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What is the relationship of cleaning frequency of inhalation equipment in the home and the contamination of that equipment?

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What is the relationship of cleaning frequency of inhalation equipment in the home and the contamination of that equipment?

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Abstract

Use of inhalation therapy equipment for the COPD patient in the home setting has increased; the efficacy of the cleaning procedure for the equipment has become a major concern. The COPD patient is at risk for development of pulmonary infections secondary to the actual disease process and resultant debilitated status. The concern that grossly contaminated equipment exposes them further to pulmonary infections is the impetus behind this study.

Summary

The sample study consisted of 18 subjects who utilized either IPPB or compressor powered nebulizers in the home setting. The data collection occurred over a 9 month period from August 1980 to March 1981. Equipment cultures were obtained from the tubings and nebulizers using the Aero-Test Sampler. The study results indicated that contamination of home inhalation equipment is a major problem. In the experimental group 11.1% of the tubings cultured were contaminated while in the control group 16.7% tubings were contaminated. Contamination of nebulizers in both groups was extensive with only three negative (16.7%) cultures in the total sample study. The experimental group was "sicker" and this factor contributed to the non-comparability of the two groups. The study results indicated equipment contamination is influenced by the following variables: corticosteroids, antimicrobials, multiple organ disease, and hospitalizations. These impinging variables directly affected equipment contamination even if the cleaning procedure used was considered effective as a decontaminate.

Chapter One

Problem Area

Introduction

The incidence of chronic obstructive pulmonary disease (COPD) has escalated in the last decade and is considered the second most prevalent chronic disease afflicting the population of the United States. The American Lung Association reports that 8.5 million individuals within the United States suffer from some form of chronic lung disease. This increase in COPD has resulted in the development of new treatment modalities with rehabilitative therapy assuming the major mode of management of patients with lung disease. The goal of rehabilitative therapy is to prevent progression of the underlying disorder and to minimize functional disturbances thereby lessening the symptoms. One common treatment modality that has been widely advocated and integrated into the treatment of patients with COPD is the long term use of intermittent positive pressure breathing (IPPB) or compressor powered nebulizers.

Historically, when IPPB was introduced in 1947 (Werko, Motley, & Cournand) it was acclaimed as an impressive new device with wide applicability and the potential for prevention of debilitating complications for the hospitalized patient. Initially, IPPB was used for the purpose of preventing complications, limiting atelectasis, and reducing the incidence of pneumonia in surgical patients. The gradual evolution of IPPB as a choice treatment modality for the COPD patient occurred in the 1960's. As a result, physicians began to use these devices to deliver sympathomimetic bronchodilators into the tracheobronchial tree. The realization by physicians that aerosolized bronchodilators could provide sustained bronchodilation of the airways encouraged the expansion of this mode of therapy. The other forms of adjunct therapy (oral bronchodilators, breathing retraining, oxygen therapy, and chest physiotherapy) continued to be

used but aerosolization or nebulizer therapy began to be used routinely in the management of patients with COPD (Lertzman and Cherniack, 1976).

The advent of rehabilitative programs along with the development of "portable" IPPB devices and compressor powered nebulizers encouraged expansion of care into the home setting (Lertzman & Cherniack, 1976; Petty, 1974). The therapeutic effects or lack of them associated with the long term use of these devices has been the focus of many research studies and the results have been conflicting.

The long term use of IPPB and compressor powered nebulizers in the supportive care of the COPD patient is controversial. In 1974, Thomas Petty evaluated the use of adjunctive therapy (IPPB) for the purpose of discovering what benefits resulted from the use of IPPB. Petty questioned whether long term use of IPPB actually changed the status of the disease process. His data validates that long term use of IPPB does not slow the progression of the disease process. In fact, he found decreases in the FEV_1 values thereby establishing that actual deterioration of pulmonary function occurred. Numerous studies (Hudson, Tyler, & Petty, 1976; Emirgil, Sobol, Norman, Moskowitz, Goyal, Wadhwani, 1969; Cherniack & Svanhill, 1976; Pierce, Edmonson, McGee, Ketchersid, Loudon, & Sanford, 1966) have substantiated Petty's observation while delineating at least one beneficial result from the use of this modality. These studies reported that patients expressed subjective improvement of symptoms even though their actual pulmonary function status had deteriorated. The controversy over the efficacy of IPPB therapy and benefits derived from its long term use continues to be a debated issue and is not explored in this study. It is, however important to acknowledge these controversies as they relate to the evolution of this treatment modaltiy.

Numerous studies have also been conducted examining the long term use of

IPPB and other inhalation devices within the hospital and the epidemiological implications of contaminated inhalation equipment. The role of inhalation equipment in the hospital setting and its implication in nosocomial pulmonary infections has been thoroughly documented (Reinarz, Mays, Pierce, & Sanford, 1965, Pierce, Thomas, Sanford, and Leonard, 1970; Pierce, Edmonson, McGee, Ketchersid, Loudon, & Sanford, 1966). These studies report a direct relationship between inhalation equipment with reservoir nebulizers and the transmission of gram-negative bacilli. In turn, the reservoir nebulizer proved to be the major site of bacterial contamination and a contributing factor to the development of nosocomial pulmonary infections (Reinarz, Pierce, Mays, & Sanford, 1965).

In spite of the controversy regarding therapeutic benefits derived, IPPB and compressor powered nebulizers are currently utilized extensively in the home setting to facilitate stabilization of the disease process. The efficacy of the assumed physiological benefits from this therapy is significant as the majority of patients in the home use these devices for periods of 2 to 10 years. Also, the probability of the inhalation equipment at home contributing to the development of nosocomial infections is a major concern. The correlation of contaminated equipment in the home with the incidence of pulmonary infections has not been studied. Pierce et colleagues (1970) acknowledged that the effluent gas stream from inhalation equipment can disseminate bacteria into the lung parenchyma thus exposing all "users" to gram-negative bacilli pulmonary infections.

For those patients who are selected to use IPPB or compressor powered nebulizers in the home, the treatments can be complicated and the care of the equipment can be physically exhausting. Appropriate cleaning for their equipment should occur on a daily basis, and the procedures for cleaning are time consuming. In view of the verified contamination that can occur, the task of cleaning the equipment assumes great importance for the patient at home.

These individuals have a high probability of developing pulmonary infections because of the following factors: debilitated physical status, inactivation or loss of normal respiratory defenses, and environmental factors. The susceptibility of the COPD patient to pulmonary infection demonstrates that an effective decontamination method for home inhalation equipment is important.

At present the cleaning procedure most generally recommended involves using a soaking solution of white vinegar (acetic acid) and water to decontaminate this home equipment. The actual frequency and dilutional strength of the white vinegar and water varies from patient to patient primarily because most physicians differ on what is believed to be an appropriate cleaning procedure for home equipment. Hyde et. al. (1979) is the only study that directly examined bacterial contamination of inhalation equipment in the home setting. They substantiated that home equipment can become easily contaminated and they found the major isolates to be gram-negative bacteria. The home units were twice as contaminated as the hospital based equipment. The greater degree of contamination in home equipment emphasizes that home equipment be appropriately decontaminated.

Use of acetic acid as a decontaminate has been examined in several hospital studies (Parker & Hoeprich, 1962; Pierce & Sanford, 1973; Pierce, Sanford, & Thomas, 1970) but few studies have explored its effectiveness in home equipment. Pierce and Sanford (1973) acknowledged that nebulizers become contaminated within 24 hours of clinical use and that immersion of Venturi nebulizers into a disinfectant does not provide adequate decontamination. The confusion about the decontamination process at home focuses on the fact that acetic acid is used as a "soaking solution" rather than a nebulizing solution as recommended by the hospital studies (Pierce & Sanford, 1973). As a result, cleaning of the equipment at home is extremely sporadic and variable; patients,

nurses, and physicians alike suffer from the lack of specific guidelines regarding decontamination of inhalation equipment.

Summary

The controversy about IPPB therapy focuses on the necessity, the efficacy, and the cost-effectiveness of this form of therapy. The increased prevalence of COPD has promoted the expansion of IPPB therapy and compressor powered nebulizers into the home setting thus creating an entirely new set of problems. Inhalation equipment can be a source for the transmission of gram-negative bacteria, and the correlation of nosocomial pulmonary infections with contaminated equipment demonstrates the need for an evaluation of home based equipment. Furthermore, the adequacy of the present cleaning procedure and the debate over the efficacy of acetic acid as a decontaminate demonstrates the importance of evaluating the cleaning and contamination of home based equipment.

Purpose of the Study

The particular question of this study was:

What is the relationship between the type of cleaning procedures used for inhalation equipment in the home and the incidence of contaminated equipment?

Significance of the Study

For those patients using inhalation equipment at home, the pulmonary regimen may involve inhalation treatments 4 times a day including an extensive cleaning procedure, oral medications, postural drainage, percussion and vibration. A basic cleaning procedure for the equipment requires patients to rinse the mouthpiece and nebulizer in tap water after every treatment. In the evening, patients are to wash the equipment (3 tubings and a manifold or 1 tubing and a manifold) in dishsoap and water, and, every other day, follow this routine with a

30 minute soaking in white vinegar and water.

As a rule, COPD patients are dyspneic, tire easily, and frequently exhibit hand tremors secondary to their drug therapy. Furthermore, these patients tend to be homebound, and isolated from the community at large; many are impoverished and frequently have minimal support systems to assist them in their care. These circumstances cause patients to feel bombarded and confused about the technicalities of "caring for themselves". The assumption that some of these patients encounter difficulties in cleaning their inhalation equipment due to physical limitations is accurate and poses a direct threat to their well-being. The possibility that inappropriate cleaning can produce contaminated equipment and, therefore, an increase in pulmonary infections demonstrates the need to evaluate the problem of cleaning this equipment at home.

In 1968 the American Thoracic Society stated that "the cleaning and sterilization of inhalation equipment cannot be overemphasized" (Statement by American Thoracic Society, p. 1). Various methods have been used to clean inhalation equipment such as steam autoclaving, sterilization by ethylene oxide, and decontamination by acetic acid or a glutaraldehyde solution. The most widely and commonly used method for decontamination of this equipment utilizes a 0.25% solution of acetic acid. Pierce and Sanford (1973) studied decontamination of inhalation equipment for the purpose of assessing which method of cleaning was most effective in reducing equipment contamination. This study used 0.25% acetic acid as a nebulizing solution for 10 minutes daily; by using it in this manner Pierce and Sanford (1973) reported only 10% contamination of the equipment while it was in use. In contrast, the American Thoracic Society recommends 2 parts of white vinegar to 3 parts sterile water to decontaminate equipment in the home in place of the 0.25% acetic acid solution. However, the American Thoracic Society fails to recommend the frequency for

using this solution or to provide data on the effectiveness of this solution. The exact length of time (24-48 hours) that the vinegar and water solution is considered effective as a decontaminate is unclear. The confusion over the frequency and actual effectiveness of the vinegar and water solution as a decontaminating solution has partially been responsible for the ambivalent approach to the cleaning of the equipment in the home that exists now.

