abnormally high electrolyte concentration in sweat, and sometimes abnormalities of other organs. Although characteristically a disease of early childhood, cystic fibrosis is seen more and more in adolescents and young adults because of improvement in early diagnosis and management.

With early recognition and institution of proper treatment, pancreatic insufficiency rarely constitutes a serious problem nowadays; whereas, the pulmonary complications of cystic fibrosis escape pulmonary involvement and most patients eventually succumb.

Etiology and incidence

Cystic fibrosis is a hereditary disease transmitted as a *Mendelian recessive* trait. The single-gene carriers (heterozygotes) have no clinically demonstrable disease. If both parents are carriers, their children, regardless of their sex, would have a 25% chance of having cystic fibrosis and a 50% chance of being carriers. Thus both parents of a child with cystic fibrosis are carriers.

Cystic fibrosis is more common in whites than blacks. Its incidence in whites has been estimated to be 1 in 2000 live births. It is the most frequent lethal hereditary disease in the United States. The basic defect in cystic fibrosis has remained unknown, but it has been postulated to be due to an inborn error in metabolism.

Pathogenesis and pathology

The two separate and distinct pathophysiologic features of cystic fibrosis are *high concentration of sweat electrolytes* and *abnormality of mucus secretion and elimination*. Sweat electrolyte abnormality is present at birth and continuously throughout the patient's life. It has no relationship to severity of disease or extent of involvement of other organs. Chloride and sodium content of sweat are particularly increased, sometimes being several times normal. As a diagnostic tool, the determination of sweat electrolytes has been most valuable. Clinical consequence of this abnormality has not been of significance, except for excessive and sometimes dangerous salt loss with prolonged exposure to heat.

The abnormality of mucus, its secretion and elimination, and the resultant obstructive changes constitute the basic mechanism of the pathologic process in cystic fibrosis.

Because of the presence of large numbers of mucus-secreting elements in the pancreas and tracheobronchial tree, these organs are particularly susceptible and are almost always involved in cystic fibrosis.

Cystic fibrosis originally referred to the pathologic changes in the pancreas, where dilated glands and ducts, fibrosis, and degeneration of parenchyma are characteristic features. Pancreatic insufficiency, causing inadequate digestion and absorption of food, may result in severe malnutrition in these patients, mostly when they are not properly treated.

The lungs are involved to varying degrees in virtually all patients with cystic fibrosis. Secretion of viscous and tenacious mucus throughout the tracheobronchial tree causes airway obstruction, which is the basis for the wide variety of pathologic changes seen in the lungs of patients dying from cystic fibrosis. Atelectasis, pneumonia, bronchiectasis, peri-bronchitis, emphysema, abscess, and fibrosis are seen in various combinations. Obstruction and retained secretions lay the ground for bacterial infection, which plays a major role in development of most of these pulmonary complications. *Staphylococcus aureus* and *Pseudomonas aeruginosa* are frequently cultured from the sputum of these patients.

Clinical Manifestations

Pancreatic insufficiency is the cause of most gastrointestinal manifestations of cystic fibrosis, which include abdominal distention, despite ravenous appetite. Sometimes obstructive complications of the gastrointestinal tract may occur. *Meconium ileus* is due to plugging of the distal end of the small intestine by putty-like meconium. The intestinal obstruction in this condition is present at birth. There are other less common extrapulmonary manifestations whose discussion is beyond the scope of this lesson. Pulmonary complications, which are by far the most important and potentially fatal manifestations of cystic fibrosis, eventually develop in all patients. The time of onset of clinical pulmonary manifestation is quite variable; they may be apparent within a few weeks of birth or may occur years later. Cough is the earliest and most common symptom. It is initially nonproductive, but may shortly be productive of thick and tenacious sputum.

Repeated bouts of respiratory-tract infection cause frequent exacerbation of these symptoms. With progressive and irreversible damage to the lungs, dyspnea is soon added to the clinical picture. Dyspnea may become quite severe with advancing disease. Hemoptysis, which may be massive, is not uncommon in these patients.

On *physical examination*, in addition to signs of malnutrition and poor body development, signs of pulmonary involvement are frequently present. The chest may be barrel-shaped and hyper-resonant to percussion. Changes in breath sounds with varying adventitious sounds are often detected. The patient may be in respiratory distress, using the accessory respiratory muscles. Cyanosis and other signs of respiratory failure, including cor pulmonale, may be apparent in more advanced stages of the disease. Clubbing of fingers and toes is a common finding.

Radiographic Study

Radiographic examination of the chest in patients with pulmonary involvement usually shows evidence of diffuse hyperinflation, increased lung markings, and irregular densities. Mucoid impaction and areas of atelectasis may be demonstrated. Frequent pulmonary infection usually manifests radiographically as new infiltration and occasionally by abscess formation. Pneumothorax and/or mediastinal emphysema may be manifest in some.

Laboratory Findings

Pancreatic insufficiency can be confirmed by special examination of stools and pancreatic secretion. The sweat test is the simplest and most reliable method for the diagnosis of cystic fibrosis. If performed properly, a positive test (i.e., a sweat chloride concentration of over 60 meq per liter in children) is diagnostic.

A pulmonary function test usually shows evidence of obstructive ventilatory impairment and hyperinflation. Vital capacity is markedly diminished, while residual volume is increased up to four or five times predicted normal.

Management

The greatest challenge in management of a patient with cystic fibrosis is related to pulmonary complications. Gastrointestinal manifestations are usually controlled by proper diet, vitamins, and pancreatic enzymes. Despite significant advances in knowledge and understanding of pulmonary problems in cystic fibrosis and availability of effective therapeutic measures, patients with this dreadful disease still succumb to pulmonary complications. Treatment of these patients should be started very early in the course of their disease before development of irreversible pulmonary damage. As was indicated earlier, the common denominator of lung lesions is obstruction of airways due to bronchial secretions. Therefore, the principle of management will center around the removal of these secretions and alleviation of obstruction.

The role of antibiotic in treatment of these patients is evident from the importance of bacterial infection in causing many of the pulmonary complications.

In considering the measures for removal of mucopurulent secretions, it should be recalled that these secretions are thick and very tenacious; therefore, the importance of proper humidification and other methods for their loosening should be emphasized. Dry air and respiratory irritants should be avoided. Use of mist tents and other methods of humidity therapy has resulted in some success in helping to reduce sputum viscosity and in making coughing and postural drainage more effective. However, the argument for and against regular use of mist tents at night continues. The results of the studies question the effectiveness of this form of therapy. Expectorants such as potassium iodide of glycerol guaiacolate have been commonly used with limited results. Mucolytic and enzymatic agents, such as acetylcysteine and pancreatic dornase, used in inhalational form, have shown some benefit.

These treatments should be followed with effective cough and postural drainage. These maneuvers should become part of the patient's daily routine. Patient's responsible family members should be thoroughly instructed in their proper use.

Gentle chest tapping, cupping, or use of mechanical devices may help the effectiveness of postural drainage. IPPB should be administered with great caution.

Antibiotic therapy is one of the most important aspects of management of patients with cystic fibrosis. The antibiotic is usually given therapeutically and, occasionally, prophylactically. Stahylococcal infection, which is the most common bacterial infection in cystic fibrosis, should be treated with a proper antistaph agent. *Pseudomonas aeruginosa*, which is frequently isolated from the sputum of these patients, is very difficult to eradicate despite use of potent antibiotics.

Lung lavage with normal saline solution has resulted in variable success in some patients with severe and life-threatening pulmonary involvement. Surgery has been rarely considered for resection of localized lesions to control massive hemoptysis.

Course and Prognosis

In most cases of cystic fibrosis, the severity of pulmonary complications determines the outcome. The degree of pancreatic insufficiency has no significant effect on the ultimate outlook if properly treated with adequate nutrition and replacement therapy. The increase in survival from less than 2 years to about 16 years in the past 30 years is mostly the result of early recognition and proper treatment. Still, some patients die in infancy or early childhood; and increasing large number survive to ages of 20 or 30 years, occasionally even longer. Death beyond the neonatal period is due to pulmonary complications such as overwhelming infection, or, frequently, respiratory failure.

Unit 3. Restrictive Pulmonary Disease:

Restrictive lung disease is characterized by a loss in lung volume. The primary factor in restrictive lung disease is a decrease in lung compliance (distensibility). This can be a result of changes in the lung tissues (parenchyma), the chest wall, or both. This decrease in lung compliance is usually the result of lung inflammation, fibrotic lung disease, neoplasms, kypohoscoliosis, and neuromuscular diseases.

As a result, lung volumes are decreased. The only single diagnostic parameter for restrictive lung disease is a significant reduction in the total lung capacity.

Included in this discussion are: interstitial pneumonia, pneumoconioses, sarcoidosis, thoracoskeletal diseases of the chest wall, neuromuscular disorders, Pickwickian syndrome, pneumothorax, pleural effusion, ARDS and pulmonary edema.

Interstitial Pneumonia:

Interstitial pneumonia is characterized histologically by alveolar wall fibroses. These pneumonias are also known as interstitial fibrosis, honey-comb lung, Hamman-Rich syndrome and cryptogenic fibrosing alveolitis.

Incidence

Interstitial pneumonia is usually seen in a person between 20 to 40 years of age regardless of sex or race.

Etiology

About 50% of the cases of interstitial pneumonia are caused by; Drug toxicity or hypersensitivity, ARDS, farmer's lung, connective tissue disease, sarcoidosis and genetic disorders. The other 50% are thought to be idiopathic.

Pathology and pathogenesis

Interstitial pneumonia occurs within the alveolar walls and the cells of the alveolar lining. These are the gas exchanging tissues of the lung. The alveolar capillaries are disrupted and the alveolar wall increases in thickness.

There are four classifications of interstitial pneumonia according to their pathology:

1. Usual interstitial pneumonia
2. Lymphocytic interstitial pneumonia
3. Desquamative interstitial pneumonia
4. Bronchiolitis obliterans or bronchial interstitial pneumonia

Clinical Manifestations

The patient with interstitial pneumonia will present with dyspnea on exertion; a dry, hacking, nonproductive cough; possible clubbing of digits; rales; and possibly cor pulmonale. Complications associated with interstitial pneumonia are spontaneous pneumothorax and carcinoma.

Roentgenographic Findings

The chest x-ray shows a pattern of diffuse reticular or nodular reticular infiltrates primarily in the basilar segments of the lung. In the advanced stages, small cystic spaces (honeycombing) are seen. Lung volumes appear reduced. Pulmonary vascular markings may be increased if cor pulmonale is present.

Laboratory Findings

Pulmonary function studies will show a decrease in lung volumes with normal flow rates. DLCO will be reduced, especially after exercise. Static lung compliance will also be reduced.

Arterial blood gases will show hypoxemia and hyperventilation. Again, exercise will worsen these values. The cause of hypoxemia is due to a ventilation/perfusion mismatch rather than a diffusion defect.

In the early stages of interstitial fibrosis, the above tests may be normal; however, with exercise, impairment is evident.

Treatment

The treatment of choice for interstitial pneumonia is corticosteroid therapy. However, once the damage is done, it is usually permanent.

Pnuemoconioses:

The pneumoconioses are defined as a group of diseases characterized by any change in the lung caused by inhaled dusts. These are also known as "occupational diseases."

Etiology

The various diseases in this group are classified according to their etiology. Table 2 shows some of the pneumoconioses and their causative factors.

Table 2

Pneumoconioses and There Causative Factors

DISEASE	EXPOSURE
Asbetosis	Asbestos
Baritosis	Barium
Aluminosis	Aluminum
Berylliosis	Beryllium
worker's pneumonia	Dust
Caolinosis	Clay
Siderosis	Iron
Silicosis	Silica Dust
Talcosis	Certain Talcs

Exposure alone to a dust does not necessarily indicate occurrence of the disease state. Many factors come into play, such as the nature, intensity and duration of exposure; the particle size of the dust; other irritating factors (i.e., cigarette smoking, air pollution); pulmonary clearance; and others.

The following pneumoconioses will be discussed:

1. Silicosis pneumoconiosis
2. Asbestosis pneumoconiosis
3. Talcosis pneumoconiosis
4. Berylliosis pneumoconiosis
5. Worker's pneumoconiosis

Silicosis is caused by inhalation of toxic silica dust. It is found predominantly in people who do sandblasting, foundry work and underground mining. Depending on the intensity of the dust, the symptoms of silicosis may show up in as little time as 18 months or up to 30 years.

Silicosis can be divided into two categories: simple and complicated. Simple silicosis is present without underlying lung disease (i.e., COPD) and is usually asymptomatic. Complicated silicosis is the more advanced stage of the disease and is usually accompanied by parenchymal fibrosis in the form of lump-like densities.

As free crystalline silica is inhaled into the lung, the toxic particles are ingested by macrophages and destroy the macrophages. More macrophages are produced and they too are destroyed after ingesting the free silica particles. Eventually, silicotic nodules are produced as the particles form collagen fibers and hyaline membranes. As more silica particles are inhaled, the amount of nodules increases and they begin to join together, enlarge and form cavitations. An added complication to silicosis is tuberculosis.

Silicosis presents clinically in varying degrees according to its severity. The patient with complicated silicosis will show symptoms of cough with expectoration, chest pain, dyspnea, wheezing and repeated respiratory infections. The patient in the late stages of the disease will show restrictive disease in pulmonary function studies along with impaired diffusion studies. Hypoxemia, pulmonary hypertension, cor pulmonale and death may ensue.

The treatment of silicosis deals mainly with preventative measures. Oxygen therapy and treatment of the congestive heart failure can be used as supportive therapy.

Asbestosis is caused by the inhalation of asbestos. This has occurred in the past in many industries, as asbestos is a commonly-used material (e.g., insulation, brake linings). There are two types of asbestos fibers: amphiboles and chrysolile. The amphibole fibers are the most dangerous to the lung as they are long and difficult for the macrophage to ingest.

When inhaled, asbestos irritates the tissues causing a pulmonary fibrosis in the terminal airways, pleura and alveoli.

The symptoms of asbestosis are dyspnea and a nonproductive cough. Roentgenographic changes may be seen in the lower lung fields and the pleura. Pleural effusions may be present. Pulmonary function tests indicate restrictive disease and decreased perfusion. In patients who suffer from asbestosis and smoke cigarettes, there is an added risk of complicating brochogenic carcinoma.

Talcosis is thought to be caused by inhalation of talc accompanied with tremolite fibers. Talcosis is characterized by large “fluffy” densities found usually in the mid-portion of the lung. Fibrosis may ensue and progress to the degree of disablement. Symptoms may be relieved with steroid therapy.

Berylliosis can be caused by the inhalation of high concentrations of beryllium over a short period of time. Following exposure, symptoms do not necessarily occur rapidly; in some cases, it may take several weeks after exposure to become symptomatic.

Symptoms include acute rhinitis, pneumonitis and tracheobronchitis. Severe hypoxemia results. Oxygen therapy and sometimes assisted ventilation are required, but there is an 80% recovery rate from the disease. The mortality rate is 10% and the remaining 10% of the victims of berylliosis suffer from a chronic condition.

Chronic beryllium granulomatosis is characterized by granulomata found in the lungs and throughout the body. The exposure to beryllium may only be slight and pass unnoticed. Susceptibility varies between, individuals and the appearance of symptoms may occur some time from the initial exposure. Symptoms of chronic berylliosis include a persistent dry cough, weight loss, severe dyspnea and sometimes fever and anorexia. Pulmonary function shows no restrictive or obstructive component, but diffusion is impaired markedly. Chest x-ray resembles sarcoidosis with hilar adenopathy. The symptoms may be temporarily relieved with steroid therapy.

Worker's pneumoconiosis ("Black Lung") is the most common form of pneumoconiosis. It is caused by inhalation of dust and is seen in workers. The severity of the disease is relative to the amount of exposure.

Worker's pneumoconiosis has two forms: early and late. In the early form, particles are seen accumulated around the respiratory and terminal bronchioles. These small airways are sometimes dilated and areas of focal emphysema are present.

In the late form of worker's pneumoconiosis (progressive massive fibrosis), large areas of fibrous tissue, black with dust, are present.

The early forms are often asymptomatic unless the worker has an underlying disease (COPD). In the later forms, the victim presents with dyspnea possibly leading to respiratory failure. The pulmonary function studies in the patient with the progressive form of the disease show a mixture of obstructive and restrictive disease.

The chest x-ray shows fine nodulation and the stages of the disease may be determined by the density of the shadows.

The treatment of worker's pneumoconiosis consists solely of preventative and supportive measures for ensuing respiratory failure.

Sarcoidosis:

Sarcoidosis is an ill-defined granulomatous disease affecting multiple body systems. Etiology is unknown. It is found throughout the world, predominately in women, with a high incidence in the southeastern United States. This is an area with a high black population, yet Sweden has almost the same rate of incidence.

Pathology

A sarcoid lesion (3 to 4 cm in diameter) is a noncaseating granuloma. It is made up of epitheloid cells, giant cells and lymphocytes. This lesion is found primarily in the lungs and on the lymph nodes. It will either resolve uneventfully or cause hyaline fibrosis and structural change in the target organ. In the lung, thickening of the alveolar membrane and fibrotic changes may occur.

Clinical Findings

Sarcoidosis is difficult to picture clinically. The patient with sarcoidosis may present with dyspnea, irritating cough, general malaise and weight loss. Sarcoidosis can be determined with the chest x-ray.

Roentgenographic Findings

The chest x-ray may show hilar lymph node enlargements with or without pulmonary involvement. When a pulmonary abnormality is seen, it usually has a bilateral, diffuse, "ground glass" appearance and is distributed from the upper lung fields to the bases. As the disease progresses, pulmonary fibrosis and scaring will present.

Laboratory Findings

A specific skin test for sarcoidosis, the Kviem tet, can be performed. It takes four to six weeks to determine the results. Other tests that can be performed are the Mantoux test and tissue biopsy.

Pulmonary function testing will demonstrate a restrictive pattern, normal flow rates and a decreased DLCO. Arterial blood gases may be normal. If abnormal, there may be a slight hypoxemia.

Treatment

The treatment of sarcoidosis is limited to treating the acute symptoms. In this case, an analgesic may be used. Occasionally, steroid therapy can be helpful.

Thoracoskeletal Diseases:

Diseases of the chest wall can also cause restrictive lung disease. These conditions can be congenital as a result of tuberculosis or neuromuscular disease. A backward curvature of the spine is termed kyphosis. A lateral curvature of the spine is called scoliosis. Kyphoscoliosis is both a lateral and backward curvature of the spine.

Any disease of the chest wall which impedes the chest expansion is termed restrictive. This is because of a moderate to severe reduction of VC and TLC in these patients.

Neuromuscular Disorders:

Any neuromuscular disorder that affects the respiratory muscles or their nerve supply is classified as a restrictive disease. Diseases falling into this category include poliomyelitis, Guillaine-Barre syndrome, myasthemia gravis and muscular dystrophy.

These diseases can cause paralysis of the accessory muscles of ventilation and the diaphragm, causing a severe reduction of VC, TLC and FEV.

Neuromuscular Diseases Affecting Respiration

The mechanisms which respiration is affected by during neuromuscular diseases are variables that include reduced muscular force for adequate ventilation, disturbance of certain reflexes important for protection of the lung from aspiration of food, gastric content, and upper airway secretions. Mechanisms are also affected by impairment of effective cough for clearing of air passages, diminished or absent periodic, voluntary or involuntary, deep-breathing maneuvers, lack of mobility with its inherent problems, and dysfunction of the respiratory center. Frequently, more than one of these mechanisms is involved.

In this section of the lesson, we shall briefly discuss some of the important conditions affecting these functions through the involvement of the muscles, neuromuscular junctions, peripheral nerves, and spinal cord.

Diseases of the Muscles

The major primary muscular diseases that may involve respiratory function are muscular dystrophies and inflammatory myopathies.

Muscular Dystrophies

Muscular dystrophies are a group of hereditary conditions characterized by a progressive degeneration of the striated muscles, resulting in increasingly severe weakness. They have been classified according to certain clinical and genetic features. The most common form is *Duchenne dystrophy*, which is inherited as X-linked, recessive trait, and therefore, is essentially a disease of males. Other forms, which have autosomal type of inheritance, are seen in both sexes.

The onset of muscular weakness, which is the only presenting symptom in most cases, is quite variable. In Duchenne dystrophy, the weakness starts early in life in the proximal muscles of the extremities. Once the child starts to walk, certain abnormalities can be detected, which become more evident as he grows older.
Movements such as getting up from a sitting position or climbing stairs, which require proximal muscular strength, become more and more difficult. In early adolescence, the victim is usually unable to walk. In other forms of muscular dystrophy, the onset is later, and they are usually designated according to the group of muscles that are primarily involved.

In *myotonic* muscular dystrophy, in addition to progressive muscular weakness, there are certain distinctive features. Difficulty of relaxing the contracted muscles such as a hand grip, known as myotonia, is quite characteristic. Early development of cataract, testicular atrophy, and frontal baldness are other associated features.

Pulmonary complications are much more common in myotonic dystrophy than in other forms. In addition to the difficulties related to respiratory muscle involvement, there are frequent problems with swallowing and aspiration. Pulmonary function studies in most cases of muscular dystrophy demonstrate some abnormalities. Reduced vital capacity, maximum voluntary ventilation, and maximum expiratory and inspiratory forces are quite common. The severity of these abnormalities depends on the degree of respiratory muscle involvement.

Pulmonary infection is a common cause of death in these unfortunate individuals. Respiratory insufficiency is frequently aggravated by an intercurrent infection, which may result in fatal respiratory failure. In overall management of these patients, the importance of proper respiratory care cannot be overemphasized.

Disorders of the Neuromuscular Junction

The junction of the motor nerve endings with the striated muscle (muscle end plate) is the area through which the nerve impulses are transmitted to the muscle.

This transmission is accomplished by liberation of *acetylcholine* from the nerve ending and its reaction with the special receptor at the muscle cell membrane.
This interaction results in increased permeability of this membrane to such cations as sodium, potassium, and calcium. The crossing of these ions through the membrane results in depolarization of muscle and initiation of its action potential and contraction.

An enzyme, called *acetylcholinesterase*, inactivates acetylcholine by hydrolysis; thus the muscle is repolarized and becomes ready for reception of another nerve impulse and initiation of another contraction. The proper function of this junctional region is, therefore, essential for orderly muscle activity.

Certain agents are known to disrupt the normal function of the neuromuscular junction. Drugs that interfere with the action of enzyme acetylcholinesterase (e.g. neostigmine) result in accumulation of acetylcholine in this region, thus facilitating the transmission of impulses through the myonerual junction; but large doses of these drugs will result in muscle weakness. On the other hand, neuromuscular blocking agents paralyze the muscles by blocking the access of acetylcholine to the motor end plate. Tubocurarine and other curare-form drugs, such as Pavulon, act through this mechanism. Succinylocholine (Anectine), another type of paralyzing agent, causes depolarizing of muscles as acetylcholine, but is inactivated much more slowly. Repolarization, which is essential for transmission of impulses from the nerve endings, is there- fore prevented. These paralyzing agents are used as an adjunct in general anesthesia and for facilitation of management of patients undergoing intubation and mechanical ventilation.
In *botulism* (a form of food poisoning from absorption of a toxin produced by a bacterium, *Clostridium botulinum*) the paralysis is due to the effect of the toxin on the nerve endings, preventing them from releasing acetylcholine. Rapid ventilatory failure due to respiratory muscle paralysis is the usual cause of death in botulism.

A certain group of antibiotics, such as kanamycin, gentamycin, neomycin, and streptomycin, may result in neruomuscular blockade by interference with the release of acetylcholine.

Myasthenia Gravis

Myasthenia gravis is a disease of the neuromuscular junction manifested by a muscular weakness and fatigability. The exact cause and pathogenesis have not been clearly understood, however, immunology seems to play an important role in this disease.
Recent studies suggest the production of autoantibodies against the acetylcholine receptors in the muscle-cell membrane.
The relationship of the thymus gland and myasthenia gravis has long been demonstrated; 70 percent of the patients have hyperplasia of this gland and another 10 percent have thymoma (neoplasm of the thymus).

Most frequently involved are the muscles of the face, eyes, pharynx, and larynx. However, every skeletal muscle may be affected. Involvement of the respiratory muscles may result in abrupt development of ventilatory failure. This grave complication is the most common cause of death from this disease.

Myasthenia gravis occurs at all ages; females are affected more often than males. The highest incidence is during the third decade of life.

Clinical Manifestations

The onset of myasthenia gravis is usually slow and insidious, but occasionally it may be abrupt. Weakness of the eye muscles, which is the most common manifestation, may result in drooping of the eyelids and double vision. Characteristic facial appearance results from the involvement of the facial muscles. Abnormal speech may be due to weakness of facial, tongue, or laryngeal muscles. These symptoms are more apparent at the end of the day or following repetitive movements of the involved muscles, and they improve with rest. Difficulty with chewing, swallowing, and choking upon eating, causes problems with nutrition. Excessive fatigability of muscles of the trunk and extremities can be demonstrated with exercise. Sometimes the weakness may be extreme, and the patient may seem to be totally paralyzed.

Myasthenia crisis refers to the rapid development of weakness to the extent of impairment of respiration. It is usually provoked by infections, especially those involving the respiratory tract. Emotional upset, surgery, discontinuation of medications, or the intake of certain drugs known to increase neuromuscular blockade are other causes of myasthenia crisis. A similar picture may develop in patients who have taken an excessive amount of anticholinesterase drugs.

The course of myasthenia gravis is usually unpredictable; it may progress rapidly or slowly, remain unchanged, or remit spontaneously. Certain factors, such as infection, general fatigue, lack of sleep, menstrual period, or other causes of physical or mental stress, may aggravate its course. Respiratory complications as a result of impairment of respiratory muscle function, difficulty with clearing the secretions, aspiration, and frequent respiratory-tract infections are continuous threats to these patients.

Diagnosis of myasthenia gravis is strongly suspected by the characteristic history and usually made by demonstration of muscular weakness and fatigue upon repetitive or sustained contraction of certain muscles, particularly the eye muscles. Regaining of strength after a period of rest further supports the diagnosis. With the administration of certain anticholinesterase drugs such as neostigmine or , preferably, edrophonium chloride (Tensilon), regaining of strength can be demonstrated in dramatic fashion. This test is also useful in differentiating the weakness of myasthenia from that of excessive anticholinestrase therapy.

The characteristic muscle fatigability can also be demonstrated by electric stimulation of muscles and recording their response (electromyography).

Management

The treatment of patients with myasthenia gravis has undergone significant changes in recent years; however the principles of management remain essentially the same. These include proper and adequate treatment of acute episodes of severe muscle weakness, including myasthenia crisis, and measures directed to alter the basic pathophysiologic process and prevent the recurrence of symptoms.

Initially, almost every patient with myasthenia gravis is hospitalized for further studies, observation of the course of the disease, and evaluation of the response to treatment. More severely involved patients are usually put in the intensive care unit. Diligent *respiratory care* is the most important part of the management of these patients during the acute phase of their illness.

Unpredictability of the progress of the disease requires frequent and regular monitoring of the patient's respiratory function, such as measuring inspiratory and expiratory pressures. They should be closely watched for problems such as difficulty with swallowing, aspiration, and clearing the airways. Infections should be detected early and treated properly. Tracheostomy is usually preferred to endotracheal intubation for this purpose. An occasional patient may need prolonged ventilatory support.

The main pharmacologic agents in treatment of acute attacks are anticholinesterase drugs, especially pyridostigmine (Mestinon) or sometimes meostimine, which result in significant improvement in most cases. Difficulty with arriving at a proper maintenance dosage, variability in response, and occasional development of refractoriness make these agents less than ideal for continuous long-term therapy. However, less severe cases can be managed safely with these agents. Mild forms may relapse. Corticosteroids, particularly predniosne, given in large single dose every other day, have been demonstrated to result in remission of cases that respond poorly to other forms of therapy.

Removal of the thymus gland (thymectomy) has been performed regularly in many centers as the treatment of choice. Although the result of surgery of the thymona is less than satisfactory, the majority of the patients with thymic hyperplasia show long-term benefits from thymectomy. In recent years, transcervical approach for removal of the thymus gland has been adopted by many surgeons. This method has obviated the need for thoracotomy, which has significant operative and postoperative morbidity and mortality in patients with myasthenia gravis.

Diseases of Peripheral Motor Nerves: Guillain – Barre' Syndrome

Peripheral nerves maybe affected by various toxic agents, metabolic disorders, inflammatory states, vascular disease, trauma and other unknown sources. Despite frequency of peripheral nerve disease in clinical practice, involvement of the respiratory motor nerves is very uncommon or insignificant. However, a form of acute polyneuritis, known as Guillian-Barre' Syndrome, frequently involves the respiratory muscles, resulting in ventilatory difficulties. The cause of death in this disease, almost often is due to respiratory complications.

Guillian-Barre' syndrome is a relatively common condition, which has its highest incidence in the young and middle-aged. There is frequently a history of preceding upper respiratory tract infection, although the etiology remains unknown. Its association with certain viral diseases, including infectious mononucleosis, has been demonstrated in some cases. Pathologically, there is segmentation of the myelin sheath of the peripheral nerves and mononuclear cell infiltration. Delayed hypersensitivity against the myelin sheath has been implicated in its pathogenesis.

Clinical Manifestations

Typically, the onset is rapid with progressive, more or less symmetrical, weakness, starting in the legs and spreading upward to affect the trunk, arms, and face. It may, however, start in the face or upper extremities. Respiratory muscles are involved in more severe cases. The paralyzed muscles are flaccid. Sensory changes may also be present. After the establishment of maximum weakness, which is quite variable in individual cases, spontaneous recovery begins. This is usually expected with 3- 4 weeks. Delayed onset of remission often results in incomplete recovery. A certain form of the disease may have a chronic relapsing course.

Involvement of the muscles of the pharynx and larynx may result in swallowing difficulty and aspiration. Weakness of the abdominal and chest muscles impairs the cough mechanism, and, thus, airway clearance, predisposing the patient to respiratory infection and atelectasis. Other respiratory muscles, including the diaphragm, may be affected. Vetilatory failure is expected under these circumstances.

Management

The management of patients with Guillain- Barre' syndrome is primarily *respiratory*. These patients should be hospitalized, preferably in an intensive- care unit, and proper respiratory care given.