Moreover, this equipment has been implicated as a vector for the transmission of gram-negative bacilli (Roberts, Cockcroft, Johnson, and Fishwick, 1973; Reinarz et al, 1965; Kelsen, McGuckin, Kelsen, Cherniack, 1977) and is associated with necrotizing pneumonia (Pierce, Edmonson, McGee, Ketchersid, Loudon, & Sanford, 1966; Lepper, 1963). These studies stress the importance of providing an appropriate cleaning procedure for equipment at home. Litsky, Botko, and Litsky (1975) implicated Pseudomonas aeruginosa as one of the most significant organisms that can contaminate inhalation equipment. Because of its physiological characteristics, Pseudomonas aeruginosa can survive and multiply in the usual moist environment of this equipment. Since Pseudomonas aeruginosa is considered to be highly pathogenic and an opportunistic gram-negative bacilli which is difficult to eradicate the efficacy of this decontaminating solution needs to be evaluated. Also, another factor that impinges on efficacy of this decontaminating solution is that patients at home use tap water as the diluent with vinegar rather than sterile water. Use and implications for the various types of water (sterile or non-sterile) used in the cleaning solution will be explored in later chapters.

The tubing attached to the IPPB or compressor powered nebulizers is another source of contamination for the patient at home. It is possible to use either disposable or permanent tubing and manifold for these devices. The permanent tubing, initially more expensive, is flexible, easy to handle by

patients, dries easily, and is preferred. The disposable setups are rigid, difficult to dismantle, and drying requires several days. Also, the disposable setups wear out (become stiffer) rapidly, and replacement can be required every one to three months, making this form of tubing more expensive in the long run. Because certain species of gram-negative bacilli (Pseudomonas Aeruginosa) thrive in a warm moist environment, and because disposable tubings may be "wet" when the patient needs to take a treatment the potential for exposure to this specific gram-negative bacilli is increased. This difficulty with equipment tubing impinges upon the patient who utilizes inhalation equipment at home.

Summary

Patients with lung disease who are using some form of inhalation equipment at home can be exposed to gram-negative bacilli because of the equipment they use. The literature has documented that inhalation equipment and its resultant contamination does contribute to the incidence of nosocomial pulmonary infections with gram-negative bacilli predominating. The present debate and confusion about the efficacy and reliability of the decontaminating procedures used in the home further contribute to the possibility of equipment contamination causing pulmonary infections. Additionally the effectiveness of the vinegar and water "soaking solution" as a decontaminate has not been thoroughly documented. It is well known that patients using inhalation equipment at home share predisposing factors that enhance their susceptibility to pulmonary infections (Valenti, Trudell, & Bentley, 1978). The possibility that inappropriate cleaning can produce contaminated equipment and an increase in pulmonary infections is the primary focus of this study.

Introduction to the Hypothesis

The impetus for this study was to evaluate whether a definite cleaning program (type and frequency) is related to the incidence of contaminated equipment in the home setting.

Hypothesis

The equipment subjected to a planned cleaning program as compared to a normal cleaning program (control group) will have less incidence of contamination.

Definition of Terms

1) Planned cleaning program: Individuals on some form of nebulizer therapy at home will be instructed to clean their equipment using a specific solution and frequency of cleaning during their participation in the study.

2) Normal cleaning program: Individuals on some form of nebulizer therapy at home will be instructed to continue cleaning their equipment as they have been doing and not to make any changes in their cleaning regime.

3) Equipment contamination: Will be based on the cultures from the Aero-Test sampler and contamination will be based on colony counts per plate.

4) Decontamination: All components of the breathing circuit (tubings, exhalation valve, nebulizer, jet, and diaphragm) should be free of vegetative microorganisms.

Chapter Two

I. Theoretical Framework

A. Introduction

Numerous physiological, immunological, and bacteriological factors increase the susceptibility of the COPD patient to pulmonary infection. The respiratory tract is protected by highly complex mechanisms of defense (specific, nonspecific, and local). Susceptibility to infection increases when a change or defect in any of these mechanisms occur. This chapter explores each of these influencing factors and how they relate to the patient with chronic lung disease. The physiological effects of IPPB and compressor powered nebulizers on lung parenchyma and hemodynamic/vascular systems is not discussed.

B. Pathophysiology of COPD

The disease category of chronic obstructive pulmonary disease (COPD) includes emphysema, bronchitis, and asthma or a combination. Each entity is distinguished by its unique pathophysiological mechanisms but all manifest similar symptomology. Allergic (extrinsic) asthma is exemplified by increased sensitivities to inhaled allergens or histamine aerosols (Bates, Macklem, & Christie, 1971). Associated clinical findings are: wheezing, bronchospasm, acute exacerbations, and occasional thoracic deformity with onset frequently occurring in childhood. Intrinsic asthma (non-allergic) is associated with severe bronchospasm; autonomic nervous system imbalance in the airways is considered a possible causative or etiological factor here (Bates, Macklem, & Christie, 1971). Bronchitis is characterized by excessive mucous secretion in the bronchial tree, airway obstruction, and ventilation perfusion defects with further definitive categories according to the mucous secretion and color (degree of chronicity) (Bates, Macklem, & Christie, 1971; Hinshaw & Murray, 1980).

Emphysema denotes architectural changes in the lung parenchyma and is

associated with progressive airway obstruction, ventilation/perfusion defects, changes in the thoracic cage, destruction of the alveolar-capillary membrane, and CO_2 retention and hypoxemia (Hinshaw & Murray, 1980; Murray, 1976). These diseases comprise COPD and share some common symptomologies and morphological changes. Most patients with COPD tend to have co-existing diseases (e.g. bronchitis or emphysema) rather than a "pure" entity (Bates, Macklem, & Christie, 1971; Hinshaw & Murray, 1980). Airway obstruction, ventilation/perfusion secondary to changes in the alveolar - capillary membrane, excessive mucous secretion, and hypoxemia are some of the shared physiological defects that result from chronic lung disease. Architectural and morphological changes resulting from chronic lung disease can increase the patient's susceptibility to pulmonary infection. Immunological and humoral defects in the COPD patient are known to increase the incidence of pulmonary infection (Grieco, 1980; Allen, 1976).

II. The Compromised Host and Pulmonary Infections

A) Introduction

The human body is protected against infection by physiologic, cellular, and immunologic defenses that are able to maintain tissue sterility in the presence of an environment rich in microorganisms (Greico, 1980). A serious defect in any component of these defense mechanisms increases the individual's vulnerability to infection. Defects in the host defense mechanisms such as disorders in the mucosal barrier, phagocytosis, chemotaxis, humoral and cellular immunity, not only cause vulnerability to infection, but also result in a compromised host (Allen, 1976; Grieco, 1980). The skin and mucous membranes of the respiratory tract provide the initial barrier and defense against infections and COPD is known to compromise and limit this essential barrier (Grieco, 1980; Allen, 1976). Furthermore, changes in the humoral and cellular immune defense mechanisms are known to occur with chronic lung disease (MacDonnell & Segal, 1977; Allen,

1976). Therefore, the COPD patient is vulnerable to infections from the usual pathogenic microorganisms that afflict the normally healthy person but, also, from "opportunistic" organisms (Psuedomonas aeruginosa, Candida albicans, and Mycobacteria) (Allen, 1976; Matthay & Greene, 1980).

Adequate functioning of the defense mechanisms are of paramount importance in order for the patient with chronic lung disease to combat infection. Factors that contribute to the pathogenesis of the opportunistic microorganisms are: alterations in the indigenous microfloras of the tracheobronchial tree, changes in the defense mechanisms, and constitutional factors (aging, activity, etc.).

III. Normal Flora of the Respiratory Tract

The indigenous microfloras of the upper respiratory tract are variable with the most common bacteria being Staphylococcus epidermidis, Nesseria species, alpha-Hemolytic streptococci, non-Hemolytic streptococci, and Diptheroids (Allen, 1976; Finegold, Martin, & Scott, 1978). The composition of the normal flora is continuously changing due to the environment of the host or to disease factors. The respiratory tract provides an easy access for transmission of infectious microorganisms. This combination of easy access and immunological defects in the COPD patients (MacDonnell & Segal, 1977) entices opportunistic microorganisms. Whether a particular organism establishes itself as a member of the normal, resident, transient flora, or as a pathogenic organism is dependent upon its relationship with the host and whether immunological defects exist in the host (MacDonnell & Segal, 1977; Allen, 1976). Changes in the regional protective mechanisms determine how these organisms establish themselves within the microflora of the respiratory tract.

IV. Local Defense Mechanism of the Respiratory Tract

The anatomic barriers of the nasopharynx, the mucocilliary transport

mechanisms within the tracheobronchial tree, the alveolar macrophages, and the immunoglobulins protect the lung from invasion by virulent organisms. Initially, the filtering action of the nasopharynx protects the respiratory tract from inhaled particles either by nose blowing or sneezing. The structure of the nasopharynx and tracheobronchial tree (hairs, bifurcating tubes with decreasing caliber) reduces the amount of airborne inoculum that can reach the lung parenchyma (Grieco, 1980, Murray, 1976).

Once inhaled particles or infectious agents reach the posterior aspect of the nasopharynx the sweeping motion of the cilia propels the particles backward over the mucus-lined, ciliated epithelium where they are swallowed. Chemical composition and size of the invading particles influence how far into the respiratory tract they penetrate before impacting (Murray, 1976). Penetration of particles beyond the nasopharynx activates the mucociliary clearance system that protects the entire conducting airways (Murray, 1976). Murray (1976) identifies two major clearance systems (mucociliary transport and macrophage transport) which remove the deposited particles from the lung parenchyma; interdigitation of the two systems is essential for adequate mechanisms of clearance. Distinction between the two clearance systems is by anatomical parameters; the tracheobronchial tree is cleared by the mucociliary transport while the terminal respiratory units are cleared by macrophage transport. Assisting in clearance of the tracheobronchial tree is the sol-gel layer of mucous on the surface epithelium and the bi-phasic stroke of the cilia which propel particles forward.

When solid particles settle on the alveolar surface two responses occur: 1) the particles are phagocytized by the alveolar macrophages where they are eliminated either by digestion or tranported out by the mucociliary system; 2) macrophages carry out phagocytic ingestion (opsonization) but transport is

through the septal areas of the lung (Murray, 1976; Grieco, 1980).

The three types of "professional scavengers" are polymorphonuclear leukocytes, circulating monocytes, and tissue macrophages. The efficiency of these three scavengers prevents the spread of the inhaled particles into the lymphatic system and thus into the blood stream (Murray, 1976; Hinshaw & Murray, 1980). Polymorphonuclear leukocytes (PMN) phagocytize the blood-borne bacteria whereas the alveolar macrophages are chiefly responsible for the phagocytic ingestion of inhaled particles (Murray, 1976; Green et al, 1977).

Changes in any of the local defense mechanisms or in the microflora of the respiratory tract enhance susceptibility of the respiratory tract to colonization by the gram-negative bacilli. Because the COPD patient experiences changes in both of these mechanisms a predilection to pulmonary infections by opportunistic microorganisms occurs.

V. Pharyngeal Flora Changes With COPD

The mucociliary clearance mechanism and the microfloras of the respiratory tract can undergo changes due to disease (COPD), medications (especially antimicrobials), acid-base imbalance (especially acidosis), environmental factors and immunosuppressives Murray, 1976; Johanson, 1977; Greico, 1980). Antimicrobials and corticosteroids are major causes of pharyngeal flora changes in the COPD patient. When these changes occur, the tracheobronchial tree becomes susceptible to colonization by a wide variety of microorganisms (Johanson, Pierce, Sanford, & Thomas, 1972).

Johanson, Pierce, and Sanford's (1969) classic study of changes in pharyngeal flora in hospitalized patients found an increased frequency of colonization of the respiratory tract by gram-negative bacilli. This study provided the basis for other investigations on immunological defects in the chronically ill person. Several studies have confirmed that the more severely ill

the person the greater the impairment of the clearance mechanisms. (Johanson, Pierce, Sanford, & Thomas, 1972; Valenti, Trudell, & Bentley, 1978).

Patients with a primary diagnosis of respiratory disease have significantly higher colonization of the respiratory tract with gram-negative bacilli (Johanson, Pierce, Sanford, & Thomas, 1972). Other contributing factors that facilitate colonization of the respiratory tract by gram-negative bacilli are: coma, hypertension, intubation, azotemia, leukocytosis, leukopenia, decreased level of activity, antibiotic therapy, and corticosteroid usage (Valenti, 1978; Eickhoff, 1980, Johnason, Pierce, Sanford, & Thomas, 1972). The hospital setting promotes greater colonization of the respiratory tract by gram-negative bacilli, and debilitating diseases contribute to this colonization (Johanson, 1977, Allen, 1976).

Once colonization occurs, the organisms multiply and bacterial pneumonias result from aspiration of the oropharyngeal secretions thus causing bacillary pneumonia (Johanson, 1977; Rippon, 1977; Pierce, Edmondson, & Sanford, 1966). Patients with chronic bronchitis experience frequent pulmonary infections because of abnormalities in mucociliary transport and changes in the microflora of the respiratory tract (Grieco, 1980). Furthermore, chronic bronchitis characterized by increased sputum production (change in sputum viscosity), acidosis, and in some a poor cough reflex encourages gram-negative colonization. Colonization is enhanced because of diminished clearance due to areas of squamous metaplasia and uncoordinated ciliary activity (Eichoff, 1980). Lastly, emphysema, bronchitis, and severe asthma may produce architectural changes that prevent adequate clearance of secretions from the affected lobular areas (Hinshaw, & Murray, 1980; MacDonnell & Segal, 1977).

Alveolar macrophages, the resident tissue macrophage of the lung, lie submerged in the extra-cellular lining of the alveolar surface (Murray, 1976). Because they are avid phagocytes, they are able to dispose of a wide variety of

microorganisms. Alveolar macrophages constitute the primary mechanism for clearing bacteria and toxins from the terminal respiratory units (Murray, 1976). Deficiencies in alveolar macrophage function contribute to the pathogenesis of pulmonary infections (Greico, 1980). Johanson and Gould (1977) report hinderance of phagocytosis by alveolar macropahges when a deficiency in the cell-mediated (T-cell) defense mechanism is present. Alveolar macrophage phagocytosis is also decreased because of corticosteroid therapy. It has been suggested that the COPD patient has decreased alveolar macrophage activity because of T-cell deficiencies (Greico, 1980).

Use of possibly contaminated inhalation equipment by this group of individuals further stresses the already compromised host defenses, thereby allowing bacterial proliferations that can result in pneumonia (Greico, 1980; Allen, 1976). Decreased overall efficiency of these local defense mechanisms and changes in the tracheobronchial microflora contribute to the increased incidence of pulmonary infections in the COPD patient (Allen, 1976; Hinshaw & Murray, 1980; Greico, 1980).

VI. Systemic Defense Mechanisms

Two types of defense mechanism have been identified within the human body: nonspecific and specific. Understanding how these two defense mechanisms are activated in the presence of invading microorganisms is essential since the COPD patient is noted to have deficiencies in both (Murray, 1976; Hinshaw & Murray, 1980; Johanson, Pierce, and Sanford, 1969). A brief overview of the nonspecific and specific defense mechanisms will provide a framework for understanding the susceptibility of the COPD patient to infections from the opportunistic microorganisms.

Nonspecific immunity, the first type, results from general processes rather than from processes directed at specific disease organisms (Murray, 1976;

Guyton, 1976). It is frequently referred to as the "natural" immunity since it results from naturally occurring processes within the human body. The nonspecific defenses are composed of clearance mechanisms (nasal, tracheobronchial, and alveolar), secretion mechanisms (mucus lining, surfactant, interferon, lysozyme, and complement), and cellular defenses (nonphagocytic and blood and tissue phagocytes) (Murray, 1976).

Specific or acquired immunity, the second type, is caused by the formation of antibodies and sensitized lymphocytes whose purpose is to attack invading agents (e.g. lethal bacteria, viruses, toxins and foreign substances) (Allen, 1976; Murray, 1976; Hinshaw & Murray, 1980). Specific (immunologic) defense mechanisms are important in fighting organisms that the body lacks natural immunity against. The specific defense mechanisms can be acquired naturally (contract mumps) or artificially (vaccination); immunity results after the initial invasion by a foreign substance or toxin.

Understanding how these two types of defense mechanisms function in the 'normal' healthy individual will explain the significance of defects in the mechanisms which occur in the person with COPD.

A) **Normal Activation**

The nonspecific defense mechanism (the filtering system of the respiratory tract) provides the initial barrier against invasive organisms. A breach in this barrier activates the specific mechanism that results in specific responses. The major role of the specific defense mechanism is to protect the lung parenchyma from invasion by pathogenic organisms.

The specific (immunologic) system is composed of two types of cells which are characterized as B or T-cells. The T-cell lymphocytes, located in the paracortical area of the peripheral lymphoid tissues, are preprocessed by the thymus and respond to an antigentic challenge through a cell-mediated response

(Murray, 1976; Hinshaw & Murray, 1980; Allen, 1976). The sensitized T-cells are known to release lymphokines and to have direct cytotoxicity against certain membrane-bound antigens. The T-cell lymphocytes are referred to as cellular or cell-mediated immunity.

The B-cell lymphocytes, the second type of cells, are believed to be derived from the bone marrow and from plasma cells (Allen, 1976). The prebursal stem cell of the B-cell lymphocyte is considered to have originated from the influence of an unknown analogue of the bursa of Fabricus (Allen, 1976; Murray, 1976; Hinshaw & Murray, 1980). The B-cell lymphocytes differentiate and undergo proliferation into antibody-producing cells called immunoglobulins of which five classes are known (IgM, IgG, IgA, IgD, IgE) (Allen, 1976; Murray, 1976). The immunoglobulins then circulate and result in an antibody-antigen response that sensitizes certain tissues against an invading organism. This antibody-mediated response of the B-cell lymphocytes is generally referred to as humoral immunity.

Collaboration between the two types of cells (B and T) must result for the full expression of immunologic responsiveness (Hinshaw & Murray, 1980; Greico, 1980). When specific immunologic deficits occur, exposure to specific bacterial invasion is facilitated. Patients with defects in the humoral immune system are susceptible to infections from pyogenic gram-positive cocci (staphylococci, pneumococci, and meningococci), enteric gram-negative bacilli, and fungal species (Gallucci & Rebeis, 1979; Matthay and Greene, 1980). Defects in the cell-mediated immune system encourage invasion by tuberculosis, candida, aspergillus, and viral species (Matthay & Greene, 1980). The COPD patient is very susceptible to infections by opportunistic microorganisms because of defects in both of these defense mechanisms.

VII Defense Mechanism Changes With COPD

Patients with COPD have decreased amounts of circulating IgA; as a result,

adequate clearance of the upper respiratory tract becomes an impossibility (Murray, 1976; Greico, 1980). This defect in the humoral system which protects the airways from invasion by specific antigens is a significant loss for the COPD patient. The five antibody classes of immunoglobulins (IgG, IgM, IgA, IgE, IgD) are responsible for the antigen resistance. In particular, the B-cell lymphocytes (humoral) provide protection in the nasal turbinates, the mucociliary system, and the bronchial mucosa due to the presence of salivary IgA (Greico, 1980; Murray, 1976). A deficiency of immunoglobulins, which are part of the pulmonary defense mechanisms, can result in failure of the primary defense systems (nonspecific) to resist the invading microorganisms (Greico, 1980). This failure of the initial defenses activates the polymorphonuclear leukocytes and other mononuclear cells, and elimination of pulmonary pathogens by these leukocytes causes exudation and tissue destruction (Greico, 1980). In reality, the COPD patient may experience not only increased infections but further destruction of his lung parenchyma.

Greico (1980) points out that the host-bacteria relationships are tenuously balanced in the COPD individual and that any additional insult further reduces his marginal resistance to infection. Such an insult is the improper cleaning of inhalation equipment and the resultant contamination of the equipment with gram-negative bacteria. If the gram-negative bacteria can survive and multiply in water (Psuedomonas species, Serratia sp., Herella sp), it can then be aerosolized into the bronchial and alveolar regions of the lung. For the healthy individual, combating infections is essentially an easy process, but for the compromised host with impaired humoral or cellular immunity resisting infections is difficult. The susceptibility of the COPD patient to infections from opportunistic microorganisms because of systemic defense mechanisms defects cannot be overstressed (Allen, 1976; Murray, 1976; Hinshaw & Murray, 1980;

Greico, 1980).

VIII Aging Factors

A) Inactivity

The seriously ill or the hospitalized patient's response to infectious processes is hampered because of his physical inactivity. Use of analgesics, opiates, debilitated physical status, or surgical procedures that limit activity predispose the patient to skin infections from decubti, pulmonary infections from atelectasis, or urinary tract infections (Allen, 1976; MacDonnell & Segal, 1977). Decreased activity and debilitated physical status can predispose the COPD patient to pulmonary infections from the gram-negative bacilli (Johanson, 1979; Greico, 1980; Allen, 1976). The COPD patient is frequently physically debilitated and experiences limitation of activity. These factors, when combined with the defects in the local and systemic defense mechanisms increase susceptibility to infection.

B) Aging

The immunologic aspects of aging in humans has not been extensively researched. A 1974 longitudinal immunological study followed 199 older adults for two years. Delayed-typed hypersensitivity (D.T.H.) cutaneous responses to five antigens were used to assess immunological response (Roberts-Thomson, Whittinham, Younchaiyd, & MacKay, 1974). The study groups consisted of twenty healthy adults under 25 in one group and sixty-eight adults ranging from 60 to 80 years in another group. The term "healthy" implied that participants could have "degenerative" diseases (cerebral vascular, ischemic heart disease, diabetes mellitus, osteoarthritis, and parkinson's) but they had to be well-nourished, ambulatory, free of cancer, and free of immunopathic diseases.

The five antigens introduced intradermally were Candida, mumps, trichophyton, tuberculin (1/1000), and streptokinase (10 units), and streptokinase

(10 units). Injection intradermally of Flagellin and phytohaemagglutin (P.H.A.) was also used to evaluate lymphycyte response. Skin testing has been the traditional method for evaluating defects in the T-cell immune system. Normally the healthy individual will react positively to one or more of the antigens while the immune repressed individual may lack reactivity (Allen, 1976; Murray, 1976). Prior studies have confirmed that D.T.H. responses are depressed with aging (Forbes, 1971). The purpose of this study was to correlate mortality in older people with failure of the cell-mediated system.

In this study, D.T.H. reactions to the five antigens were grouped according to their reactivity (zero to one reaction; two to five reactions) to the antigens used. The younger group had 100% reactivity to two or more of the antigens used compared to 43% reactivity to two or more antigens for the aged. Positive reactivity to intradermal injection to at least 2 out of the 5 antigens is an accepted criteria for the normal host without any immune defects. The positive reactivity (100%) of the younger group was statistically significant ($p = 0.001$). At the end of the 2 years, participants who reacted with less than two positive reactions had a significantly greater mortality ($p = 0.005$) compared to those with more than two positive reactions in the same antigen grouping. This finding suggested that the aged individual does have T-cell depression that indicates a deficiency in the cellular immune mechanism. The patient with COPD is usually older and suffers defects in his defense mechanisms secondary to disease states and aging.