This includes regular monitoring of the respiratory function, with frequent measurement of vital capacity and maximum inspiratory and expiratory pressures, careful broncho-pulmonary toilet, and ventilatory assistance.

These patient's recoveries will depend on adequate maintenance of their respiratory status; therefore, the importance of respiratory care in their management cannot be overstressed.

When there is evidence of respiratory difficulty, as judged by significant reduction in vital capacity and respiratory forces, and other signs of ventilatory failure; mechanical ventilatory support should be instituted. Tracheostomy is the preferred mode of intubation for this purpose. As the vast majority of patients will eventually recover despite marked impairment of their muscle function, every effort should be made to support their lives until remission takes place. The respiratory therapists and nurses play crucial roles in this rewarding endeavor. An occasional patient may require prolonged mechanical ventilation before any sign of improvement can be demonstrated. Definite beneficial result from the use of corticosteroids in this disease has not been unequivocally demonstrated. The most important and essential measure is still supportive therapy, particularly maintenance of adequate respiration until remission occurs.

Disorders of the Spinal Chord

Acute anterior poliomyelitis, commonly known as *polio*, used to be the most important cause of ventilatory failure of neruomuscular origin. Fortunately, it is now almost totally eradicated, and its importance has become historical. A catastrophic epidemic of poliomyelitis, which occurred in 1952 in Copenhagen, was an important impetus in the movement of mechanical ventilators. The ventilators continued to improve, while poliomyelitis continued to vanish.

Many other diseases of the spinal cord may occasionally result in respiratory difficulty. Diseases such as amyotrophic lateral sclerosis, multiple sclerosis, and various forms of inflammatory or neoplastic diseases of the spinal cord may result in respiratory muscle weakness and ventilatory failure.

As the origin of the phrenic nerves is from the high cervical cord, diseases that involve only the lower regions spare this important inspiratory muscle, and adequate ventilation is maintained. However, significant weakness of other respiratory muscles, especially abdominals, may result in impairment of effective cough, and, thus, cause respiratory problems.

Traumatic injury to the cervical spinal cord below the fourth cervical vertebra results in *quadriplegia* with maintenance of respiration by the unaffected diaphragm. Injury above this level, however, will result in complete respiratory paralysis. Even with intact diaphragmatic function, quadriplegics are predisposed to respiratory difficulties from frequent bouts of pneumonia and atelectasis as a result of impairment of cough and clearing the airways, as well as a lack of mobility. In addition, these patients are prone to develop thrombophlebitis and repeated pulmonary embolism. Most early deaths following acute traumatic quadriplegia are due to pulmonary complications; therefore, the importance of adequate respiratory care in management of such patients should be stressed. Continuous respiratory care should be included in overall chronic management and rehabilitation of these unfortunate patients.

Pickwickian Syndrome:

The Pickwickian syndrome is a term applied to excessive obesity which restricts the movement of the chest wall. Tissue mass alone causes the patient to hypoventilate.

Pickwickian patients present with cyanosis, polycythemia, hypoxemia, hypercapnia, pulmonary hypertension and often cor pulmonale. These patients may complain of somnolent episodes and nocturnal apnea.

Treatment of the Pickwickian patient involves ventilatory support and weight loss. This can be achieved with proper diet, counseling and much work on the part of the patient.

Pneumothorax:

A pneumothorax is caused by air entering the pleural space causing the lung to partially or totally collapse. The degree of collapse is quantitatively assessed as a percentage (e.g., 30% pneumothorax, 50% pneumothorax). Air can enter the pleural space either through the chest wall, such as with a penetrating injury, or from the lung as occurs when a bleb or bullae ruptures.

A spontaneous pneumothorax occurs when there is a rupture of visceral pleura without evidence of pulmonary disease. An open pneumothorax indicates that a free flow of air exists in and out of the pleural space with each ventilation. A closed pneumothorax is one in which there is no air movement. A tension pneumothorax is described as one in which air enters the pleural space during inspiration and cannot escape during exhalation.

Etiology

The most common type of pneumothorax is the spontaneous pneumothorax. It is usually cased by rupture of a bleb, bullae or a weakened area in the lung periphery. Disease in which these pathologies are common are chronic obstructive lung diseases, status ticus and end-state sarcoidosis.

Spontaneous pneumothorax is often seen in young males between twenty and forty, particularly those who are tall and thin. This is thought to be due to an increased stress to the upper lung fields.

Spontaneous pneumothoraces usually occur while the patient is in a resting state, rather than with the strain and increased physical effort as would be expected.

A pneumothrorax is also caused when there is penetration through the chest wall and the visceral pleura. This type of pneumothorax can be caused by rib fractures, stab wounds, gun-shot wounds, thoracotomy procedures, thoracentesis, transthroracic needle biopsy or similar events.

Pathogenesis

Normally, the intrathoracic pressure (within the pleural space) is negative and less than the intrapulmonary pressure (within the airways). When a pneumothorax occurs, this pressure gradient slowly or rapidly decreases. If collapsed lung tissue does not block the source of the pneumothorax, the pressure gradient can reach zero. If a tension pneumothorax occurs and air enters the space on inspiration through a "valve-like" opening but cannot escape on expiration, the pressure within the pnuemothorax can increase enough to compress the opposite lung by a mediastinal shift.

Clinical Findings

The major clinical features of pnuemothorax are dyspnea, chest pain on the affected side, a decrease in chest expansion, distant or absent breath sounds and hyper-resonance. In addition, there may be subcutaneous emphysema over the neck and chest; in the tension pnuemothorax, hyperdistention of the thorax and cyanosis may occur. If any or all of these signs are present and a pnuemothorax is suspected, a chest x-ray should be taken immediately to assess the presence or absence of a pneumothorax. The patient with COPD may normally exhibit these signs, especially in the presence of a respiratory infection. If the pnuemothorax is small, it can be difficult to clinically diagnose. For this reason, the chest x-ray is an important tool in the diagnosis of a pneumothorax.

Roentgenographic Findings

The chest x-ray confirms the diagnosis of pneumothorax, regardless of its cause. A P-A film will show the area of collapsed lung accompanied by a peripheral area of translucency bordered by the visceral pleural line. The translucent area is caused by the air in the pleural space.

If the pneumothorax is large, the trachea and mediastinum will shift away from the affected side. The hemidiaphragm on the affected side may be depressed.
A small pneumothorax can be more difficult to identify due to the presence of skeletal shadows, which can obliterate the line of the visceral pleura.

Laboratory Findings

Possible symptoms of the pneumothorax that can be determined by arterial blood gases are hypoxemia and hypocapnia. Hypoxemia may occur from the interruption of gas exchange, which could cause the patient to hyperventilate with a resultant hypocapnia. A pulmonary function test will show a reduction of FVC and FC.

Treatment

The treatment of a pnuemothorax is to re-expand the lung and prevent further pnuemothoraces. A small, spontaneous pnuemothorax (less than 20%) can resolve itself. With sufficient rest, the body can fully reabsorb the air in the pleural cavity. Serial chest x-rays must be performed to monitor progress and spot a recurrent pneumothorax.

A tension pneumothorax is considered a medical emergency and must be treated immediately with the introduction of a large-bore needle into the pleural space. If positive pressure exists in the pleural space, a rush of air will exit upon insertion of the needle. The needle should be replaced with a chest tube connected to underwater seal drainage.

A chest tube permits air to leave the pleural space and prevents additional air from entering. In this way, pleural pressures can return to normal and the lung is allowed to re-expand. When the lung is fully expanded, the suction is discontinued and the tube is clamped. This allows assessment of the pleural space for normal function prior to removal of the tube. When the lung is capable of remaining re-expanded without the use of a chest tube, the tube is removed and the incision covered tightly with a bandage and allowed healing.

Any pneumothorax greater than 20% should be treated with the use of a chest tube for re-expansion. This is especially true in the pneumothorax that causes dyspnea. The tube should remain in place at least 24 hours and then until re-expansion is complete.

Pleural Effusion:

A pleural effusion is the presence of fluid rather than air in the pleural space. This is a common occurrence usually seen in conjunction with another disease entity.

Etiology and Pathology

A pleural effusion is caused by fluid passing across the pleural membrane due to the hydrostatic and osmotic pressure grandients across the membrane. Examples of pleural effusion are empyema (pus in the pleural space), hemothorax (blood in the pleural space) and chylothorax (lymph in the pleural space).

The nature of the pleural fluid can be determined through aspiration and laboratory analysis. The types of fluid can be divided into transudates (having few cellular elements) and exudates (having more cellular elements), according to the protein content. A transudative effusion can be formed with congestive heart failure, cirrhosis and the nephrotic syndrome. A transudative effusion is a localized tendency for edema fluid to form.

An exudative effusion can result if the pleural membranes are irritated. This type of effusion can occur with lung infections, pneumonia, lung abscess, fungal diseases, tuberculosis and malignancies.

Empyema is commonly due to pneumonia or lung abscess. It is defined as purulent fluid in the pleural cavity. Hemothorax usually is caused through chest trauma, and subsequent hemorrhaging into the pleural space.

A chylothorax is due to a leakage of chyle (lymph) into the pleural space. This can be due to blockage of the thoracic duct or trauma.

Clinical Findings

If there is a large effusion, the patient will complain of dyspnea and pleuritic pain.
There will be dullness to percussion over the effusion, absent breath sounds and a decreased movement of the chest wall. The symptoms of a pleural effusion will vary with the extent of the effusion.

Roentgenographic Findings

The upright chest x-ray can be used to diagnose a pleural effusion. It will appear as a shadow which fills the costophrenic angle. If the effusion is large, a triangular shadow will appear, lying against the lateral chest wall.

Laboratory Findings

Once the presence and location of a pleural effusion is detected by chest x-ray, a diagnostic thoracentesis can be performed. The pleural fluid is then analyzed for type of fluid and its origin is determined. If the cause of the effusion is known, such as in congestive heart failure, this invasive procedure may be omitted.

If the effusion is large enough to cause dyspnea, hypoxemia and hypocapnia can be present. A pulmonary function test would show a reduction of FVC and VC.

Treatment

The treatment for a pleural effusion should deal with determining the cause of the effusion and treating the underlying disease. Occasionally, if the pleural effusion impairs lung function enough to cause dyspnea, a thoracentesis can be performed to evacuate the fluid from the pleural space.

Adult (Acute) Respiratory Distress Syndrome (A.R.D.S.):

Adult Respiratory Distress Syndrome is a term which refers to a collection of clinical, physiologic and pathologic features occurring in the setting of an acute injury or illness. The initial lung injury damages the pulmonary capillary endothelium, stimulating platelet aggregation and intravascular thrombus formation. Platelets release substances that attract and activate neutrophils. Other chemotactic factors include endotoxin, present in sepsis, a common cause of A.R.D.S., tumor necrosis factor and interleukin-I (IL-1). Endothelial damage also initiates the complement cascade simulating neutrophil activity and the inflammatory response.

The role of neutrophils is central to the development of A.R.D.S. Activated nutrophils release a battery of inflammatory mediators, including proteolytic enzymes, toxic oxygen, arachidonic acid metabolites (prostaglandins, thromboxanes, leukotrienes), and platelet-activating factor. These mediators extensively damage the alveolcapillary membrane and greatly increase capillary membrane permeability. This allows fluids, proteins, and blood cells to leak from the capillary bed into the pulmonary interstitium and alveoli. The resulting pulmonary edema and hemorrhage severely reduces lung compliance and impairs alveolar ventilation. Mediators released by neutrophils and macrophages also cause pulmonary vasoconstricion. Pulmonary hypertension results, and because vasoconstriction occurs to varying degrees in the vascular beds, V/Q mismatching occurs.

This syndrome has been described as a distinct form of diffuse pulmonary injury of various causes, characterized by rapidly progressive dyspnea, tachypnea, refractory hypoxemia, diffuse pulmonary infiltration, and reduced lung volumes and compliance. This generalized inflammatory response results from the soft tissue damage followed by an episode of hypotension. The sympathetic response to systemic hypotension is pulmonary vasoconstriction. Subsequent vigorous fluid resuscitation is required to maintain systemic permeability (noncardiogenic pulmonary edema), a decrease in pulmonary compliance and a widening of the Alveolar-arterial gradient.

On average, the amount of extravascular water in the lungs of patients with A.R.D.S. is about three times the upper limit of normal (which is approximately 500 ml.) but may be as much as six to eight times the upper limit.

Pathologic changes are a collection of atelectasis, interstitial and alveolar edema, and hemorrhage, and sometimes, hyaline membrane formation. Regardless of how it started, the ensuing events result in more or less similar pathologic and physiologic alteration of the lungs. Both endothelium and epithelium are damaged resulting in leaky alveolar capillary membrane. As part of the host defensive response and reparative effort, local and systemic inflammation develops. The inflammatory reaction itself causes further lung injury.

ACUTE LUNG INJURY AND/OR A.R.D.S:

IF WE ARE TO INCREASE OUR SURVIVAL RATE IN THIS PATIENT POPULATION, EARLY INTERVENTION IS IMPERATIVE.

In conditions that result in lung injury directly, inflammation is secondary, whereas in sepsis the lung injury is the consequence of inflammation. Sepsis, which is the most common cause of ARDS, is defined as the systemic inflammatory response to infection that may be local or systemic (septicemia). Among the infectious agents, gram-negative bacteria are notorious for inciting severe systemic reaction. Endotoxin produced by these microorganisms is a major reason for their characteristic pathogenic features. In addition to infection, other clinical conditions may also result in systemic inflammatory response syndrome (SIRS).

They include multiple trauma, extensive burn, acute pancreatitis, and shock from noninfectious causes. When severe enough, SIRS affects the function of several organs including the lungs (multiple-organ dysfunction syndrome) and may even cause their failure (multiple-organ system failure). Lung injury and A.R.D.S. almost always represent the predominant feature of these events.

According to the European- American Consensus Conference on A.R.D.S. (1994) the following working definitions were developed. Since both conditions involve impaired oxygenation, defined as a ratio of the partial pressure of arterial oxygen to the fraction of inspired oxygen, and since RESPIRATORY THERAPY IS A SCIENCE, the following components are useful in establishing an operational definition:

Bilateral infiltrates on CXR
Pulmonary capillary wedge pressure less or equal to 18 mm.Hg.
And calculation of the PAO2/ FIO2 ratio

If a patient has a high pulse rate, he or she is ANXIOUS. One of the classic signs of refractory hypoxemia is tachypnea and TACHYCARDIA accompanied by a clear chest film. If you encounter a trauma patient who has a rapid pulse:

1. COMPUTE A-a DO2
2. If the patient is on a ventilator, compute the patient's compliance

If the patient has a PaO2/FIO2 of <300
Treat the patient for Acute Lung Injury
If the patient has a PaO2/FIO2 of <200
Treat the patient for A.R.D.S.

Although the exact mechanism is unclear (toxic molecules including oxygen radicals/ proteases), A.R.D.S. is a complex inflammatory response, there is evidence that overall survival has improved in recent years due, in part, to early intervention.

Whether as a primary cause of or contributing factor in A.L.I, inflammation has been regarded as the major pathogenic mechanism in ARDS. Despite extensive animal and human research in recent years, the exact mechanisms of lung injury by various inflammatory cells and mediators are not fully understood. Although many inflammatory cells and mediators of lung injury have been recognized, their exact roles, the sequence of their involvement, and the degree of their importance have remained speculative.