IX Antimicrobials/Corticosteroids

A) Antimicrobials

The effects of oral antibiotics on the normal flora of the tracheobronchial tree have been extensively investigated (Allen, 1976; Sanders, Sanders, & Harrowe, 1976; Johanson & Gould, 1979). Antimicrobials can contribute to gram-

negative colonization of the respiratory tract if certain physical and environmental factors are present (chronic disease, aging, hospitalization) (Valenti, Trudell, & Bentley, 1980; Johanson, Pierce, Sanford, & Thomas, 1972). Large doses of antimicrobials, especially the broad spectrum ones, are noted to suppress the normal flora in healthy individuals (Valenti, Trudell, & Bentley, 1980; Eickhoff, 1980). The lower respiratory tract is essentially sterile and any inhabitation of this site with gram-negative bacilli reflects endogenous changes in the microflora (Johanson, Pierce, & Sanford, 1969; Sanders, Sanders, & Harrowe, 1976).

Sanders, Sanders, and Harrowe (1976) studied the use of Penicillin V.K. and Tetracycline HCL in thirty healthy adults age 19 to 33. Three study groups were formed; group one used Pen V.K., group two used Tetracycline, and group three served as an untreated control group. Testing included throat cultures, identification and quantitation of the normal flora, and interference assays (used for screening throat flora for inhibitory activity against group A streptococci). The purpose of this study was to evaluate how long normal floras remained suppressed after antibiotic therapy. Both group one and two were placed on antibiotics four times a day for seven days. Sanders et. al. found that both Pen V.K. and tetracycline suppressed the normal flora for up to 3 weeks after completion of therapy and that Pen V.K. (penicillin) hindered interference activity against group A streptococci. Phrased differently, penicillin therapy facilitates colonization of the tracheobronchial tree with this specific bacteria (group A streptococci).

This is significant since COPD patients use tetracycline and ampicillin for prophylactic and acute treatment of pulmonary infections (Sanders, Sanders, & Harrowe, 1976). Furthermore, tetracyline may actually cause Candida albicans and Pseudomonas aeruginosa overgrowth (increased colonization) by changing the

microflora of the respiratory tract; both of these antibiotics interfere with lymphocyte activity, chemotaxis, and the clearance mechanisms (Greico, 1980). Antibiotic therapy in combination with the disease process of COPD creates another aspect of susceptibility for this group of patients.

B) **Corticosteroids**

Corticosteroids, which have many side effects, are widely used in treating the patient with chronic lung disease who has an asthmatic or reversible airway component. It has been documented that corticosteroids decrease the number of phagocytes in the blood stream and tissues, impair the function of the remaining phagocytes by limiting their responses to chemotactic stimuli, and thus hindering the ability of phagocytes to destroy the invader (Allen, 1976; Hinshaw & Murray, 1980). Long term use of corticosteroids (10 mg. daily for 1 year) causes T-cell dysfunction (Greico, 1980). The immunosuppressive state created by large doses of corticosteroids facilitates invasion of the pharyngeal flora by the opportunistic fungi (Aspergillus species, Candida sp., Cryptococcus sp., and Mucorates sp.) (Rippon, 1977).

Corticosteroid therapy contributes to the impaired host by limiting host defense mechanisms; it depresses leukocytic bactericidal, humoral, and cellular function, and interferes with the granulocytic response to infection (Matthay & Greene, 1980). As a result the pulmonary defense mechanisms have limited ability to combat infections from the opportunistic microorganisms. Many patients with chronic lung disease are on long term corticosteroid therapy and this potentiates the defects in their defense mechanisms already discussed.

Summary

The patient with COPD experiences defects in all of the body's defense mechanisms, and aging, inactivity, the disease state, antimicrobials, and corticosteroids cause further defects in the defense mechanisms. Also, the fact

that inhalation equipment is a vector for the transmission of gram-negative bacilli contributes to the probability that these patients using equipment at home can become colonized by the opportunistic microorganisms. The great susceptibility these patients have to the development of pulmonary infections validates the importance of examining how clean this equipment is at home and whether contamination of the equipment is, indeed, a problem.

II. Review of Relevant Literature

Introduction

The present literature on the uses and implications of inhalation therapy dates back a decade for these investigative studies. The role of inhalation equipment as a vector for the transmission of gram-negative bacilli has been documented. Examining whether the home based equipment can become contaminated and whether cleaning is important deserves an answer.

A) Contamination of Hospital Equipment

Equipment contamination in the hospital setting and nosocomial pulmonary infections has been widely researched. Whether home inhalation equipment can become readily contaminated in the same manner as hospital equipment is a gray area. Reinarz, Mays, Pierce, & Sanford (1965) sampled respirators with mainstream reservoir nebulizers and without reservoirs in six hospitals (n=100) using the Anderson air sampler to assess whether inhalation therapy equipment can aerosolize viable bacteria and in what amount. The Anderson Air sampler is used to sample effluent air of respirators. It is capable of sorting viable airborne particles into six ranges according to the size of the particles (micron), and the maximum range is 2,500 particles per 7.5 liters of air. For the purpose of their study bacteria were viable if the particle size ranged from 1.4 to 3.5 micron particles per 7.5 L of air. This particle size of bacteria is capable of penetration

beyond the level of ciliated bronchial epithelium (Hoeprich, 1972; Murray, 1976). Their procedures were standardized according to the sampling time, the cycling time, the type of equipment, and the type of tubing. Cultures of three different sources (ambient air, distilled water, normal saline) were taken before, after, and during treatments using the Anderson air sampler. The cultures were incubated on Trypticase soy agar for 48 hours at 37 degrees centigrade.

Reinarz et al (1965) found that 45% of the mainstream reservoir nebulizers generated large numbers of viable bacteria; all of the bacteria generated were gram-negative bacilli (Pseudomonas species, Flavobacteria sp., Herellea sp., Alcaligenes fecalis, and Achromobacter sp.). They acknowledged that inhalation equipment was capable of aerosolization of bacteria and that the gram-negative bacteria could be implicated as a source of nosocomial pulmonary infections.

One problem with their study was that the equipment was cleaned with a 1% phenolic solution, a solution since determined to be ineffective as a decontaminate (Pierce, Sanford, Thomas, & Leonard, 1970; Block, 1977; Hoeprich, 1972). They did provide the impetus for others to evaluate the role of gram-negative bacilli in nosocomial infections and, especially, the role of IPPB therapy as a possible source for transmission of this bacteria.

Pierce and Sanford (1973) examined inhalation equipment as a vector for hospital-acquired pneumonia due to gram-negative bacilli. They reported that IPPB devices are capable of generating bacterial aerosols and that contaminated nebulizing solutions along with pitted jets are sources for inhabitation of gram-negative bacilli. Pseudomonas aeruginosa was found to be the most frequent offender in this hospital study. Furthermore, they reported that inhalation devices placed in a clinical setting will become readily contaminated because of the airborne "hospital flora" (Kelsen, McGuckin, Kelsen, & Cherniack, 1977). Kelsen et. al. (1977) agreed that airborne contamination of fine-particle

nebulizers does occur in the hospital setting. Kelsen et. al. found that in patient areas having a predominance of airborne gram-negative bacteria this bacteria could be entrained by nebulizers and then aerosolized into the tracheobronchial tree. The problem of environmental bacteria being entrained by inhalation devices and then inoculated into the respiratory tract has been validated by other studies (Nazemi, Musher, Martin, 1972: Grieble, Colton, Bird, Toigo, & Griffith, 1979).

It is known that colonization of the upper respiratory tract by "hospital flora" occurs rapidly in the hospitalized patient in the absence of inhalation therapy (Allen, 1976; Matthay & Green, 1980; Johanson & Gould, 1977). Patients using inhalation equipment in the hospital are doubly exposed to gram-negative bacteria and thus are very susceptible to the development of bacterial pneumonias. The hospitalized COPD patient is very susceptible to a gram-negative pulmonary infection. This is especially true if the patient is on inhalation treatments and has deficient defense mechanisms (Greico, 1980).

The prevalence of bacterial pneumonias in the hospitalized, chronically ill patient is associated with the use of inhalation equipment, use of antimicrobials, use of steroids, and changes in the host resistance (Pierce, Edmonson, McGee, Ketchersid, Loudon, Sanford, 1966; Johanson & Gould, 1977). The fact that IPPB devices are noted to become easily contaminated, both by entrainment of air and nebulizing solutions, increases the probability of inoculation of gram-negative bacilli into the airways.

These gram-negative bacilli (Escherichia coli, Klebsiella species, Pseudomonas sp., Enterobacter sp., Proteus sp., and Serratia marcescens) have been implicated as causing bacterial pneumonias in persons both within the hospital and in the community (Matthay & Green, 1980). The hospital setting is an ideal environment for growth of gram-negative bacilli whereas the home

setting facilitates growth of gram-negative bacilli to a lesser degree (Matthay & Greene, 1980; Hyde, Moore, and Higgins, 1979). Inhalation devices with reservoir nebulizers are sources for the transmission of gram-negative bacteria in the hospital setting (Grieble, Colton, Bird, Toigo, Griffith, 1970). Patients who use the same devices in the home supposedly are exposed to the same contaminants. However, this point has not been validated by research.

B) **Contamination of Water**

Numerous studies have confirmed that contamination of aerosols can result when either the medication (bronchodilator) or the water (nebulizing solution) introduced into the nebulizer is contaminated (Pierce, Sanford, Thomas, Leonard, 1970; Hoeprich, 1972; Koss, Conine, Eitzen, Losasso, 1979). All three of the nebulizing solutions (distilled, purified, & sterile water) have been noted to become contaminated with gram-negative bacilli (Peterson, Carson, Favero, Marshall, & Bond, 1975; Dumas, 1972). It has been thoroughly documented (Avery, 1980; Dumas, 1972; Peterson et colleagues, 1975) that distilled water encourages growth and multiplication of gram-negative bacteria. Distilled water is not the only source that has been implicated as a medium for the growth of gram-negative bacilli. Pseudomonas aeruginosa isolates were found growing in tap water of humidifiers in the hospital based study by Grieble, Colton, Bird, Toigo, and Griffith, 1970). Contaminated aerosols are also known to cause bacterial colonization of the airway, nosocomial pneumonia, and gram-negative sepsis (Kelsen, McGuckin, Kelsen, & Cherniack, 1977; Johanson, Pierce, Sanford, Thomas, 1972; Eickhoff, 1980).

All of these studies stressed that specific handling of the solutions and equipment, inadequate cleaning and decontamination of the equipment, and possible environmental air contamination were variables for contamination of the water sources. Even though all of these studies were hospital based some

similarities exist in the home situation, and the potential for contamination of equipment with gram-negative bacilli seems quite significant at this time.

Changing the water sources may not be the solution since other variables have been implicated, and there is the reality that distilled water remains an inexpensive, readily available, and easy to handle solution for the patient at home. The fact that contamination of the nebulizing solution can occur has been documented. Improper cleaning and environmental contamination has been implicated as the primary cause. Tap water has the potential to be less contaminated with gram-negative bacilli because it is drawn directly from the faucet rather than a storage container. It has been shown that tap water with chlorine hinders the growth of some gram-negative bacilli (Avery, 1980). It should also be pointed out that tap water can become contaminated easily especially if the faucet has been contaminated or if the environment is "dirty". This particular study did not examine water (nebulizing solution) contamination, however, the investigator acknowledges its existence and it would be an interesting study but beyond the scope of this paper.

C) **Equipment at Home**

The only study that examined contamination of inhalation equipment in the home setting was conducted by Hyde, Moore, and Higgins (1979). The purpose of their survey was to ascertain if fungi (Aspergillus species, Candida sp., Cryptococcus sp.,) could be implicated as potential contaminants of IPPB equipment. Aspergillus, Candida, and Crytococcus are recognized as pathogenic opportunistic microorganisms; this bacteria is capable of invading the depressed host (Matthay & Greene, 1980). COPD patients treated with corticosteroids and antibiotics are especially susceptible to fungal infections (Matthay and Greene, 1980). Saprophytic fungi can act either as an allergen or as an infectious agent.

Hyde et. al., compared a hospital based group of COPD patients on IPPB (n=138) with a similar group on IPPB at home (n=30). Airstream sampling using

the Nalgen Powder Funnel was used to culture the equipment. They implemented one, two, and five minute exposure times with effluent impact on a Sabouraud's dextrose agar plate. Sterile saline (3 ml) was the nebulizing solution for the hospital group. However, the nebulizing solution used in the home was whatever the individual patients were presently using.

In the home, four reservoir and twenty-six IPPB machines were tested using the same sampling technique. Contamination was read by colony counts with class 2 (6-20 colonies) reflective of contamination, while both class 3 (21-100 colonies) and class 4 (greater than 100 colonies) were indicative of gross contamination. Bacteria was broadly classified by gram-stain morphology and specie identification was not attempted.

Sixty-four percent of the hospital samples showed no bacterial growth compared to ten percent of the home samples. Eight (26.7%) of the home samples were in the class 4 degree of contamination while only one hospital unit (0.7%) fell into this category. Fifteen home units (50%) were found to have growth in class 3 and 4 which was defined as "significant contamination". In comparison, only 3.6% of the hospital units feel into this degree-of-contamination category. Heavy fungi contamination of six (20%) home units with yeast and yeast-like organisms was predominant.

The concern that home inhalation equipment can become readily contaminated is validated by Hyde et. al.'s findings. Gram-negative bacilli dominated the fungi as the major bacteria isolated from the home equipment. Hyde et. al. suggested that because the hospital equipment showed little contamination the sterilization procedure used in the hospital was adequate. Because of the greater contamination of home equipment, they questioned the efficacy of the cleaning procedure used at home. They proposed that future studies should explore the cleaning of inhalation equipment at home.

There are several constraints surrounding this study. First, there is a difference in the type of airflora between the two environments and very little information is available about the airflora of the home environment. Secondly, Hyde et. al. did not state how the equipment at home was cleaned but indicated that acetic acid rinse was used. Other studies have confirmed that certain fungi (Candida) and certain gram-negative bacteria (Pseudomonas) actually thrive on and prefer an acid environment (Block, 1977; Hoeprich, 1972; Koss, Conine, Eitzen, & LoSasso, 1979). Thirdly, the patient's own nebulizing solutions were used for the home samples. Whether the nebulizing solutions were obtained from large containers or small bottles in the home was not stated. Contamination of large containers (distilled water) of nebulizing solution can occur easily and has been reported as a source of contamination (Avery, 1980). The exact type of solutions that were nebulized in the home samples was not reported. Various water sources (distilled, purified and tap) available for nebulization have all been implicated as encouraging growth of gram-negative bacilli (Lefcoe & Patterson, 1973; Dumas, 1972; Koss, Conine, Eitzen, & LoSasso, 1979; Peterson, Carson, Favero, Marshall, & Bond, 1975).It is unclear whether the contamination represented in these home samples reflect contamination of the water source, contamination of air, or inadequate cleaning procedures. Clarification of the actual source of contamination in the home setting would have provided extremely beneficial results. This study does leave many questions unanswered, but the acknowledgement that contamination of home equipment does occur is its major strength.

D) **Current Studies**

A study conducted in the San Francisco bay area examined the total home environment (airflora) and its' relationship to contamination of home based inhalation equipment (Avery, 1980). Effluent airstream sampling of IPPB and

compressor powered nebulizers using the Aero-Test Sampler were performed. Other aspects of the home environment that were sampled were: ambient air, medication (bronchodilator), nebulizing solution, sputum, throat culture, and general water supply. This was not a controlled study and the purpose was to assess what types of contamination existed in the home environment of patients on inhalation equipment. The results from this study will not be published until the fall of 1981. The investigator has reported finding contamination in the majority of the home equipment (n=25). Gram-negative bacilli and water-pigmented bacteria were the primary isolates obtained. Conclusions about the source of contamination are not available, but the results could provide new insights into home equipment contamination. The major drawbacks to this study are (a) the lack of control and (2) the fact that cleaning of the equipment was not included as a possible variable.

Summary

Inhalation equipment contamination with gram-negative bacilli has been documented by the literature as occurring in the hospital and at home. Implicated as possible causes for contamination are use of contaminated nebulizing solutions and bronchodilators, poor cleaning methods, and neglectful handling of the equipment.

E) **Decontamination**

Acetic acid solution (white vinegar and water) is the primary mode of decontaminating inhalation equipment at home. However, there has not been any substantive research to validate its effectiveness as a decontaminant for home inhalation equipment.

Decontamination procedures and, in particular, disinfecting solutions that provide bactericidal action were explored by Parker and Hoeprich (1962). The purpose of their study was to find an effective means of preventing urinary tract

infections caused by indwelling catheters. This study is applicable since nosocomial bladder infections are considered to be analogous to nosocomial pulmonary infections. Analysis of bladder irrigation (in vitro) was done comparing acetic acid (0.25%) in distilled water, sodium acetate buffer (pH 5.0), acetic acid (0.25%) in sodium acetate buffer, triclobisonium chloride in sodium acetate buffer (pH 5.0), chlorhexidine diacetate in sodium acetate buffer (pH 5.0), and chlorhexidine digluconate in sodium acetate buffer (pH 5.0).

These solutions were tested for bactericidal activity by in vitro intermittent and continuous bladder irrigations. How the samples were obtained from the different irrigating solutions was not explained. They reported that out of the 149 isolates cultured (gram-positive and gram-negative bacteria) acetic acid in acetate buffer killed 66% of all the isolates. Acetic acid in distilled water was reported to have killed 49 (33%) out of the 149 isolates. The non-buffered acetic acid, with a lower pH (3.0), was considered less bactericidal and therefore less effective as a decontaminant. They concluded that acetic acid in distilled water had limited bactericidal action against some of the normal bacterial floras that can become pathogenic (Klebsiella species, E. Coli, Pseudomonas aeruginosa) (Grieble, Colton, Bird, Toigo, & Griffith, 1970; Block, 1977).

The major problem with this study is the failure of the researchers to identify how they collected the samples, and whether there was any difference between continuous or intermittent irrigations. The actual applicability of these findings to use of acetic acid at home may be inappropriate.

Another study evaluating acetic acid as a decontaminating solution was conducted by Pierce and Sanford (1973). Pierce and Sanford studied acetic acid because it is the most widely used and extensively reported method for cleaning in-use Venturi reservoir nebulizers. They reported that the driving gases of the

Venturi nebulizers are unlikely sources of potential contamination. However, this point has been proven inaccurate by other studies (Block, 1977; Litsky, Botko, and Litsky, 1975).Pierce and Sanford examined decontamination of inhalation equipment because the small-volume Venturi nebulizers (IPPB) are capable of inoculating bacteria into the respiratory tree. Various methods of decontamination have been suggested (ethylene oxide sterilization, glutaraldehyde, and 1% phenolic solution) and disputed in their ability to actively decontaminate or sterilize equipment (Block, 1977; Hoeprich, 1972; Grieble, Colton, Bird, Toigo, & Griffith, 1970). As a result, acetic acid 0.25% nebulizing solution (10 minutes) became popular while its effectiveness remained questionable (Block, 1977; Grieble, Colton, Bird, Toigo, Griffith, 1970).In their study, Pierce and Sanford (1973) nebulized in a laboratory setting 0.25% acetic acid for 10 minutes and this solution was extremely effective as a decontaminate (tested with air sampler). However, when it was introduced into the hospital setting as a nebulizing decontaminate the results were variable. They reported less than 10% contamination when the disinfection was coupled with close surveillance of equipment, surveillance of conditions, and surveillance of personnel.

Grieble et colleagues (1970) also evaluated the use of acetic acid 0.25% and 2% phenolic solution as decontaminates for inhalation equipment with differing results. They examined the use of these solutions for decontaminating humidifiers which are functionally similar to IPPB. Both the acetic acid 0.25% and the 2% phenolic solution were aerosolized for 15 minutes through the humidifiers. The 2% phenolic solution actually increased the bacterial count of Pseudomonas aeruginosa while the acetic acid was found to contain an anti-pseudomonas effect. They found acetic acid initially decontaminated the reservoirs but other parts of the setup (tubing) remained contaminated and

transmitted bacteria (Pseudomonas a.) as early as 15 minutes after the decontaminating procedure. Furthermore, they reported "Pseudomonas can survive 24-hour exposure to 0.25% acetic acid of pH 3, and Serratia marcescens resisted 30 minutes nebulization of 0.25% acetic acid" (p. 534). Grieble et. al. indicated 0.25% acetic acid (nebulizing solution) was ineffective in controlling Pseudomonas and found the most effective alternative was daily sterilization by ethylene oxide. The effectiveness of ethylene oxide in eradicating Pseudomonas was not clearly stated other than that it proved to be the best. This is not a realistic method for home patients because they do not have autoclaves available and because it is too costly.

Sykes (1970) suggested that placing a copper sponge in the reservoir of inhalation equipment would maintain sterility, but erratic results have occurred (Harris, Richards, & Blake, 1973). Other studies report that changing tubing or using fresh tubing every 24 hours will reduce contamination. Most hospitals are presently changing tubings every 24 hours with reduction in contamination. The home patient cannot afford to change or have fresh tubing every day because it is quite expensive.

The American Thoracic Society (1968) recommended using 2 parts of white vinegar to 3 parts sterile water to decontaminate home equipment. It did not suggest a frequency for cleaning the equipment and neglected to consider that most patients cannot afford sterile water to dilute the vinegar. At this point in time, the effectiveness of vinegar and water as a decontaminant is questionable. The additional fact surrounding the efficacy of this solution is that certain fungi and gram-negative bacilli are refractory to its bactericidal activity. Furthermore, water sources as diluents remain a concern because they can become readily contaminated and contribute to the overall problem of equipment contamination. The cleaning procedure in the home is of major importance in

preventing equipment contamination, and the efficacy of the procedure is uncertain.

All of these studies share the common fact that cleaning and maintaining this equipment is difficult. Inappropriate cleaning definitely increases the risk of patients developing pulmonary infections. Meticulous handling of the equipment, decontaminating every 24 hours, maintaining sterile medications, and providing bacteria-free nebulizing water are essential to prevent equipment contamination. None of the studies examined acetic acid as a "soaking solution" which is how it is used in the home. Extrapolation that the bactericidal effectiveness of acetic acid as a nebulizing solution is the same as a soaking solution is rather inappropriate. Until the efficacy of a vinegar and water soaking solution is clarified the concern about the possible contamination of home inhalation equipment will persist.