Among the cells involved, polymorphonuclear neutophils (P.M.N.'s) play the central role in inflammation. They are sequestered in pulmonary microvasculature; become activated; and produce various proteolytic enzymes, toxic oxygen metabolites, and other inflammatory mediators.

Other cells that participate in the process are macrophages, lymphocytes, and fibroblasts. They interact with the help of cytokines and produce various mediators of inflammation. Complement activation, involvement of coagulation factors, and participation of certain vasoactive substances (including nitric oxide), have also been considered to be important components of the pathogenic process in A.L.I. and A.R.D.S.

A.L.I resulting from the initial event and sustained by subsequent inflammation is the basic mechanism of structural change and a functional impairment characteristic of A.R.D.S. In addition to accumulation of fluid and inflammatory cells in the lungs, other changes also take place. As a result of injury to alveolar cells and presence of edema fluid in the alveoli, surfactant is markedly reduced and becomes qualitatively abnormal. Some of the small vessels are obliterated from fibrin-platelet aggregation. Certain growth factors released by inflammatory cells results in early proliferation of fibroblast and beginning fibrosis tissue formation.

Although the underlying etiologic factors are quite varied, pathologic changes are almost similar. Diffuse alveolar damage with resultant changes secondary to alveolar-capillary leak and inflammatory reaction are the bases of morphologic abnormalities as seen in the lungs of patients with A.R.D.S.

Early acute changes include destructive lesions and loss of endothelial and Type 1 alveolar epithelial cells, edema, hemorrhage, infiltration with inflammatory cells, microatelectasis, and hyaline membrane formation. These changes are not homogeneous; whereas some alveoli are severely damaged or filled with proteinaceous fluid and cells, others may be more or less intact. Pathologic lesions have a predilection for chronic changes of the organizing stage, in which Type II alveolar cells proliferate. Increased numbers of fibroblasts denote the beginning of fibrosis that may develop rapidly. At this stage, there are also changes in pulmonary microvasculature characterized by disruption of the vascular bed and thrombotic or embolic occulsion of some of the small vessels.

Bronchoalveolar lavage (B.A.L.) fluid recovered from patients with A.R.D.S. contains large numbers of P.M.N.s. B.A.L. fluid surfactant is deficient in its surface- tension lowering activity as a result of its chemical composition.

Severe hypoxemia, an essential component of A.R.D.S., is the result of marked maldistribution of ventilation and perfusion and their mismatching. Intrapulmonary shunting from perfusion of unventilated lung regions is the hallmark of pathophysiologic change of the syndrome. Alveolar and interstitial edema, inflammatory changes, reduced and ineffective surfactant, and early fibrotic changes are known causes of reduced lung compliance. Considering the heterogeneity of lung injury, however, decreased compliance may be in large part from reduced volume of ventilating lung units.
Such units, per se, may have normal compliance. This notion is important for understanding the deleterious effect of mechanical ventilation with large tidal volumes in A.R.D.S.

Because of vascular disruption and obstruction of some of the small vessels, certain ventilated lung units may have reduced or no perfusion resulting in increased dead-space ventilation. High minute ventilation, together with reduced lung compliance markedly increases the work of breathing. Tachypnea is the result of increased minute ventilation and low tidal volumes. Pulmonary hypertension, a common occurrence in A.R.D.S., is secondary to vascular obstruction and increased vascular tone from imbalance of vasoactive susbstances and hypoxemia.

The initial assessment of arterial blood gas analysis will usually reveal respiratory alkalosis and varying degrees of hypoxemia. The hypoxemia is often relatively resistant to supplemental oxygen (refractory hypoxemia). As alveolar fluid accumulates, hypoxemia continues to worsen and mechanical ventilatory support becomes necessary. After a latent period of 24 or more hours, fluffy alveolar infiltrates develop and are visible on the chest roentgenogram, and arterial blood gas analyses show progressive hypoxemia due to a shunt-like effect. Pulmonary compliance progressively falls. According to the European-American Consensus Conference on A.R.D.S. (1994) the following working definitions were developed.

Since both conditions involve impaired oxygenation, defined as a ratio of the partial pressure of arterial oxygen to the fraction of inspired oxygen. The following components are useful in establishing an operational definition: (a) Bilateral infiltrates on frontal CXR, (b) Pulmonary capillary wedge pressure less or equal to 18 mm.Hg. and (c) calculation of the PAO2/ FIO2 ratio. If a patient has a high pulse rate, or he or she is anxious, refractory hypoxemia should be considered. One of the classic signs of refractory hypoxemia is tachypnea and tachycardia accompanied by a clear chest film. If one encounters a trauma patient who has a rapid pulse, compute the patient's A-a DO2. If the patient is on a ventilator, compute the patient's pulmonary compliance. If the patient has a Pa02/FIO2 of <300, treat the patient for Acute Lung Injury. If the patient has a PaO2/FIO2 of <200, treat the patient for A.R.D.S although the exact mechanism is unclear (toxic molecules including oxygen radicals/ proteases), A.R.D.S. is a complex inflammatory response,
which requires early assessment and intervention. There is evidence that overall survival has improved in recent years due, in part, to both early intervention and technological advances.

The pancreas is unique in that it has both endocrine and exocrine functions. The endocrine pancreas secretes hormones: insulin, glucagon, somatostatin, and pancreatic polypeptide. Arterial blood is supplied to the pancreas by branches of the celiac and superior mesenteric arteries. Venous blood leaves the head of the pancreas through the portal vein, with the body and tail being drained through the splenic vein. All hormonal pancreatic secretions also pass through the portal vein to the liver. Acute pancreatitis is a clinical syndrome resulting from inflammation and destructive autodigestion of the pancreas and peripancreatic tissues. Clinically, acute pancreatitis is a common and important cause of upper abdominal pain, nausea, vomiting, and fever. Laboratory findings of marked elevations of serum amylase and lipase help to differentiate it from other entities causing these symptoms. The severity of inflammation varies, and the prognosis ranges from mild, and self-limiting illness lasting 1 to 2 days to death from pancreatic necrosis, hemorrhage or sepsis. Pleuropulmonar complications of acute pancreatitis include pleural effusion, pulmonary edema, and acute repiratory distress syndrome.

Characteristically, the pleural fluid is an exudate with high protein, LDH, and amylase levels (normal serum amylase = 60-180 Somogyi units/ml. and normal serum lipase = 1.5 Somogyi units/ml.)

ACUTE LUNG INJURY + VOLUME = VOLUTRAUMA

Since its introduction into clinical practice more than 40 years ago, mechanical ventilation has utilized large tidal volumes (10 to 15 ml./Kg.) to prevent the development of progressive atelectasis and hypoxemia that was routinely seen with physiological tidal volumes (5 to 7ml./Kg.). Recent insights into the pathophysiology of acute lung injury has led to changes in routine approaches to mechanical ventilation.

Heterogeneous alterations in the anatomy and function of the lung are characteristic of lung injury.
Experimental evidence strongly suggests that traditional approaches to mechanical ventilation with high airway pressures produce insidious physiologic and morphologic changes in previously normal lungs.

This process, termed VOLUTRAUMA, impedes lung healing and may extend damage to previously unaffected areas. This realization has led to a strategy that is designed to avoid tidal volume collapse using physiologic transalveolar pressures while allowing alveolar hypoventilation and hypercapnia. Debate continues regarding the risks and benefits of pressure-limited versus volume-limited mechanical ventilation to achieve this goal. Research is ongoing regarding the role and techniques for non-conventional methods of cardiorespiratory support for this severely ill group of patients. The alterations which ultimately produce acute lung injury can progress to multiple organ failure.

Mechanical ventilation with high peak inspiratory pressures and large tidal volumes in normal rats produces pulmonary edema associated with severe permeability alterations, increased filtration and diffuse alveolar damage. It has also been demonstrated that lung overdistention, not the elevated airway pressures, is the responsible factor for these findings, because negative-pressure ventilation with excessively large tidal volumes will produce similar injury.

This finding was further confirmed by the demonstration that tidal volume limitation via thoracoabdominal binding during high-pressure ventilation inhibits the development of permeability changes and edema. These experiments led to the evolution of the concept that mechanism of high- pressure ventilation inhibits the development of permeability changes and edema. These experiments led to the evolution of the concept that the mechanism of high –pressure pulmonary edema and lung injury is due to barotrauma, to "VOLUTRAUMA". It appears that end-inspiratory volumes are the primary determinant of volutrauma, and thus measures to decrease pressure- volume swings and high flow rates may not prove sufficient to protect the lungs from ventilator induced injury.

REMEMBER: "OXYGEN IS THE FUEL OF THE BODY"

Roentgenographic Findings

The chest x-ray will show diffuse alveolar infiltrates in a honeycombed pattern in the later stages of ARDS. Early in the onset of ARDS, the chest film can appear normal.

Laboratory Findings

A severe hypoxemia and an eventual hypercapnia (as the patient tires) will be seen. Pulmonary artery wedge pressure will be normal or high FRC will be decreased. Peak airway pressures on the mechanical ventilator will steadily rise as compliance falls.

Treatment

Treatment involves supportive measures with oxygen therapy, fluid management and steroid therapy.

<u>Pulmonary Edema</u>:

Pulmonary edema is a term given to a condition where fluid collects in the extravascular spaces and lung tissues.

Etiology

Pulmonary edema is considered a complication of another disease entity. Etiology of pulmonary edema involves an interruption of the delicate pressure gradients across the alveolar capillary membrane. Included in these causes are myocardial infarction, mitral stenosis, fluid overload, oxygen toxicity, radiation, silicosis, rapid removal of pleural effusion or pneumothorax, over-transfuction, hypo-proteinemia, high altitude and heroin overdose.

Pathophysiology

The pathophysiology varies with each causative agent, but the alveolar-capillary (a-c) membrane is generally disturbed by one of the following:

1. An increase in capillary hydrostatic pressure
2. An increase in capillary permeability
3. Lymphatic insufficiency
4. A decrease in interstitial pressure
5. A decrease in colloid osmotic pressure

Each of these disturbances of the delicate alveolar-capillary membrane can cause fluid to leak out of the capillary into the interstitium or alveoli.

Clinical Findings

Respirations will be shallow and rapid and the patient will complain of dyspnea. Orthopnea is often present along with paroxysmal nocturnal dyspnea and Cheyne-Stokes respirations. A dry cough may present. In acute pulmonary edema, frothy pink sputum may be expectorated or aspirated from the trachea. The chest wall will reveal diffuse fine inspiratory rales. There will be an increase in tactile fremitus.

Roentgenographic Findings

A common sign of pulmonary edema is cardiomegaly and increased vascular markings. Short horizontal lines (Kerley lines) may appear in the lower lung fields. Blotchy shadowing will radiate from the hilar areas in acute pulmonary edema.

Laboratory Findings

Hypoxemia will be present with a normal or low carbon dioxide level. Pulmonary function studies would indicate an increased elastance, airway resistance and a decreased compliance.

Treatment

Pulmonary edema involves treatment of the underlying cause. Oxygenation must be supported. If the underlying disease involves fluid overload, the patient is diuresed. If the underlying problem involves hypo-proteinemia of the blood, albumin is given.

Unit 4. Infectious Pulmonary Disease:

The infectious pulmonary diseases are inflammatory processes brought about within the lung by the introduction of an infectious pathogen. The pathogenic agent can be bacterial, viral or fungal. Included in this discussion are pneumonias, pulmonary fungal infection and pulmonary tuberculosis. Treatment of all these diseases involves identification and control of the invading organism.

Pneumonia:

Pneumonia is defined as an acute inflammation of the lung parenchyma. Pneumonia can be grouped into two major categories according to the etiologic agent: bacterial or non-bacterial (atypical). The non-bacterial pneumonias include mycoplasm pneumoniae, adenovirus pneumonia and psittacosis. Most pneumonias are of the bacterial type, acute in onset and associated with alveolar filling by exudate.

Bacterial Pneumonia

Bacterial pneumonias are classified according to their etiologic agent and the area of involvement on the chest x-ray examination (e.g., bronchopneumonia, interstitial, alveolar or lobar).

Etiology

The primary infecting agents in bacterial pneumonia are Streptococcus pneumoniae (pneumococcus), Staphylococcus pyogenes (Staphylococcus) and Klebsiella pneumonia (Friedlander's bacillus). Less frequently encountered are Streptococcus pyogenes and Hemophilus influenzae.

Incidence

Bacterial pneumonia is usually found in middle-aged to elderly patients. Pneumonia tends to strike debilitated patients or those with pre-existing illnesses. Several predisposing factors to bacterial pneumonia include chronic airway infection, viral infections of the upper respiratory tract, aspiration, or anything that lowers an individual's resistance to infection.

Pathology

The transmission of the bacterial infecting agent is usually by inhalation into the lung. Less commonly, the infection is spread through the blood from an infection elsewhere in the body. An inflammatory reaction occurs, usually in a large bronchus. The infection can spread through a lobe or segment to the lung periphery. Bacterial pneumonias are usually "lobar" or "segmental." If the infection is limited to the bronchus and the airways, it is termed bronchopneumonia.

The inflammatory reaction consists of local vasodilation followed by production of an exudative fluid which fills the surrounding alveoli and small airways.

If an area of the lung becomes filled with fluid and contains no air, it is called a "consolidated" area; if the tissue begins to necrose, a lung abscess may form; if the inflammation reaches the pleura, a pleural effusion may form; and if the infection reaches the bloodstream, septicemia may result. Leukocytes and macrophages appear and remove the bacteria and debris by ingestion. Once the causative agent (bacteria) is removed, the inflammation will eventually cease and the pneumonic process will clear. However, some localized complications may result (e.g., fibrosis, lung abscess and bronchiectasis) due to lung injury.

Clinical Findings

The symptoms of bacterial pneumonia and non-bacterial pneumonia are similar, although a specific infection may present with a certain group of symptoms.

The patient with pneumonia will present with an acute onset of malaise, fever, chills, sweating, shivering, pleuritic chest pain, a productive cough, hemoptysis, headache and dyspnea. The pleuritic pain is often localized to the site of the inflammation.

The patient will be tachycardiac, tachypnic and febrile. The skin will be hot, dry and flushed. Breath sounds may be decreased over the affected area with rales and possibly a pleural rub.
If consolidation occurs, there will be dullness or flatness to percussion, bronchial breath sounds egophany and whispering pectoriloquy. Depending on the degree of inflammation, chest expansion may be decreased.

Roentgenographic Findings

Pneumonia can usually be visualized on the chest x-ray as an abnormality. With the chest x-ray, it can be determined whether the pneumonia involves the alveolar areas, the interstitium, if it is lobar, segmental or bronchopneumonia. If consolidation occurs, the chest x-ray will show opacification.

Laboratory Findings

The white blood cell count will be elevated in the patient with an active bacterial pneumonia, ranging from 15,000 to 30,000 per cubic millimeter (microliter). The patient may exhibit moderate to severe hypoxemia and possible hypercarbia in the presence of an underlying lung disease.

A sputum specimen must be obtained to determine the causative organism. It is paramount that the sputum specimen obtained is from the lower bronchial tree. If a good specimen cannot be expectorated, a transtracheal aspiration will usually provide a sputum specimen. Once the specimen is obtained, the causative organism can be identified and appropriate antibiotic treatment instituted.