Conclusion

It is accepted that inhalation equipment can be readily contaminated with gram-negative bacilli (Johanson, 1978; Block, 1977; Pierce and Sanford, 1973). Also, patients with COPD have changes in their defense mechanisms that predispose them to pulmonary infections by opportunistic microorganisms (Allen, 1976; Johanson, 1979; Matthay and Greene, 1980; Greico, 1980). Other factors facilitating colonization of the respiratory tract with gram-negative bacilli include: severity of illness, inactivity, aging, smoking, antimicrobials, corticosteroids, and lung parenchyma changes (Hinshaw & Murray, 1976; Greico, 1980; Valenti, Trudell & Bentley, 1978).

Further compounding the susceptibility of the COPD patient to gram-negative pulmonary infections is the ineffectiveness of the decontaminating solution against bacteria that frequently colonize the respiratory mucosa. Validating vinegar and water soakingsolution as an effective decontaminant has

Chapter Three

III. Methodology

A. Introduction

In order to examine how patients clean their equipment at home, the investigator felt it was essential to examine how patients presently cleaned their equipment in comparison to a group of patients using a specific, standardized cleaning program. It was assumed this type of comparison would provide practical information on the problems existing around cleaning of the inhalation equipment at home and whether any of these problems could be resolved with a detailed, clearly outlined cleaning program for this equipment. As a result of this criteria, the investigator selected a quasi-experimental design for this study.

B. Research Method

The design of this study was quasi-experimental and consisted of two groups of COPD patients who were on some form of nebulizer therapy (IPPB or compressor powered nebulizer) in the home setting. Each patient was randomly placed, by the toss of a coin (heads = control/tails = experimental) into either the control or experimental group. The untreated group design with pretest and posttest (Cook & Campbell, 1979; Campbell & Stanley, 1963) was used:

0_1	X	0_2
0_1		0_2

0_1 = pretest
0_2 = posttest
X = teaching/treatment

Prior to enrolling patients into the study permission from their attending physicians was obtained allowing the patients to participate fully. The three physicians were thoroughly informed of the protocols prior to implementation of the study and their overall permission was granted to the investigator.

A telephone contact was made before the initial visit and participants were informed about the study and their responsibilities in the study. The telephone interview was used for screening purposes to ensure that patients were on some

form of IPPB or compressor powered nebulizer and that they were willing and able to clean their own equipment. Several patients admitted to having these devices in the home when in reality they possessed other devices. The telephone interview also allowed the participants to make the decision voluntarily to join or not to join the study.

During the first visit, in the home, participants were informed that a demographic profile would be obtained along with their vital signs, lung and heart sounds, and a symptom questionnaire. These tests would be re-administered at the end of the study period. Subjects were queried about their cleaning methods used in the home. Subjects received a log on which they were to record how and when they cleaned their equipment and which would be returned at the end of the study. Lastly, they were informed that their equipment would be cultured at two sites at the end of their participation.

Data collection occurred from August, 1980 to March, 1981. Subjects were placed into the control or experimental group during the initial visit. Prior to the visit, the investigator tossed a coin (heads = control/tails = experimental) and placement was made accordingly. Only one participant refused the original placement into the experimental group since she felt it was extra work and was re-assigned into the control group. All participants were informed that their involvement in the study would last 10 to 14 days.

A patient profile was obtained during the initial visit which included: disease history, smoking history, medications (antimicrobials, corticosteroids, bronchodilators), type of nebulizing solution, method of cleaning including frequency, type of tubing (disposable or permanent), and length of time the equipment had been used in the home. (see Appendix A for form).

A symptom questionnaire was completed during the first visit, and subjects were asked to rank their symptomologies. They were asked to rank their

symptoms a second time at the conclusion of their involvement. All were informed that only one ranking was desired, but if they absolutely had to they could make a second choice due to extensive symptoms. A number of the less stable subjects chose a second selection of symptom rank and it was marked accordingly. (see Appendix A for form). Ausucultation of their lungs and heart sounds occurred during the initial and closing visits and was recorded. This questionnaire also assessed such symptoms as: cough, sputum, shortness of breath, wheezing, fluid retention. (see Appendix for form).

Members of the control group were asked to stay with their present pulmonary regime and to make no changes in their present cleaning regime. These subjects received at least one telephone call 2 to 6 days after the initial home visit. Usually a specific day was selected by the investigator and the participant; this call was used to clarify any questions, problems, difficulties, or concerns that may have arisen since the initial visit. Furthermore, it allowed the investigator an opportunity to ascertain whether the participant was keeping a record of the cleaning method being used.

Members of the experimental group were asked during the initial home visit to clean their eqiupment in a specific manner. They received the necessary supplies to ensure that they could adequately perform the cleaning procedure. Each participant was verbally instructed in the cleaning procedure and a demonstration was provided. Each participant received a written copy of the cleaning instructions to keep in the home to refer to when performing the procedure. Questions about the procedure were sought before the end of the first home visit to prevent confusion. It was emphasized to these subjects that it was very important to the success of the study that they maintain and adhere to the instructions for cleaning and that they record in the log this procedure. Three of the subjects in the experimental group received a second home visit (2 days later) after the initial visit because they were having difficulty in

understanding the procedure and in dismantling the equipment. All of the other subjects, including these three, received one or more telephone interviews 2 to 6 days after the initial visit. The purpose of the call was to provide a time for the participants to clarify questions or difficulties and for the investigator to assess their compliance.

Participants were informed that two groups existed and that a difference between these two groups existed. Limited information was given to prevent bias on the part of the participants. The participants were informed that the purpose of the study was to look at how home patients with inhalation equipment cleaned their equipment. A statement was made to the effect that cleaning of this equipment was known to be time consuming and difficult and that the study hoped to find a better way to clean the equipment. The control group was offered the opportunity to have the teaching/cleaning program at the conclusion of their participation and all chose to have the program. Lastly, participants were informed that a copy of the results from the study would be in their physician's office and available for them to read.

Those participants that had cultures reflective of contamination were so informed along with their physicians.

C. Research Setting

Collection of the data occurred in the individual participants' homes or apartments. The homes and apartments were located in the East Bay area of San Francisco and included: Castro Valley, San Ramon, Emeryville, Oakland, Piedmont, and Montclair. Patients were followed by their private physician (whose practices are in Oakland) if they had any medical difficulties during their involvement in the study.

D. Sample

1. Human subjects' assurance

Patients were informed of the purpose and the risks of the study prior to entrance into the study. They were informed that participation was voluntary and that they could withdraw form the study at any time. An informed consent (Appendix B) was obtained prior to enrollment into the study. Since invasive techniques were not performed, no emergency precautions were necessary. If a subject was found to be in respiratory distress or exhibited any difficulties, his physician was immediately contacted. Participants in this study were not compensated in any manner.

2. Nature and size

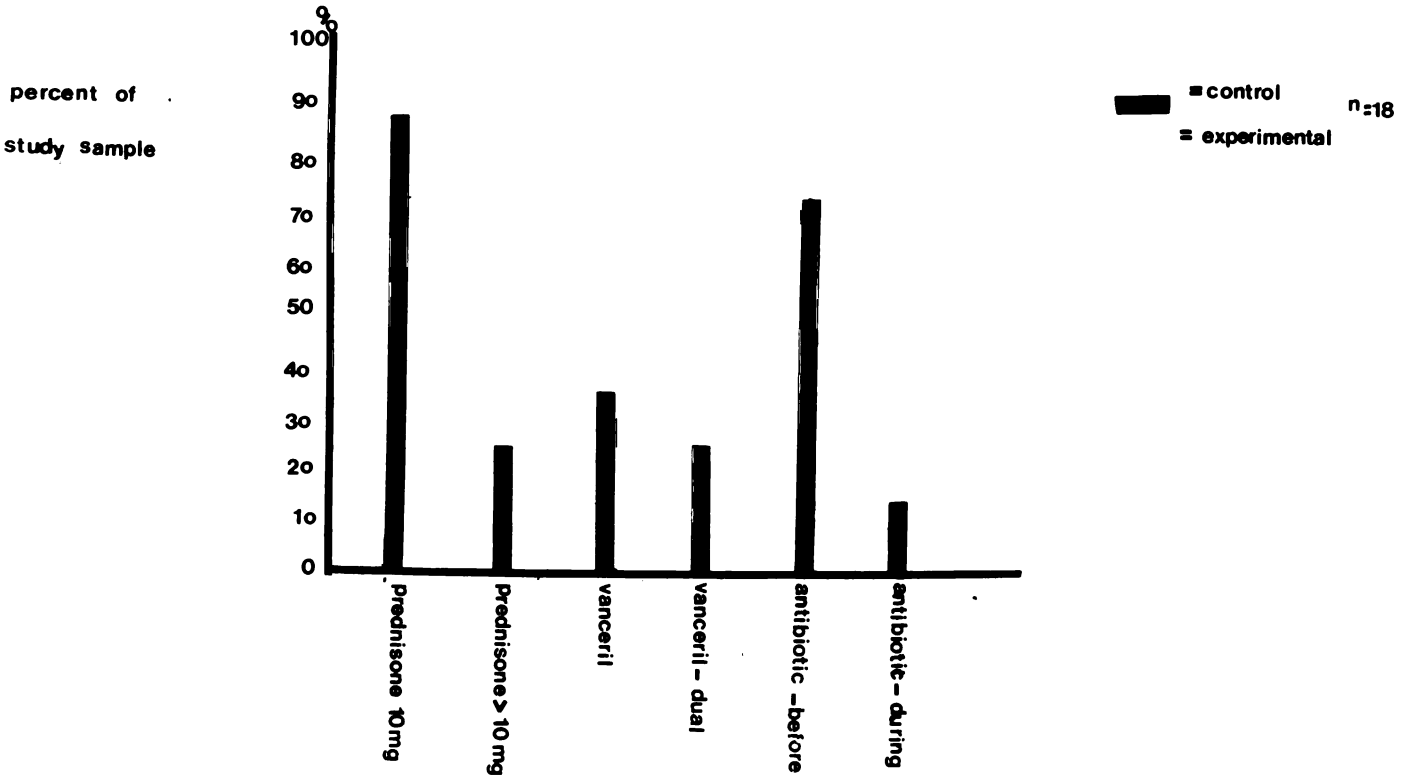
The study sample included eighteen adults ranging in age from 40 to 82 years with the mean age of the sample 66.1 years. The total sample consisted of eleven females whose mean age was 68.7 years and seven males whose mean age was 64.45 years. All of the participants except for two had multiple diagnoses of emphysema, bronchitis, and asthma. Sixteen participants had multiple organ disease and only two participants had primary pulmonary disease (see Appendix B for details). Those who shared the diagnosis of COPD had a mean length of 6.73 years for females compared to 10.64 years for the males. Of the toal study sample 44.4% had been diagnosed as having COPD for at least eight years compared to 22.2% with a diagnosis of less than three years. One subject had been diagnosed as having pulmonary disease for over twenty-five years.