Treatment

Antibiotic therapy can be used to control a bacterial pneumonic process and destroy the causative organism. However, some of the symptoms of pneumonia must be treated to aid in the effective removal of the exudate. If the patient is hypoxemic, this should be treated with supplemental oxygen. Pleuritic pain should be treated with analgesics to the extent that cough and ventilatory drive are not suppressed. Dehydration should be prevented and/or treated with fluid replacement.
The patient with bacterial pneumonia requires aggressive respiratory care to promote hydration and expectoration of retained secretions.

Non-Bacterial (Atypical) Pneumonia

A typical pneumonia includes mycoplasmal pneumonia, adenovirus pneumonia and psittacosis. These types of pneumonia differ from bacterial pneumonia in that the onset is not as acute.

Mycoplasmal pneumonia is common in younger people and may be epidemic in schools and the community. Symptoms usually begin with a sore throat and cough and soon the patient presents with headache, general malaise, fever, shivering and lymphadenopathy.

The cough is dry and productive of small amounts of viscid mucus. Dyspnea and chest pain are rare. The chest examination is commonly negative. Chest x-ray shows a unilateral lower lobe bronchopneumonia.

Symptoms and disease courses for mycoplasma, adenovirus and psittacosis pneumonia are quite similar. Adenovirus pneumonia can be fatal in small children and infants. However, adenovirus pneumonia is usually benign in the child and adult.

Psittacosis is acquired by inhalation of droplets from bird droppings, particularly sick birds in the parrot family. It differs from the other non-bacterial pneumonias in that symptoms may be more severe and prolonged.

Pulmonary Fungal Infection:

Inhalation of infectious fungal organisms can cause an illness termed as mycoses that results from direct tissue invasion by the organism. These infections are usually not spread from individual to individual; however, the organism will spread within the lung. These diseases commonly appear in the patient with a chronic debilitating disease.

Diagnosis of the mycoses consists of sputum specimen collections and analysis. Treatment involves administration of the appropriate antifungal agent.

Several of the more common diseases caused by fungal infection are discussed below. Histoplasmosis is caused by a fungus commonly found in chicken, bat, pigeon and starling droppings. It is found predominantly in river valleys and in the United States, it is prevalent in the Mississippi River Valley.

Histoplasmosis presents as an influenza-type bronchopneumonia. The chest x-ray is similar to that seen in tuberculosis with calcification, fibrosis and cavitations. Treatment involves the administration of Amphotericin B.

Coccidiomycosis is caused by inhalation of a fungal agent found in the soil. It is prevalent in the arid areas of Utah, California, New Mexico, Arizona and Texas. It is also know as San Joaquin fever, desert rheumatism, valley fever and the "bumps."
The symptoms of coccidiomycosis are similar to those of atypical pneumonia. Diagnosis can often be made with the coccidioidin skin test. Treatment involves the administration of Amphotericin B.

Blastomycosis is also caused by a fungal agent found in the soil. The agent is inhaled and, if disseminated, can affect not only the lungs, but the skin, bones, sinuses and genitourinary tract. Diagnosis can be confirmed with sputum culture and/or tissue biopsy. Treatment involves Amphotericin B and 2-hydroxystilbamidine.

Aspergillosis, also known as farmer's lung, is caused by inhalation of an airborne fungus often found in hay or compost piles. Infection can cause an tic-type illness or development of VC, cysts and necrosis.

Diagnosis of the tic-type of aspergillosis (allergic bronchial aspergillosis) involves sputum culture and skin testing. Cavitating aspergillosis (aspergilloma) can be diagnosed with sputum culture, chest x-ray and tomography. Aspergillosis may resolve spontaneously or be treated with Natamycin or Amphotericin B.

Cryptococcosis is caused by a fungus found in pigeon and bird droppings. Infection is associated with pleural effusion, lung cavitation and calcification. Treatment involves Amphotericin B, although cryptococcosis often is fatal due to its predilection for the brain and its meninges.

Pulmonary Tuberculosis:

Pulmonary tuberculosis is an infection by Mycobacterium tuberculosis. Once the host is infected, the living organism remains in the lung for years.

Depending on the immunologic status of the host, the tuberculosis will exist in an active, contagious state or an inactive, dormant state.

Incidence

The incidence of tuberculosis has declined sharply since the advent of appropriate chemotherapy and improved hygiene. Tuberculosis is a worldwide disease and affects all people regardless of age, sex or race. There has been an increase recently in new cases of pulmonary tuberculosis in the United States.

This is thought to be due, in part, to the Asian refugees who are arriving in the United States after an exposure period to over-crowded, poorly ventilated quarters and subsequent poor hygiene.

Etiology

Mycobacterium tuberculosis is an obligate, aerobic, gram-positive rod and, therefore, finds the lung to be an ideal, dark, moist and warm home.

Pathology

Mycobacterium tuberculosis is carried as an airborne droplet which is transmitted from the tuberculosis patient as he coughs, talks and breathes. The particle is inhaled, travels to the alveoli and is deposited in the lower lung fields. The bacilli develop slowly over a period and infiltrate into the lymph and blood, thereby spreading to other organs.

In the lung, a lesion appears that contains the bacilli, surrounded by necrotic tissue and leukocytes. This is known as a caseated lesion. A fibrous tissue forms around the tubercle lesion; if long standing, the tubercle becomes calcified. Once the tubercle has encased the bacilli, the infection usually will not spread, but will remain in the lower lung field. This type of tubercle is called a primary focus.

If the patient is reinfected (usually by breakdown of a primary focus), the new lesion will usually occur in an apical segment of the lung. Frequently, more than one lesion will appear and the disease again can spread throughout the body.

Clinical Findings

Tuberculosis is difficult to diagnose clinically due to its insidious onset. It should always be suspected in the elderly, chronically-ill patient with unexplained febrile episodes.

Symptoms include weight loss, persistent cough, hemoptysis, chest pain, malaise, anorexia and dyspnea.
Physical examination of the chest reveals tracheal deviation to the affected side, post-tussive rales at the apices and signs of pleural effusion consolidation and pneumothorax.

Roentgenographic Findings
The chest x-ray may show an area of calcification within a shadow accompanied by fibrosis. This area is usually found in the apices. Cavitation is commonly seen. In miliary tuberculosis, widespread bilateral miliary shadowing is seen.
If soft shadows do not appear in a series of chest x-rays, tuberculosis should be suspected.

Laboratory Findings

A diagnosis of pulmonary tuberculosis can be made by a histologic biopsy of a tubercle, pleural effusion, liver or bone marrow. The patient with tuberculosis is moderately anemic with a normal or low white blood cell count. Commonly, a polymorphonuclear leukocytosis will appear.
A tuberculin skin test can indicate exposure to tuberculosis. A negative test usually will determine the absence of tuberculosis. A positive test indicates the need for further testing, including x-rays and sputum samples.

Treatment

Treatment of tuberculosis involves extensive chemotherapy regimens including the use of Isoniazid (INH), Streptomycin, Ethambutol, Rifampin or Pyrazinamide. Treatment should continue for at least 12 to 18 months; two years of therapy may be necessary in the cavitating state.

Unit 5. Pulmonary Embolic Disease:

When circulation slows, venous blood tends to pool and thrombosis may occur. A thrombus is defined as a blood clot. When a blood clot dislodges and travels through the bloodstream, it is termed an embolus. When an embolus travels to the pulmonary circulation and lodges there, it is termed a pulmonary embolism.

Pulmonary embolism is a common occurrence in the hospital patient who spends much of this time lying inactive. The most common source of venous thrombosis is the lower leg and calf area.

The clinical signs of pulmonary embolism depend upon the size of the embolus. It can be small, medium-sized or massive. A small embolus may pass unrecognized; whereas, a massive embolus can cause shock, pallor, unconsciousness, centralized chest pain, hypotension, neck vein distention, acute dyspnea and tachycardia.

An EKG will indicate right ventricular strain. Thirty percent of the cases involving massive pulmonary emboli prove fatal.

Treatment of pulmonary embolism involves support of the cardiopulmonary system with supplemental oxygen therapy, treatment of hypoxemia and use of anticoagulant therapy to prevent further embolization.

Unit 6. Pulmonary Pathology Due to Trauma:

Lung trauma or injury can result in damage to the lung tissue itself, the pleura and/or chest wall. Forms of trauma that will be discussed here are burns/smoke inhalation, penetrating chest wounds, flail chest/rib fractures, gastric aspiration, near-drowning and hypothermia.

Burns/Smoke Inhalation:

The principle cause of death from burn injury is due to respiratory failure. This can be directly attributed to inhalation of toxic smoke and fumes accompanied by pulmonary injury to the upper airway. Carbon monoxide inhalation causes death in a number of burn victims. Burn victims rarely present with carboxyhemoglobin levels over 15%, as death will usually occur before this level is reached.

The burn victim will commonly present with pulmonary edema in the first 24 hours following initial injury. A diffuse bronchitis can occur. The pulmonary complications of burns/smoke inhalation can be divided into three categories; early, intermediate and late.

Early complications are associated with inhalation of steam, flames and toxic fumes. Damage occurs in both the upper and lower tracheobronchial tree and is thought to be caused primarily by the toxic substances rather than the heat. Inhalation of steam and high temperature gas can cause edema and lead to upper airway obstruction. Inhalation of toxic substances can cause acute tracheobronchitis, airway edema and interstitial edema.

Intermediate complications can develop anywhere between 24 and 72 hours following the initial injury. These include atelectasis caused by mucosal sloughing and pulmonary hemorrhage. A productive cough will reveal flecks of carbon in the sputum.

Late complications of burns/smoke inhalation include pneumonia, sepsis, pulmonary embolism and ARDS.

Pulmonary function studies will show a decreased TLC, increased airway resistance and increased work of breathing. Arterial blood gases initially reveal hypoxemia, hypercapnia and respiratory acidosis. Later, a metabolic acidosis will cause respiratory cessation and hyperventilation.

A fluid loss due to the burns will cause low cardiac output, increased oxygen consumption and tachycardia. Bronchospasm may be caused by the pulmonary injury and auscultation will reveal wheezing. If pulmonary edema is present, rales will be heard. The chest x-ray on admission may be normal, but within 12 to 24 hours abnormalities will appear if lung damage has occurred. Treatment of burns/smoke inhalation injury involves cardiopulmonary support and prevention of pulmonary complications. This may involve an artificial airway, mechanical ventilation oxygen therapy, PEEP, bronchodilation, massive fluid replacement, antibiotic therapy or steroid therapy depending upon the degree of pulmonary complications.

Penetrating Chest Wounds:

A penetrating injury to the chest wall can cause a tension pneumothorax. As discussed earlier in this module, this is a life-threatening injury and must be treated immediately by insertion of a chest tube.

Flail Chest/Rib Fractures:

Flail chest is a condition caused by fracture of the ribs or sternum in two or more places. The result is paradoxical respirations or a flailing of the chest. Upon normal inspiration, the chest wall moves outward and then inward on expiration.
With flail chest, the unstable chest wall will be pulled in on inspiration and pushed out with expiration. If the rib fractures are limited to one side of the chest, the movements of the right and left sides will be paradoxical to each other.

Flail chest can rapidly lead to respiratory insufficiency if not treated immediately. Treatment consists of endotracheal intubation and mechanical ventilation in order to stabilize the chest wall. Frequently, PEEP is required to maintain oxygenation. Mechanical ventilation is continued until the chest wall is sufficiently healed to facilitate spontaneous ventilation.

Rib fractures can also cause a penetrating injury to the chest and a subsequent tension pneumothorax. Again, this must be immediately treated with the insertion of a chest tube.

Gastric Aspiration:

Aspiration of gastric contents into the tracheobronchial tree can cause severe upper respiratory burns and subsequent inflammatory processes. Gastric aspiration can lead to respiratory failure and serious pulmonary complications. Treatment involves vigorous suctioning and saline lavage. The cardiopulmonary system must be supported as adverse symptoms occur. The major treatment of gastric aspiration is prevention with placement of a nasogastric tube for suction in the debilitated patient.

Near-Drowning:

Technically, near-drowning is a form of aspiration; however, 10% of deaths due to drowning are caused by laryngospasm and asphyxiation.
In seawater drownings, a large amount of fluid will be found in the lungs on autopsy. In a freshwater drowning, there
will be minimal volumes of water found in the lung due to absorption of the hypotonic solution.

Hypothermia:

When the body temperature accidentally drops below 35°C, there is a 50% mortality rate. Hypothermia can occur as a result of exposure, Addison's disease, hypoglycemia, pituitary insufficiency, myxedema, stroke, myocardial infarction, near-drowning, pancreatitis and cirrhosis. Hypothermia is more apt to occur in the neonatal or bed-ridden patient.

The effects of hypothermia include bradycardia, decreased cardiac output, decreased oxygen consumption, atelectasis, hypoventilation, increased blood viscosity and increased chance of thrombosis.

The major complications due to hypothermia relate not to the cooling process, but rather to the warming process in the treatment of hypothermia. If rapid warming processes are instituted, oxygen consumption will increase rapidly. Cardiac output will not increase as rapidly and circulatory failure may result. In the presence of pre-existing heart disease, this stress may cause the ventricles to fibrillate and death may result. An increase in carbon dioxide production and V_D/V_T may result with re-warming of the hypothermic patient. Arterial blood gases (temperature corrected) must be monitored closely and ventilation and oxygenation must be supported.

As the patient is rewarmed, he may again become comatose due to an increase in the cerebrospinal fluid pressure.
This may be accompanied by pulmonary edema. Lumbar puncture and removal of a small amount of CSF will reduce the pressure and relieve the symptoms.

The rewarming process should involve insulation of the body to prevent further heat loss and "core" rewarming. Rewarming by external means has proven to be dangerous, especially to the patient with heart disease. "Core" rewarming can be accomplished with a heart-lung machine and cardiopulmonary bypass. Blood is taken from the femoral vein, heparinized, warmed and returned to the aorta. At 34°C, the heart is defibrillated and the heart-lung machine is disconnected.

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PART VI. CLINICAL PROCEDURES

Due to the specific content of this module, no clinical procedures are presented. The student is encouraged to read and review all current literature dealing with changes and development in the field of respiratory pathology.

PART VII. CASE STUDIES

Case No. 1 Asthma in a 28-Year-Old Female:

A 28-year-old female is admitted to the emergency room with severe dyspnea and bronchospasm. Her husband has recently been painting the upstairs bedroom using oil based enamel. She is a known asthamtic and uses albuterol metered dose inhaler at home as necessary to relieve bronchospasm. She is also being maintained on a low dose of Prednisone. She has had to use her inhaler every one to two hours during the night, the previous day and this morning. The treatment gave her minimal relief from bronchospasm.

Clinically, she presents as a thin, white female in acute respiratory distress. Breathing is labored and shallow. Respirations are 28 per minute. Breath sounds are decreased to absent with a faint end-expiratory wheeze over the posterior basal lung fields. The expiratory phase is prolonged. There is hyper-resonance to percussion. The patient is tachycardic, flushed and diaphoretic.

She is immediately placed on oxygen via nasal cannula at 5 lpm after an Oxygen Saturation of 88% on room air was measured via pulse oximetry. Respiratory therapy is called to administer 2.5 mg albuterol via a nebulizer. This therapy is repeated every 30 minutes for three treatments. She is begun on an intravenous aminophylline drip and her Prednisone dose is increased.

The patient is admitted to the medical ward and the intense bronchodilator regimen is continued. She is given Albuterol treatments every four hours via nebulizer for the first 24 hours and then switched to metered dose inhaler. The aminophylline is discontinued.

A week later, she is discharged from the hospital with instructions to gradually taper the Prednisone dose with physician's instruction and use Albuterol inhaler PRN for relief of bronchospasm. She is encouraged to avoid any inhaled irritants

Case No. 2 Pneumonia in a 38-Year-Old Male:

A 38-year-old male is admitted to the emergency room with fever, chills, general malaise and a left-sided pleuritic pain. He has recently suffered a bout of viral influenza and just recently returned to work after a seven-day sick leave.

Physical examination reveals a moderately obese, white male in mild respiratory distress. Skin is hot and dry. Temperature is 101.8°F. The patient is tachycardic and tachypneic with a pulse of 120 and a respiratory rate of 24 per minute. Breath sounds are unequal to auscultation with expiratory wheezes throughout the left, but there is a marked decrease in aeration in the left base. There is an accompanying dullness to percussion.

ABG's on 4 lpm are:

pH	=	7.45
$PaC0_2$	=	35 torr
$Pa0_2$	=	67 torr
$HC0_3$	=	24 mEq/L

A chest x-ray is taken and a left lower lobe pneumonia is diagnosed. A sputum specimen is obtained and cultured. The culture results are positive for Streptococcus pneumoniae. The patient is started on appropriate antibiotic therapy. Respiratory therapy is called to evaluate the patient and begin appropriate therapy.

The patient is started on nebulizer treatments with 2.5mg albuterol adding 400mg mucomyst with every other treatment, appropriate hydration and continued antibiotic therapy. The patient begins to expectorate thick, mucopurulent, green sputum and rales are heard over the left base of the lung.

Treatment is continued and, after one week, the chest x-ray shows a marked improvement in the pneumonic process. The patient is now asymptomatic.

After a 7 day stay in the hospital, the patient is discharged home.

Case No. 3 ARDS following Viral Pneumonitis in a 28- Year-Old Male

A 28- year-old, previously healthy man was admitted to the hospital complaining of high fever and total body aching. Upon examination he was found to have a fever of 102.3, respiratory rate of 32, with elevated WBC of 28,000, HR of 143, and BP of 140/100. On his third hospital day he became increasingly dyspneic, tachypneic, and revealed a refractory hypoxemia to 100% oxygen. His chest x-ray interpretation changed from "resolving interstitial pneumonitis" to a "typical ARDS pattern." Upon transfer to the intensive care unit he was noted to be very short of breath, using all accessory muscles, and unable to speak more than three words at a time. Arterial Blood Gas values were:

pH = 7.46
PaCO2 = 29
Pa O2 = 41

After a careful explanation of the proposed therapy, the patient's nose was appropriately anesthetized and he was intubated by the nasotracheal route. He was placed on a CPAP system with 5 cm H2O CPAP and 60% inspired oxygen; 15 minutes following intubation ABG's were:

pH = 7.44
PaCO2 = 31
PaO2 = 45

CPAP was increased to 10 cm H2O; 5 minutes later:

pH = 7.44

PCO_2 = 30

PaO_2 = 43

CPAP was increased to 15 H2O: 10 minutes later;

pH = 7.42

$PaCO_2$ = 32

PaO_2 = 58

The patient was observed for another 10 minutes, during which vital signs, clinical condition, and blood gases remained unchanged. CPAP was then increased to 20 cm H2O. Ten minutes later:

pH = 7.37

$PaCO_2$ = 35

PaO_2 = 110

The patient's breathing was significantly less labored, and he communicated that he felt very much improved; 15 minutes later on 20 cm H2O CPAP:

pH = 7.35

$PaCO_2$ = 38

PaO_2 = 180

The FIO2 was decreased to .50; 10 minutes later vital signs were unchanged and ABG's were:

pH = 7.36

$PaCO_2$= 38

PaO_2 = 110

The FIO2 was decreased to .40. 20 minutes later vital signs were unchanged with ABG's:

pH = 7.36

$PaCO_2$ = 37

PaO_2 = 73

It should be noted that at this time with proper levels CPAP adjusted at 10 to 15 minute intervals the patients PaO2 was corrected without the intervention of mechanical ventilation. With initiation of CPAP therapy the clinical goals were reached, namely:

1. Circulatory and perfusion status remained adequate and stable.
2. Adequate arterial oxygenation at FIO_2 of .40
3. Work of breathing was significantly diminished; the patient was comfortable.

The patient remained on CPAP therapy for an additional 48 hours and was successfully placed on 21/m Nasal Cannula.

Case No. 4 Chronic Bronchitis (Blue Bloater) in a 56-Year –Old Male:

A 56- Year-old male is admitted to the emergency room extremely short of breath, with fever, chills and a non-productive cough. He reports a hospitalization for pneumonia last month and returned to work this past week, but complains of lethargy and difficulty in focusing and concentrating. He has a 38- year history of smoking a pack a day. He has tried to quit on several occasions but has been unsuccessful. He also has a history of high blood pressure, congestive heart failure and Type II diabetes. The patient states that he has had a chronic cough but has difficulty in raising secretions. He has to use his inhaler every one to two hours during the night. The M.D.I. contains albuterol and it gives him minimal relief.

Clinically, he presents as an obese, Caucasian male in moderately severe distress. His breathing is labored, rapid, and shallow. His respirations are 28 breaths per minute. Breath sounds are decreased to absent with a faint end-expiratory wheeze over the posterior basal lung fields. The expiratory phase is prolonged. There is a hyperresonance to percussion. Tactile fremitus is present over both fields. The patient is tachycardic, and at times flushed and diaphoretic.

Vital signs:

temperature 37 ° C (98.6 °F)

pulse=124 bpm

respirations = 32/ min

B.P.= 185/112

Physical examination reveals a moderately obese patient with a barrel chest. He has clubbing of his fingers. He is cyanotic and 2+ pitting edema of his ankles. Respirations are labored and he is pursed lip breathing. He is using his accessory muscles of respirations and appears tired and weak. He also appears dehydrated. The patient's skin is hot and moist. Breath sounds are unequal to auscultation with expiratory wheezes throughout both lung fields. He has a weak cough with small amounts of a thick tenacious yellow secretion. A Chest X-ray and an EKG were obtained and a venous blood sample was collected for Hematology and Chemistry analysis.

Arterial blood gasses were drawn on room air and the results reveal:

pH_a = 7.55

P_aCO2= 58 mm. Hg.

HCO3= 32 mEq./L.

P_aO2 = 42 mm. Hg.

The patient is admitted to the hospital and he is immediately placed on oxygen via a nasal cannula at 2 lpm. An I.V. is started to rehydrate the patient and a catheter was inserted to monitor his intake and output. The patient was placed on the appropriate cardiac and pulmonary medications as well as a diuretic and potassium replacement.

One-week later, prior to discharge, Pulmonary Function studies were performed. Spirometry revealed an FEV of 75% of predicted. The patient was discharged from the hospital on 1.5 L./min. of oxygen via a nasal cannula with instructions for referral to enroll in a Pulmonary Rehabilitation Program. Prior to discharge, the patient's arterial blood gas values were:

pH_a = 7.37

P_aCO2 =65mm. Hg.

HCO3 = 35 mEq./L

P_aO2 = 65 mm. Hg.

Case No. 5 Bronchlectasis in a 25- Year Old Female:

A 25-Year-old female is admitted to the emergency room with fever, chills, general malaise and a chronic cough. She has a continuous productive cough of large amounts of foul-smelling, purulent sputum. She also has some hemoptysis.

She complains of constant and severe shortness of breath and appears cyanotic. She is pursed-lip breathing and uses her accessory muscles to breathe. During each coughing episode, she expectorates large amounts of foul-smelling, yellowish-green sputum.

Clinically, she presents as a well-nourished Hispanic female in moderate distress. Her breathing is labored at times with a frequent forceful cough. Respirations are 28 per minute. Breath sounds reveal both sonorous rales and rhonchi. The expiratory phase is prolonged. There is a hyperresonance to percussion. The patient is tachycardic, flushed and diaphoretic. She reports a productive cough whenever she changes positions, especially when she is lying in bed. When she collects her sputum in a container, it separates into three distinctive layers.

Vital signs:

temperature = 37.9° C (100.2° F)

pulse =115bpm

respirations=28/min

BP =180/95

Physical examination reveals a moderately obese, female in mild respiratory distress. The patient's skin is warm and dry. Breath sounds are equal to auscultation with expiratory wheezes throughout. She appears somewhat cyanotic with some digital clubbing.

Her white count was elevated and a sputum sample was obtained for a culture and sensitivity. Arterial blood gases were drawn on 2 L/min. of oxygen and the results reveal:

$pH_a = 7.28$

$P_aCO2 + 54$ mm. Hg

HCO_3 = 32m Eq./SL

P_aO_2=66mm. Hg.

Her chest X-rays revealed a generalized cystic bronchiectasis, alveolar hyperinflation, and increased bronchovascular markings. The current chest film revealed opacity in the left lower lobe that is consistent with atelectasis or an acute pneumonic process.

She was placed on oxygen via a nasal cannula at 3 lpm, given fluids orally and started on a gram-negative antibiotic.

After several days, the patient began to feel better. Pulmonary Function studies were performed. Spirometry revealed a marked improvement in both her static lung volume and her flowrates. Her FEV1 increased to 73%.

The sputum culture revealed Pseudomonas aeruginosa and some colonies of Streptococcus pneumoniae. Her antibiotic regimen was modified accordingly. She was placed in a Secretion Management Protocol, which was used in conjunction with positive expiratory pressure (P.E.P.), and the Percussionaire. This was maintained for several days until the amount of sputum produced was less than 5 ml. per therapy.

The patient was discharged with instructions on how to perform effective autogenic bronchopulmonary drainage. The patient was also instructed on the use of a mechanical mucus clearance device (Flutter Valve).

Case No. 6 Cystic fibrosis in an 18-Year-Old Male:

An 18- year –old Caucasian male is admitted to the emergency room with fever, chills general malaise and a chronic productive cough. He has a continuous productive cough of large amounts of sweet-smelling, purulent sputum. He also has some hemoptysis.

The patient complains of constant and severe shortness of breath and appears cyanotic. He utilizes his accessory muscles of respiration.

In addition he is pursed-lip breathing and appears agitated. During each coughing episode, he expectorates large amounts of sweet- smelling, thick, tenacious yellow-green sputum. Clinically, he presents as a pale, thin emaciated white male in moderate distress. He has a barrel chest and his breathing is labored at times with a frequent, forceful cough. Respirations are 28 per minute. Breath sounds are decreased to absent with faint end-expiratory wheezes over the posterior basal lung fields. Crackles and rhonchi are heard throughout both lung fields. His expiration phase is prolonged. There is a hyperresonance to percussion. The patient is tachycardic, flushed and diaphoretic. He also has a huge appetite and fatty, foul-smelling stools.

Vital signs:

temperature = 38.9° C (101.2 °F)

pulse = 116 bpm

respirations = 20/min

B.P=135/85

Physical examination reveals a thin, pale white male in mild respiratory distress. The patient's skin is warm and dry. Breath sounds are equal with inspiratory and expiratory wheezes throughout. There is an accompanying hyperresonance to percussion. His neck veins are distended and he shows mild to moderate peripheral edema. He states that his shortness of breath has been getting progressively worse.

Arterial blood gasses were drawn and the results reveal:

pH_a = 7.52

P_aCO2 = 58mm. Hg.

HCO3 = 43 mEq./L.

P_aO2 = 62 mm. Hg.

He is placed on oxygen via a nasal cannula at 4 lpm. The patient is admitted to the hospital for a "tune-up". Venous blood was obtained for Hematology and Chemistry. Sputum was obtained for culture and sensitivity. Patient was also started on a pulmonary toilet regimen.

The sputum culture revealed Pseudomonas aeruginosa and some colonies of Streptococcus pneumoniae. His antibiotic regimen was modified accordingly. He was placed in a Secretion Management Protocol, which was used in conjunction with positive expiratory pressure (P.E.P.), and the Percussionaire. This was maintained for several days until the amount of sputum produced was less than 5 ml. per therapy.

One week later the patient was discharged.

Case No. 7 Pickwickian Syndrome in a 25-Year-Old Female:

A 25-year-old, 5 foot, 350 pound female is admitted to the emergency room in an obtunded state. She complains of a constant headache, lethargy and the inability to focus, concentrate or stay awake. Clinically, she presents as an obese white female in severe respiratory failure.

Vital signs:

temperature = 37° C (98.6 °F)
pulse = 130 bpm
respirations = 6/min
B.P. = 100/65

Physical examination reveals a morbidly obese, white female in respiratory failure. The patient's skin is cool and dry. Breath sounds are decreased, but equal to auscultation.

Arterial blood gasses are drawn on room air and the results reveal:

pHa = 7.20
P_aCO2 = 86 mm. Hg.
HCO3 = 18 mEq./L.
P_aO2 = 46 mm. Hg.

The Alveolar-arterial (PaO2-PaO2) gradient is normal. The patient is intubated, committed to mechanical ventilation and transferred to the Medical Intensive Care Unit.

The patient is placed on a vigorous weight reduction program and a Gastro-Intestinal consult is requested.

Six days later the patient was weaned from the ventilator. In preparation for extubation, the procedure was explained to the patient. The patient was then hyperoxygenated with 100% oxygen for several minutes. Secretions were aspirated from the trachea and then from the oro and nasopharynx. The cuff was deflated and the tube removed at the point of end inspiration. Then the patient was coached to cough as deeply as possible to clear secretions.

Just prior to discharge, the patient's arterial blood gas values were:

$pH_a = 7.40$

$P_aCO2 = 40mm. Hg.$

$HCO3 = 24 m. Eq./L.$

$P_aO2 = 92 mm. Hg.$

The patient was discharged on a nutrition-exercise program and referred to a behavior modification support group.

Case No. 8 Pneumonia (Bacterial) in a 25-Year-Old Male:

A 25-year-old Caucasian male schoolteacher is admitted to the emergency room with mild fever, chills, general malaise and a hacking, non-productive cough. He complains of constant lethargy and "flu-like" symptoms for the past five days, and he is getting progressively worse. He is very short of breath and his cough is becoming more frequent.

Clinically, he presents as a tall, thin, white male in moderate respiratory distress. His breathing appears labored at times with a frequent, forceful cough. Respirations are 28 per minute. Breath sounds are decreased to absent with a faint end-expiratory wheezes over the posterior basal lung fields. The patient states that he is very short of breath. He has a frequent, strong,"hacking" sometime productive cough. The patient coughs up a small amount of yellow-brown, blood-streaked sputum.

Vital signs:

temperature = 38.3 °C (101.2° F)

pulse = 126 bpm

respirations = 32/min

B.P. = 166/89

Physical examination reveals a well developed, well nourished white male in mild respiratory distress. His skin is pale and damp. Breath sounds are unequal to auscultation with bronchial breath sounds over the right lower lung regions posteriorly. There are also faint expiratory wheezes throughout the right lower lung. There is an accompanying dullness to percussion posteriorly over the right lower same.