All of the participants were selected from the practice of three physicians; two of the physicians share a practice. Both practices were located in Oakland, California and the physicians were affiliated at the same hospitals. Because patients with COPD are noted to have frequent exacerbations of their disease, the investigator chose to solicit information that might reflect degree of chronicity, frequency of exacerbations, or instability. Therefore, patients were

asked specific information about antibiotics, steroids, and hospitalizations. Out of the total sample study of eighteen participants sixteen (89%) used some form of oral or inhaled steroids on a daily basis; most had done so for at least 2 years (Table 1). A dual usage of oral steroids and inhaled steroids was seen in 6 (34%) participants. Use of prophylactic antibiotics (tetracycline and ampicillin) occurred in thirteen (72%) of the patients during the course of the study (Table 1). Seven (54%) of the thirteen who used prophylactic antibiotics experienced changes in their sputum during the course of the study and were started on antibiotics.

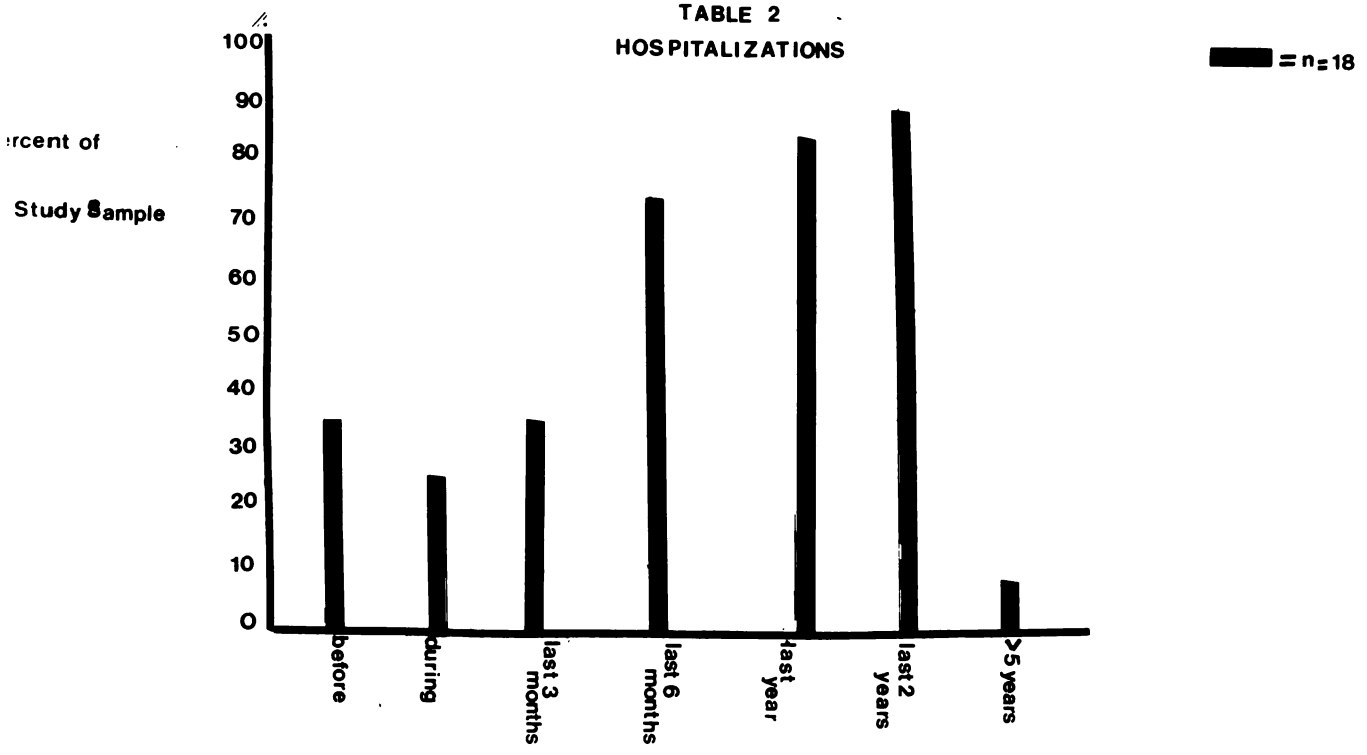
TABLE 1

CORTICOSTEROID & ANTIMICROBIAL USE



Frequent hospitalizations of the subjects due to respiratory complications occurred during, before, and after conclusion of this study. Due to the complexity of this issue refer to Table 2.

TABLE 2
HOSPITALIZATIONS



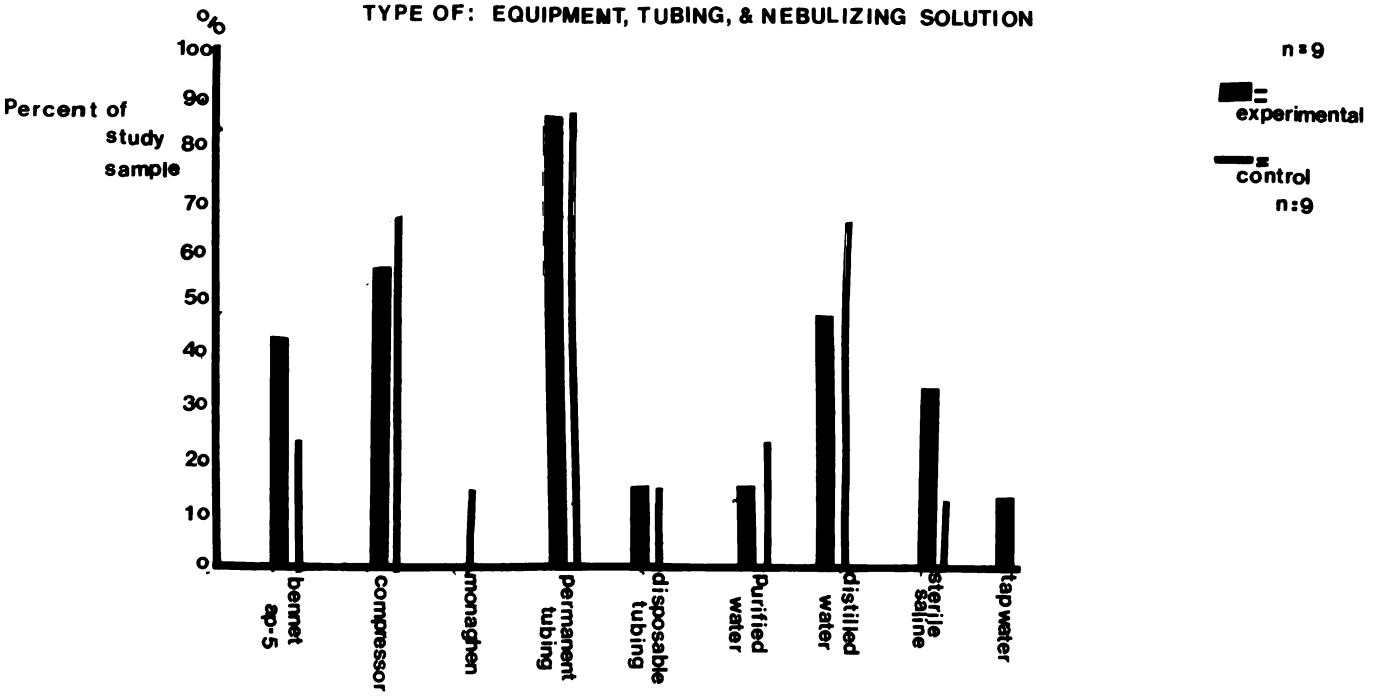
The pulmonary instability of the entire study sample was a significant problem. Only one subject (.05%) out of the total sample remained stable and her last hospitalization was five years ago. Within the experimental group two (22%) out of the nine subjects were hospitalized due to respiratory complications during their study participation; they later rejoined the study after discharge from the hospital. One of those two subjects was hospitalized a second time before he was able to complete the study. Out of the total of eighteen participants sixteen (89%) have been hospitalized in the last two years, and thirteen (81%) out of the sixteen have been hospitalized in the past year due to respiratory complications. This group of patients exemplified many of the problems associated with chronic pulmonary disease.

Oxygen use in the home was also examined since it indicates chronicity and end stage pulmonary disease. Of the total sample, six (33%) of the eighteen subjects used either continuous or hourly (more than 12 hours daily) oxygen therapy in the home. For those who used oxygen therapy at home, most had been using it for over two years; one subject had used continuous oxygen for eight years. Cleaning and maintaining of the oxygen equipment was not examined in this study.

However, whether a particular subject used an IPPB or compressor powered nebulizer was examined in this study. The Bennett AP-5 (IPPB) was utilized by six (33%) out of the eighteen subjects, and only one subject used a Monaghen. Of the total sample study, 61% used a compressor powered nebulizer. The majority (89%) of the subjects had permanent tubing and only two (11%) used the disposable form of respirator tubing. Distilled water was the preferred nebulizing solution (10 out of 18) and sterile saline was used by four of the subjects. Purified water was the choice solution for three of the subjects and only one subject used tap water to nebulize with the bronchodilator. Table 3 illustrates this data according to experimental and control group.

TABLE 3

TYPE OF: EQUIPMENT, TUBING, & NEBULIZING SOLUTION



3. Criteria for Sample Selection

1. Acceptance criteria

To be enrolled into the study participants had to be on inhalation equipment (IPPB or compressor powered nebulizer) in the home for at least 3 months; be responsible for cleaning his/her own equipment; consent to be a participant in the study; and have a diagnosis of emphysema, bronchitis, asthma or a combination thereof.

2. Rejection criteria

Those subjects who refused to participate or who did not clean their equipment were excluded.

E. Techniques for Data Collection

1. Protocols

Experimental Group: The experimental group which consisted of nine participants (three females and six males) received a teaching/cleaning program during the initial home visit. The teaching/cleaning program was the treatment and the exact information provided to the group is presented in table 4.

Table 4

A) After every treatment, thoroughly rinse the mouthpiece and nebulizer in warm, clean water. Reassemble.
B) Every evening, dismantle the manifold, plug the diaphragem, place parts into a solution of Joy and water. Wash thoroughly, rinse in tepid water. Clean jet.
C) Place pieces on a clean, lint-free cloth, cover lightly till dry. (Do not dry pieces and do not wash tubing).
D) Twice a week, after performing B, place the pieces and the 3 tubings or 1 tubing into a solution of 2 parts white* vinegar and 3 parts of water. Soak for 30 minutes.
E) Rinse the parts in tepid water, shake out excess water, and hang the tubings up to dry. (Small tubing from compressor may be blown out by compressor outlet).
F) Perform C as above. Store dry equipment in a clean, plastic bag and seal until next use.

*(The American Thoracic Society recommends this dilution for white vinegar and water in the home)

These cleaning instructions were new to all of the participants. All of the participants were cleaning their equipment, but their procedures varied and excluded some of the aspects emphasized in this cleaning/teaching program. For example, some did not use vinegar and water as a soak, some used weak dilutions of vinegar, and some did not dismantle their equipment in order to clean it. A complete outline of the different cleaning methods is explained in Appendix B.

Participants were instructed how to perform the cleaning/treatment program and provided a demonstration. Written instructions (teaching content) were given to each experimental participant and left in the home; the exact amounts of vinegar and water to use were written down for those who needed this information (number of cups to use per each solution). Each participant recieved a jar of white vinegar and a bottle of Joy detergent in order to assist and foster compliance. They were asked to keep a written log of their daily cleaning activities (a log dated with timed cleaning periods was left in the home). It was emphasized that keeping an accurate log would help the investigator know if they were following the cleaning instructions appropriately. If, for some reason, they had difficulty in dismantling their equipment and re-assembling it they were asked to call the investigator. On several occasions re-visits were made to evaluate the cleaning procedure for those participants who expressed difficulty in managing this new technique.