A venous blood sample is obtained for a C.B.C. and a sputum specimen for culture and sensitivity. Both are sent to the clinical laboratory.

Arterial blood gases are drawn and the results reveal:

pH_a = 7.56

P_aCO2 = 24 mm. Hg.

HCO3 = 20 mEq. /L.

P_aO2 = 58 mm. Hg.

The patient's chest film reveals right lower lobe infiltrates, air bronchograms, and alveolar consolidation. His W.B.C. is 23,500. The patient is hydrated, placed on oxygen via a nasal cannula at 5 lpm and started on the appropriate antibiotic therapy. The next morning, the chest X-ray indicates partial resolution of the pneumonic process but a persistent atelectasis or consolidation in the right lower and middle lobes. The tactile and vocal fremitus has increased. Dull percussion notes and bronchial breath sounds are still heard. The patient is hospitalized for an additional day.

On the next day, the arterial blood gases are drawn on room air and the results reveal:

pH_a = 7.44

P_aCO2 = 36mm. Hg.

HCO_3 = 22 mEq. /L.

P_aO_2 = 58 mm. Hg.

The patient is discharged to home to resume his normal daily activities.

Case no. 9 Spontaneous Pneumothorax in a 16 Year-Old Male:

A 16 year-old white male is admitted to the emergency room with severe dyspnea and in obvious respiratory distress. The patient was jogging around the track of a local college when he suddenly became short of breath. The patient states that one minute he was breathing fine and the next minute he suddenly became short of breath.

Clinically, the patient presents as a thin, well-developed, well nourished white male in moderate distress. His breathing is labored with a frequent, forceful cough. Respirations are 22 per minute. Breath sounds are decreased to absent on both sides with a faint end-expiratory wheeze over the posterior basal lung fields. The expiratory phase is prolonged. There is a hyperresonance to percussion bilaterally. The patient is tachycardic, flushed and slightly diaphoretic.

Vital signs:

temperature = 37.9° C (100.2 °F)

pulse=95 bpm

respirations=22/min

B.P.= 140/85

Physical examination reveals a thin white male in mild respiratory distress. The patient's skin is warm and dry.

Breath sound is unequal to auscultion with expiratory wheezes throughout the left. There is an accompanying dullness to percussion on the left side. The trachea is shifted to the right side.

Arterial blood gasses were drawn on room air and the results revealed:

pH_a = 7.52

P_aCO_2=48 mm. Hg.

HCO3 = 36 mEq. /L.

P_aO2 = 58 mm. Hg.

Chest X-ray revealed a dark, translucent area ion the left lung field confirming the presence of air in the intrapleural space on the left side. The patient was immediately placed on oxygen via a nasal cannula at 6 lpm to assist in the reduction of the left-sided pneumothorax. After eight hours, a repeat X-ray shows no substantial reduction in the pneumothrax, therefore, a chest tube was inserted and connected to a pleuravac and 25-cm. of negative pressure. The patient was admitted to the medical floor. On the following day, a follow-up chest film showed the lung to be fully reexpanded.

The patient was discharged to home.

Case No. 10 Pulmonary Embolic Disease in a 25-Year Old Female:

An agitated 25-year-old Asian female truck driver is admitted to the emergency room with extreme shortness of breath. She complains of a constant and severe shortness of breath and she appears both shockey and cyanotic. She has a productive cough, of small quantity of blood-tinged sputum. She states she has a feeling of impending doom. The patient went on to state that she feels awful and has both chest pressure, chest pain, and is light headed. She experiences marked dyspnea with minimal exertion.

Vital signs:

Temperature = 37.9∗C (100.2*F)

pulse = 125 bpm

respirations = 32/min

B.P. = 90/45

Chest examination is unremarkable. The patient denies smoking cigarettes, but her fingers have tobacco stains. Her E.K.G. alternates between normal sinus rhythm, sinus tachycardia, and atria flutter. Echocardiography reveals right atrial and right ventricular enlargement. The estimated pulmonary artery pressure is 55 mm.Hg.

Arterial blood gases were drawn and the results reveal:

$PH_a = 7.54$

$P_aCO^2 = 28$ mm. Hg.

$HCO_3 = 21$mEQ./L.

$P_aO_2 = 53$mm. Hg.

The patient is immediately placed on oxygen via a nasal cannula at 51pm with instructions to keep the $SaO_2 > 92\%$. A ventilation-perfusion scan is ordered and I.V. streptokinase administered. The patient is admitted to the Coronary unit and prepped for a P.T.E.

A Pulmonary Function study demonstrated a rather significant restrictive defect with a total lung capacity of 62% of predicted. There is also a mild obstructive component with an FEV_1/FVC of 70%. The DLCO is 12.3 (38% of predicted).

A ventilation/perfusion scan describes matched ventilation/perfusion defects in each lung. The chest CT scan demonstrates apparent filling defects in both proximal lower lobe pulmonary arteries. Also noted were apical bullous emphysematous changes. The patient has significant lung disease and a level of dyspnea, which seems disproportionate to her pulmonary hypertension. A formal evaluation did confirm the presence of a pulmonary thrombus. The patient underwent a pulmonary thromboendarterectomy. Her recovery was uneventful.

One week later, the patient was discharged from the hospital.

Case No. 11 Pulmonary Edema in a 72-Year-Old Male:

An anxious 72-year-old black male presentes to the emergency room in respiratory distress with a frequent cough. He was producing small amounts of frothy pink secretions. His neck veins and abdomen are distended, and there is 3+ pedal edema. He complains of constant and severe shortness of breath and appears cyanotic.

Clinically, he presents as a thin, emaciated black male in moderate distress. He has a barrel chest and uses his accessory muscles of respiration. His breathing is labored at times with a frequent, forceful cough. Respirations are 28 per minute. Breath sounds are decreased to absent with faint end-expiratory wheezes over the posterior basal lung fields. The expiratory phase is prolonged. There is a hyper-resonance to percussion. The patient is tachycardic, flushed and diaphoretic.

Vital signs:

temperature = 37.9° C (100.2° F)

pulse = 145 bpm

respirations = 24/min

B.P. = 180/125

Physical examination reveals a emaciated, black male in acute respiratory distress. His skin is cool and moist. Breath sounds were decreased and equal to ausculation with expiratory wheezes throughout. There is an accompanying dullness to percussion.

Arterial blood gases are drawn on 4 lpm. And the results reveal:

pH_a = 7.54

P_aCO2 = 28 mm. Hg.

HCO3 = 22 mEq. /L.

P_aO2 = 61 mm. Hg.

His oxygen via a nasal cannula is increased to 5 lpm. The chest X-ray shows dense, fluffy opacities over the lower lung areas bilaterally. It is also noted the presence of right ventricular hypertrophy. The patient is stabilized in the emergency room and then transferred to the cardiac Care Unit.

Since the patient was still hemodynamically unstable, a Swan-Ganz multi-lumen flow-directed catheter was inserted and hemodynamic measurements were obtained. His cardiac output was 2.5 L/min., which is reduced, as well as his cardiac index.

His SVR was 650 and his PVR was 225. His wedge pressure (L.V.E.D.P.) was reduced indicating Left Ventricular Congestive Heart Failure. Blood Urea Nitrogen was elevated and the serum sodium was reduced. Intake and Output reflect positive fluid balance.

The patient was placed on the appropriate cardiogenic medications to increase the mean arterial pressure and a diuretic to establish the fluid compartments. He responded hemodynamically and began to normalize.

The patient was transferred out of the unit and enrolled in the Cardiac Rehabilitation Program.

Case No 12 Pulmonary Tuberculosis in a 25-Year-Old Female:

A 25-year-old black female is admitted to the emergency room reporting night sweats, chills, general malaise and a chronic cough. A few days ago, she developed episodes of coughing that were more severe than usual. On several occasions, the coughing episodes lasted for up to an hour. During these episodes, her sputum was sometimes blood-tinged (hemoptysis). Prior to this admission, she coughed up “frank” bright-red blood. She complains of constant and severe shortness of breath and she has difficulty in catching her breath. During each of these coughing episodes, she brings up large amounts of blood with sometimes yellowish sputum.

Clinically, she presents as a thin, almost emaciated black female in moderate distress. She uses her accessory muscles for respiration. Her breathing is labored at times with a frequent, forceful cough. Respirations are 25 per minute. Breath sounds are decreased to absent with faint end-expiratory wheezes over the posterior basal lung fields. Although palpation of the chest is negative, dull percussion notes and increased tactile and vocal fremitus are noted over the lung bases. Bronchial breath sounds are heard over the right and left lung bases. Crackles and rhonchi are noted over the left upper lobe.

Vital signs:

temperature = 38.9° C (101.2°.F)

pulse =120 bpm

respirations=28/min

B.P. = 130/85

Physical examination reveals a black female in respiratory distress. The patient's skin is warm and dry. The Chest X-ray reveals an increase in opacities consistent with pneumonia in the left lower lobe. The white count came back at 16,500.

Arterial blood gases are drawn and the results reveal:

pH_a = 7.54

P_aCO_2 = 50 mm. Hg.

HCO3 = 41 mEq/L.

P_aO2 = 50 mm. Hg.

She is immediately placed on oxygen via a nasal cannula at 4 lpm.

The P.P.D. (Mantoux) skin test is placed. A sputum specimen is obtained and sent to the clinical laboratory for an A.F.B. and the patient is started on antituberclin medications.

The patient is started on Isoniazid, Ethambutol and Streptomycin, but develops a skin rash after two days. The Ethambutol is discontinued and replaced with Rifampin.

After several days, the patient began to show improvement and her oxygen was reduced to 2 lpm. A repeat Chest X-ray showed that the parenchymal densities had improved. The sputum showed Mycobacterium tuberculosis.

The patient was discharged two days later.

Case No. 13 Near Drowning in a 19-Year-Old Male:

An agitated 19-year old white male near-drowning victim presents to the emergency room in respiratory distress breathing spontaneously.

He and some of his friends were having a party aboard a pleasure craft on Mission Bay. The young man was sitting on the railing when something jarred the craft and the patient went overboard. He was not breathing when he was pulled from the water. The owner of the boat called the harbor police and the Coast Guard and two friends began CPR When the harbor police arrived, they continued C.P.R. The patient remained unconscious and his pupils were fixed and dilated. He had no cardiac activity and no spontaneous respirations. An esophageal obturator was inserted and the patient was placed on 100 % oxygen while C.P.R was continued. Within several minutes, the boat docked at shore where an ambulance was waiting. The patient was transported to Mission Bay Hospital. He was semiconscious, combative and belligerent. His pupils were no longer fixed and dilated.

Clinically, he presents as a well-nourished, white male in respiratory distress. He has symetric chest excursion. His breathing is labored at times with a frequent, forceful cough. Respirations are 28 per minute.

Breath sounds are decreased to absent with faint fine crackles and end-expiratory wheezes over the posterior basal lung fields. The expiratory phase is prolonged. The patient is tachycardic.

Vital signs:

temperature = 32.3° C (90.3 °F)

pulse= 122 bpm

respriations= 28/min

B.P. = 124/82

Physical examination reveals an obtunded patient in respiratory distress. Arterial blood gasses were drawn on 100% and the results revealed:

$pH_a = 7.10$

$P_aCO_2 = 75$ mm. Hg.

HCO_3 = 16 mEq./L.

P_aO2 = 54 mm. Hg.

The patient was intubated, placed on a mechanical ventilator and transferred to the Surgical Intensive Care Unit. After he was sedated and stabilized, another arterial blood gas was drawn and a portable chest film taken. The Chest X-ray showed that the endotracheal tube was correctly positioned and bilaterally patchy infiltrates were noted in both lower lobes. There were also dense, fluffy opacities over the lower lung areas bilaterally. The arterial blood gasses were drawn on an F_1O_2 of .50 and the results reveal:

pH_a = 7.23.

P_a CO2 = 51 mm. Hg

HCO3 = 19 mEq. /L.

P_aO2 = 54 mm. Hg.

A Swan-Ganz catheter was placed. The patient's condition began to deteriorate. Dull percussion notes were now heard throughout both lung fields, and more prominent crackles and rhonchi were heard over both lungs. A moderate amount of frothy, white sputum was being suctioned.

The patient's cardiopulmonary status remained critical. Despite aggressive suctioning, crackles and rhonchi were now abundant throughout all lung fields. Frothy, pink secretions continued to be suctioned. A follow-up Chest X-ray revealed fluffy infiltrates throughout both lung fields and was consistent with a pulmonary edema pattern.

The patient was discharged two day later.

Glossary of Terms

Acinus *pl.* Acini [L, *grape*], 1. Any small sac-like structure, particularly one found in a gland. 2. A subdivision of the lung consisting of the tissue distal to a terminal bronchiole. Also called alveolus.

Antigen [Gk, *anti* + *genein*, to produce], a substance, usually a protein, that causes the formation of an antibody and reacts specifically with that antibody.

Apices [L, *apices*], referring to the upper lobe of one lung.

Auscultation [L, *auscultare*, to listen], the act of listening for sounds within the body to evaluate the condition of the heart, blood vessels, lungs, pleura, intestines or other organs. Auscultation may be performed directly, but most commonly a stethoscope is used to determine the frequency, intensity, duration, and quality of the sounds. During auscultation of the lungs the patient usually sits upright and is instructed to breathe slowly and deeply through the mouth. The anterior and posterior surfaces of the thorax are auscultated from apex to base with comparasons made between the right and left sides; when the posterior chest is examined, the patient is asked to bring the shoulders forward so that a greater surface of lung can be auscultated.

Bronchiole [L, *bronchiolus*, little windpipe], a small airway of the respiratory system extending from the bronchi into the lobes of the lung.
There are two divisions of bronchioles: The terminal bronchioles pass inspired air from the bronchi to the respiratory bronchioles and expired waste gases from the respiratory bronchioles to the bronchi. The respiratory bronchioles function similarly, allowing the exchange of air and waste gases between the alveolar ducts and the terminal bronchioles.

Calcification [L, *calx* + *facere*, to make], the accumulation of calcium salts in tissues. Normally, about 99% of all the calcium entering the human body is deposited in bones and teeth; the remaining 1% is dissolved in body fluids such as the blood. Disorders

affecting the delicate balance between calcium and other minerals, parathyroid hormone, and vitamin D can result in calcium deposits in arteries, kidneys, lung alveoli, and other tissues, interfering with usual organ function.

Caseation [L, *caseus*, cheese], a form of tissue necrosis in which there is loss of cellular outline and the appearance is that of crumbly cheese. It is typical of tuberculosis.

Cavernous rale [L, *caverna* + Fr *rattle*], an abnormal hollow, metallic sound heard during auscultation of the thorax. It is caused by contraction and expansion of a pulmonary cavity during respiration and indicates a pathologic condition.

Compliance [L, *complere*, to complete], 1. Fulfuillment by the patient of the care-giver's prescribed course of treatment. 2. A measure of distensibility of the lung volume produced by a unit pressure change.

Crepitus [L, crackling], a sound that resembles the crackling noise heard on an open fire. The rales of a consolidated area of the lung in pneumonia. Also called crepitation.

Croup [Scot, to croak], an acute viral infection of the upper and lower respiratory tract that occurs pimarily in infants and young children 3 months to 3 years of age after an upper respiratory tract infection. It is characterized by hoarseness, fever, a distinctive harsh, brassy cough, respiratory distress resulting from obstruction of the larynx.
The most common causative agents are the parainfluenza viruses, especially type 1, followed by the respiratory syncytial viruses (RSV) and influenza A and B viruses.

Diagnosis *pl.* diagnoses [Gk, *dia* + *gnosis*, knowledge]. 1. Identification of a disease or condition by a scientific evaluation of physical signs, symptoms, history, laboratory test, and procedures. Kinds of diagnoses are clinical diagnosis, differential diagnosis, laboratory diagnosis, nursing diagnosis and physical diagnosis. 2. The art of naming a disease or condition.

Dyspnea [Gk, *dys* + *pnoia*, breathing], a shortness of breath of a difficulty in breathing that may be caused by certain heart conditions, strenuous exercise, anxiety or a verity of pulmonary conditions.

Effusion [L, *effundere*, to pour out], 1. The escape of fluid from blood vessels because of rupture of seepage, usually into a body cavity. The condition is usually associated with a circulatory or renal disorder and is often an early sign of congestive heart disease. The term may be associated with a affected body area, as pleural or pericardial effusion

Etiology [Gk., *atia*, cause, *logos,* science], 1. The study of all factors that may be involved in the development of a disease, including susceptibility of the patient, the nature of the disease agent, and the way in which the patient's body is invaded by the agent. 2. The cause of a disease.

Fibrous [L, *fibra*, fiber], consisting mainly of fibers or fiber-containing materials, such as fibrous connective tissue.

Hypoxemia [Gk, *hypo* + *oxys*, sharp, *genein*, to produce, *hamia*, blood], an abnormal deficiency of oxygen in the arterial blood. Symptoms of acute hypoxemia are cyanosis, restlessness, stupor, coma, Cheyne- Stokes breathing, apnea, increased blood pressure, tachycardia, and an initial increase in cardiac output that later falls, resulting in hypotension and ventricular fibrillation or asystole. Chronic hypoxemia stimulates red blood cell production by the bone marrow, leading to secondary polycythemia. Hypoxemia caused by decreased alveolar oxygen tension or underventilation improves with oxygen therapy.

Hypoxemia resulting from shunting of blood from the right side of the heart to the left side of the heart without exchange of gases in the lungs is treated with bronchial hygiene and positive and expiratory pressure therapy.

Insidious [L, *insidiousus*, cunning], of, pertaining to, or describing a development that is gradual, subtle, or imperceptible. Certain chronic disease, such as glaucoma, can develop insidiously with symptoms that are not detected by the patent until the disorder is established.

Lesion [L, laesus, an injury], 1. A wound, injury or pathogenic change in body tissue. 2. Any visible, local abnormality of the tissues of the skin, such as a wound, sore, rash or boil. A lesion may be described as benign, cancerous, gross, occult, or primary.

Leukocytosis [Gk, *leukos= kytos*, cell, *osis*, condition], an abnormal increases in the number of circulating white blood cells. An increase often accompanies bacterial, but not usually viral, infections. The normal range is 5000 to 10,000 white cells per cubic millimeter of blood. Leukemia may be associated with a white blood cell count as high as 500,000 to 1 million per cubic millimeter of blood, the increase being either equally or disproportionately distributed among all types. Kinds of leukosytosis include basophilia, eosinophilia, and neutophilia.

Membrane [L, *membrana*, thin skin], a thin layer of tissue that covers a surface, lines a cavity, or divides a space, such as the abdominal membrane that lines the abdominal wall and Descernet's membrane between the subtantia propria and the endothelium of the cornea. The principal kinds of membranes are mucous membrane, serous membrane, synovial membrane, and cutaneous membrane.

Mucopurulent [L, *mucus* + *purulentus*, puss], characteristic of combination of mucus and pus.

Orthopnea [Gk, *orthos* + *pnosis*, breath], an abnormal condition in which a person must sit or stand to breathe deeply or comfortably. It occurs in many disorders of the cardiac and respiratory systems, such as , pulmonary edema, emphysema, pneumonia, and angina pectoris.

Panlobular [Gk, *pan* + *lobos*, all lobes], any condition involving all areas of the lung.

Paradoxical breathing [Gk, *paradoxos*; AS, *breath*], a condition in which a part of the lung deflates during inspiration and inflates during expiration. The condition usually is associated with a chest trauma, such as an open chest wound or rib cage damage.

In such cases, the paradoxical breathing that occurs spontaneously is sometimes called internal paradoxical breathing. External paradoxical breathing may be observed during deep general anesthesia.

Parencyma [Gk, *papa* + *enchyma*, infusion], the tissue of an organ as distinguished from supporting or connective tissue.

Pleura [Gk, rib], a delicate serous membrane enclosing the lung, composed of a single layer of flattened mesothelial cells resting on a delicate membrane of connective tissue. Beneath the membrane is a stroma of collagenous tissue containing yellow elastic fibers. The pleura divides into the visceral pleura, which covers the lung, dipping into the fissures between the lobes, and the parietal pleura, which lines the chest wall, covers the diaphragm, and reflects over the structures in the mediastinum. The parietal and visceral pleurae are separated from each other by a small amount of fluid that acts as a lubricant, as the lungs expand and contract during respiration.

Purulent [L, containing pus], producing or containing pus.

Shunt [ME, *shunten*], 1. To redirect the flow of a body fluid from one cavity or vessel to another. 2. A tube or device implanted in the body to redirect a body fluid from one cavity or vessel to another.

Steroid [Gk, *stereos* + *eidos*, form], any of a large number of hormonal substances with a similar basic chemical structure, produces mainly in the adrenal cortex and gonads.

Thoracostomy [Gk, *thorax* + *stoma*, mouth], an incision made into the chest wall to provide an opening for the purpose of drainage.

Thoracotomy [Gk, *thorax* + *temnein,* to cut], a surgical opening into the thoracic cavity.

Tissue [Fr, *tissu*, fabric], a collection of similar cells acting together to perform a particular function.

Trypsin [Gk, *tripsis*, rubbing], a proteolytic digestive enzyme produced by the exocrine pancreas that catalyzes in the small intestine the breakdown of dietary proteins to peptones, peptides, and amino acids.

Tussive fremitus [L, *tussis*, cough + *fremitus*, murmuring], a vibratory cough that can be felt by a hand over the chest of the patient.

RESPIRATORY DISEASES

POST TEST

Instructions: Select the single most correct response.

1. The key factor in the etiology of chronic bronchitis is:

    a. Heredity

    b. Sex

    c. Age

    d. Cigarette smoking

2. Centrilobular emphysema primarily affects the:

    a. Middle of the acinus and respiratory bronchioles

    b. Entire acinus

    c. Large airways

    d. Pleural space

3. Typical findings with Emphysema include a prolonged expiratory phase and "barrel" chest appearance.

    a. True

    b. False

4. $Alpha_1$ antitrypsin is a:

    a. Fungus

    b. Enzyme

    c. Proteinase inhibitor

    d. Virus

5. Which of the following are associated with emphysema:

I. Hyperinflation

II. Barrel chest

III. Dyspnea

IV. Orthopnea

a. II and III

b. I, III and IV

c. I, II and III

d. I, II, III and IV

6. In Bronchiectasis (Case #5), the sputum collected from her will typically separate into three distinct layers in the sputum collection container.

a. True

b. False

7. Which of the following are types of bronchiectasis:

I. Panacinar

II. Cylindrical

III. Saccular

IV. Parcicatrical

a. I and II

b. II and III

c. I, II and III

d. I, II, III and IV

8. Asthma is:

a. Common chronic lung disease

b. May be triggered by allergens, cold weather, or exercise

c. Has episodes of bronchospasm that are usually short lived

d. All the above

9. Which of the following diseases has a reversible component:

a. Chronic bronchitis

b. Emphysema

c. Asthma

d. Bronchiectasis

10. Cystic Fibrosis is a contagious disease usually contracted by a mother or father of the patient.

a. True

b. False

11. If both parents of a child are carriers of the mendelian recessive trait, the child has a 75% chance of developing cystic fibrosis.

a. True

b. False

12. Pulmonary secretions of the patient with cystic fibrosis are usually:

a. Thin and watery

b. Pink and frothy

c. Bloody

d. Viscous and tenacious

13. Upon physical examination of the cystic fibrosis patient, you may find:

I. Barrel-shaped chest

II. Chest hyper-resonance to percussion

III. Cyanosis

IV. Clubbing of fingers

V. Obesity

a. I, II, III

b. IV, V

c. I, II, III, IV

d. V only

14. Differential diagnosis for Cystic Fibrosis includes:

a. Elevated blood potassium with decreased sodium

b. Increased sweat chloride

c. Arterial Blood gas with metabolic alkalemia

d. Elevated blood urea nitrogen and creatine

15. Cystic Fibrosis is an exocrine disease which affects both the pulmonary system and the digestive system.

a. True

b. False

16. *Staphylococcus aureus* and *Pseudomonas aeruginosa* are frequently cultured from the sputum of cystic fibrosis patients.

a. True

b. False

17. Which of the following is a mucolytic or enzymatic agent

I. Sterile water

II. Acetylcystiene

III. Albuterol

IV. Isoproterenol

V. Dornase

a. II only

b. I, II, IV

c. III, V

d. II, V

e. I only

18. Pneumothorax or Mediastinal Emphysema may occur in patients with cystic fibrosis.

a. True

b. False

19. The greatest challenge in management of the patient with cystic fibrosis is related to gastro-intestinal complications.

    a. True

    b. False

20. The etiology of the pneumoconioses pertains to:

    a. Inhaled dust

    b. Inhaled droplets from bird droppings

    c. A fungal infection

    d. A bacterial infection

21. The following diseases fall into the category of pneumoconiosis:

    I. Asbestosis

    II. Silicosis

    III. Berylliosis

    IV. Sarcoidosis

    a. I and II

    b. I, II and III

    c. I, II, III and IV

    d. I, II and IV

22. A lateral curvature of the spine is:

    a. Kyphosis

    b. Scoliosis

    c. Kyphoscoliosis

    d. Pectus excavatum

23. “Black lung” refers to:

    a. Sarcoidosis

    b. Worker’s pneumoconiosis

    c. Asbestosis

    d. Silicosis

24. The primary diagnostic tool in the determination of sarcoidosis is:

a. The physical examination

b. The chest x-ray

c. Blood gases

d. Pulmonary function studies

25. Major primary muscular diseases affecting respiratory function are:

I Cystic Fibrosis

II. Muscular Dystrophies

III. Aspiration Pneumonia

IV. Inflammatory Myopathies

a. I, II

b. I, II, IV

c. II, III, IV

d. II, IV

26. The most common Muscular Dystrophy is:

a. Guillain Barre'

b. Kussmaul

c. Duchenne

d. Poliomyelitis

27. In Duchenne dystrophy, the muscle weakness starts late in life.

a. True

b. False

28. Nerve Impulses are transmitted to the muscles by liberation of:

a. Acetylcholinesterase

b. Acetylcholine

c. Neostigmine

d. Potassium

29. Diagnosis of Myasthenia Gravis is made by:

a. Patient history of progressive muscle weakness with repetitive use of certain muscles

b. Sudden onset of difficulty breathing with chest pain.

c. History of recent foreign travel.

d. Swimming in polluted water.

30. The most important part of management in the patient with Myasthenia Gravis is:

a. Electrolyte Balance

b. Nutrition

c. Diligent respiratory care

d. Hydration

31. A common form of acute polyneuritis is:

a. Myasthenia Gravis

b. Muscular Dystrophy

c. Duchenne Dystrophy

d. Guillain Barre'

32. The primary treatment in the Pickwickian patient involves:

a. Antibiotic therapy

b. Steroid therapy

c. Weight loss

d. Thoracotomy

33. An open pneumothorax is a condition in which there exists:

a. Air entering the pleural space, but not allowed to escape

b. No air movement in or out of the pleural space

c. A free flow of air in and out of the pleural space

d. Air cannot enter the pleural space, but can escape

34. A pleural effusion occurs as a result of:

a. Fluid passing across the pleural membrane

b. Rupture of the pleural membrane

c. Hypoxemia

d. A “shunt effect”

35. A pleural effusion is:

a. Air in the pleural space

b. Fluid in the pleural space

c. Air and fluid in the pleural space

d. The absence of a pleural space

36. ARDS is characterized by:

I. Decreased lung compliance

II. Acute hypoxemia

III. Pulmonary hypertension

IV. An increase in pulmonary vascular resistance

a. I and II

b. I, II and III

c. I, III and IV

d. I, II, III and IV

37. An acute inflammation of the lung parenchyma is called:

a. Pneumonia

b. Emphysema

c. Mucoviscidosis

d. Bronchiectasis

38. Examples of bacterial pneumonia are:

I. Streptococcus pneumoniae

II. Staphylococcus pyogenes

III. Klebsiella pneumoniae

IV. Friedlander's bacillus

a. I and II

b. I, II and III

c. I, II and IV

d. I, II, III and IV

39. Infection caused by fungus include:

I. Coccidiomycosis

II. Blastomycosis

III. Histoplasmosis

IV. Silicosis

a. I and IV

b. I and II

c. I, II and III

d. II and IV

40. A drug found to be effective in the treatment of histoplasmosis is:

a. Beclomethasone

b. Garamycin

c. Penicillin

d. Amphotericin B

41. Treatment of bacterial pneumonia involves:

a. Steroid therapy

b. Antibiotic therapy

c. Amphotericin B

d. INH

42. Active tuberculosis can be positively identified:

a. With a skin test

b. With a skin test and chest x-ray

c. By physical examination

d. By pulmonary function studies

43. Tuberculosis is transmitted:

a. By physical contact

b. By the blood

c. By the feces

d. As an airborne droplet

44. The principle cause of death from burn injury is due to:

a. Upper airway obstruction

b. Respiratory failure

c. Skin damage

d. Dehydration

45. Lung trauma or injury, resulting in ARDS, may be caused by:

I. Trauma

II. Near-drowning

III. Shock

IV. Aspiration of gastric contents

V. Sepsis

a. I, II and IV

b. I, III and IV

c. III, IV and V

d. I, II, III, IV and V

46. Flail chest is characterized by:

a. A tension pneumothorax

b. Paradoxical chest wall movement

c. Symmetrical chest wall movement

d. No chest wall movement

47. Orthopnea is the:

a. Inability to breathe well while standing

b. Inability to breathe well while sitting

c. Bone disorder

d. A condition in which a person must sit or stand to breathe deeply or comfortably

48. In Chronic Bronchitis (Case #4), the 2+ pitting edema of the ankles is the result of the right-sided Congestive Heart Failure (C.H.F.), secondary to his pulmonary disease (Cor Pulmonale).

a. True

b. False

48. An acute viral infection of the upper and lower respiratory tract that occurs primarily in infants and young children 3 months to 3 years of age is:

a. Croup

b. Mucoviscidosis

c. Emphysema

d. Bronchitis

50. An Effusion is:

a. The escape of fluid from blood vessels because of rupture of seepage

b. A condition is usually associated with a circulatory or renal disorder

c. May be associated with a affected body area, as pleural or pericardial effusion

d. All the above

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