Control group: The participants in this group were asked to continue their equipment cleaning in the same manner as before enrollment into the study. The control group was comprised of nine adults which included eight females and one male. At the end of their participation all members of the control group were instructed in the teaching/cleaning program.

2. Instruments

The independent variable was the cleaning program offered the experimental group as a teaching program. The dependent variables were the symptoms recorded on the questionnaire and the equipment cultures (Aero-Test Sampler). Because of the noted confusion about the cleaning of this inhalation equipment (Pierce and Sanford, 1973; Block, 1977), the investigator felt that developing a detailed, clearly outlined cleaning/teaching program would provide information on the efficacy of presently used cleaning procedures. Specifying an exact, standardized amount of vinegar and water to use with the decontamination procedure was viewed as a method of evaluating the effectiveness of acetic acid and water as a soaking solution. The exact time frequency for cleaning was standardized in the experimental group. This was done to assess whether cleaning frequency was another possible variable influencing equipment contamination.

The symptom questionnaire that participants were asked to rank by themselves was used for evaluating any change in their physical status (exacerbation or infection) during the course of the study. This questionnaire was selected because certain changes in physical symptoms have been correlated with contamination of inhalation equipment (Johanson, Pierce, Sanford, and Thomas, 1972; Pierce and Sanford, 1973). The Aero-Test sampler was utilized for the primary purpose of evaluating the efficacy of the cleaning/teaching program and whether any contamination of equipment was present.

A) Collection of Cultures

The dependent variable was the Aero-Test Sampler (Olympic Medical Corp., Seattle, Wash.) which was used to culture the nebulizer and the tubing of all the participant's equipment. This sampler is a complete unit consisting of a 60 mm petri dish designed so that the lid and plate fit either end of the sampling cylinder (Figure 1). Culture samples were taken by inserting the respirator

effluent tubing into the Aero-Test truncated funnel with direct impact of flow onto the Trypticase Soy Agar plate (Figure 2). The reservoir nebulizer and the effluent tubing were both sampled. Collection of the sample from the nebulizer required directing the nebulizer mouthpiece into the Aero-Test truncated funnel and obstructing this airway to ensure direct impact of the flow onto the Trypticase Soy Agar plate (Figure 3). Twelve of the cultures were collected in the morning and six were collected in the afternoon.

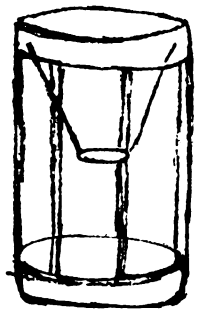


figure 1

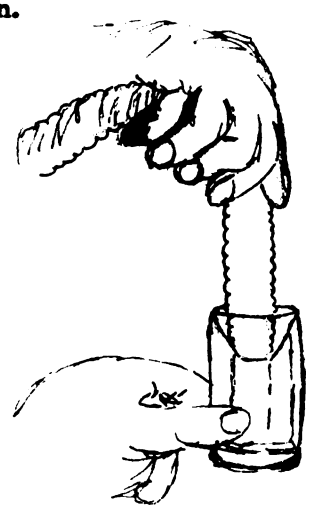


figure 2 & 3

B) <u>Collection Process</u>

If the subject had just taken a treatment prior to the visit for collection of the culture, the investigator thoroughly rinsed out the nebulizer with tap water. This was done in an attempt to prevent any of the subject's own bronchodilator or nebulizing solution from getting into the culture thus invalidating the study results. The IPPB units were set at maximum pressure aerosol production with the nebulizer control turned fully on. They were cycled for 15 cycles or 10 seconds before the culture was collected. The 10 second cycling prior to collection of the culture was recommended by the manufacturer. Maximum

pressure on the Bennett AP-5 was obtained by turning the pressure gauge all the way up; the pressure was re-adjusted to the prior level after the culture was obtained. The compressor powered nebulizers were set at the highest level of sensitivity for maximum output of effluent flow. The Monaghen was cultured using the same technique as for the Bennett AP-5's.

A ten second cycling time was used in obtaining all of the culture samples. Although preliminary studies demonstrated that cycling times of 20 to 30 seconds and 30 to 45 seconds increase the number of bacteria detected, the shorter cycling time was chosen based on the manufacturer's guidelines.

The tubing cultures were obtained first (10 second sample) followed by sampling of the nebulizers. The nebulizers were emptied and rinsed out with tap water; 3 ml. of non-bacteriostatic saline were placed into the nebulizer. After the initial 10 second cycling, another 10 second cycling was performed and sample was obtained. Immediately following the culturing, the plate and funnel were separated (sampling cyclinder) and the plate was covered with the lid. The culture plates were incubated at 37 degrees centigrade for 48 hours. At the end of 48 hours culture plates were read by colony counts and colony selection was performed.

C) **Colony Selection**

Colony selection occurred at the end of the 48 hour incubation. The degree of contamination was based on the guidelines provided by the manufacturer (Olympic Medical Corp.) and are considered standard parameters (Ryan & Mihalyi, 1977). All of the colonies selected were identified by a microbiologist at the conclusion of the study. The procedure was:

1. Collection of the cultures at the end of 10 to 14 days after the subject was enrolled into the study.
2. Plates were incubated at 37 degrees centigrade for 48 hours and all plates were read at the end of that time.

3. Colonies selected were placed into a special extract agar tube which was sealed and incubated at room temperature until the conclusion of the study.
4. Contamination was based on colony counts and read accordingly. (Figures 4 and 5).

Table 5

Contamination

	24 hours	48 hours
0-5 colonies	contamination unlikely, incubate additional 24 hours	no contamination
6-40 colonies	suspect contamination	suspect contamination moderate
40+ colonies	suspect heavy contamination	suspect heavy contamination

D) Specie Identification

The specie identification was performed by a clinical microbiologist. The gram-negative bacilli were identified using the API 203. The yeast were identified by API 20C, and morphology on corn meal with tween 80. The Neisseria species were identified on the basis of gram stain, morphology, and the oxidase test. The Staphylococcis epidermidis isolates were identified on the basis of Gram stain, morphology, and catalase and coagulase test.

E) Symptom Questionnaire

The symptom questionnaire included: cough, sputum, change in the color of sputum, shortness of breath, wheezing, fluid retention, and exercise tolerance. The subjects were able to rank their symptoms according to the degree that they exhibited that symptom. They had the option of selecting none (absence of symptom) to severe degree of symptom. This questionnaire was administered at the beginning and end of each participant's involvement. The investigator

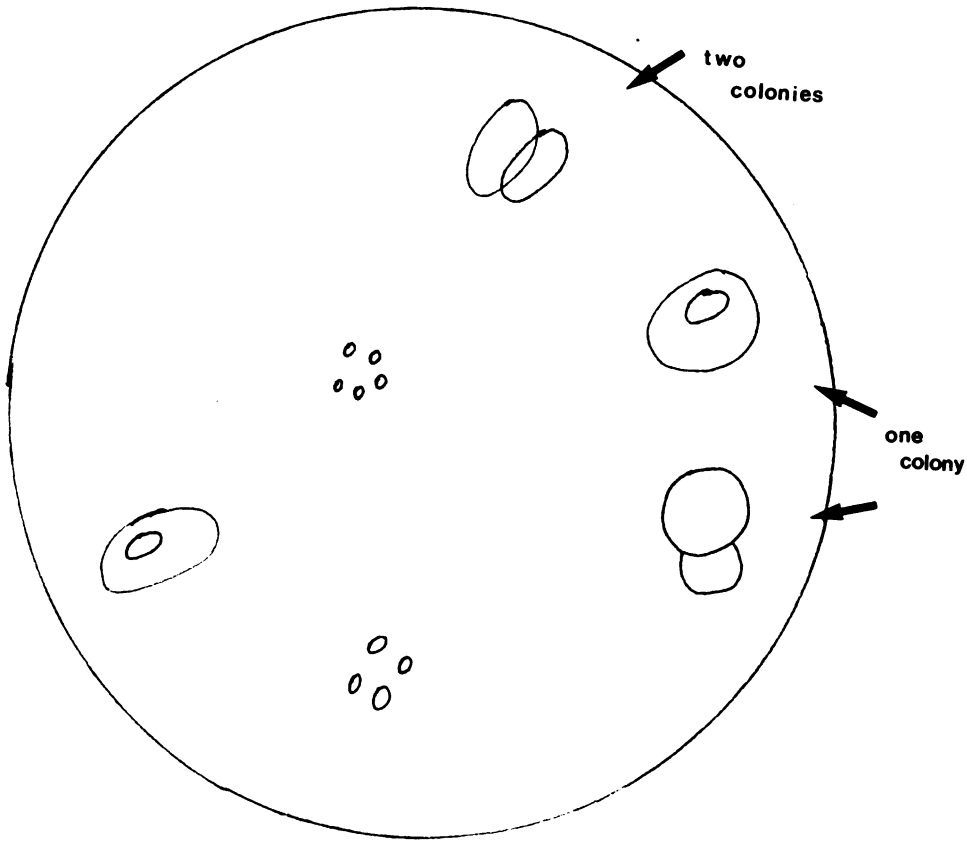


figure 4 & 5 COLONY COUNT AND READING

observed subjects who ranked their symptoms low (none) when they exhibited a greater degree of impairment. For example, one subject on continuous oxygen therapy ranked her shortness of breath as minimal even though she appeared dyspneic. This questionnaire has been proven to be a valid instrument and has been correlated with indicating exacerbation of pulmonary disease (Hopewill, 1981).

3. Instrument Validity

Ryan and Mihalyi (1977) evaluated the Aero-Test sampler's ability to detect bacteriological contamination or respirator-generated aerosols; they compared it to the Anderson Air Sampler. The Anderson air sampler is a viable particle measurement of bacteria whereas the Aero-Test sampler is read directly as colony counts. To simulate contamination, respirators (Bennett AP-5, MA-1, PR-1) were intentionally contaminated with laboratory isolated bacteria (Escherichia coli, Pseudomonas A., and Acinetobacter calcoaceticus var. anitratus). They standardized cycling time, pressure setting (according to type of respirator), flow rates, sensitivity, and nebulization. A 10 second sampling time was used. Cultures were incubated at 35 degrees centigrade for 24 hours.

A total of 77 respirator effluent tubing samples were obtained. The viable particle counts (Anderson sampler) that contained fewer than 20 bacteria/0.028m^3 gave negative results (no colonies) by the Aero-Test sampler. Ryan and Mihalyi were able to correlate moderate contamination by the Aero-Test sampler with more than 20 bacteria particles per plate from the Anderson sampler. They reported that Aero-Test colony counts of 40 colonies indicated heavy contamination of equipment. They suggest because of the sensitivity of this sampler it provides an easy in-use sampling of equipment to assess breaks in disinfection, handling technique, and cleaning programs. Ryan and Mihalyi reported colony counts greater than 40 colonies indicated a risk of nosocomial

infection, whereas 1 to 40 colonies reflected greater contamination than the hospital environment.

IV. Design Validity

In a quasi-experimental design the full experimental control is lacking; thus the ability to infer cause and effect relationships is limited. Because of the small sample size (18), the statistical power was low and the possibility of making an incorrect no-difference conclusion (type II error) does increase. Due to the inability to control extraneous variables, valid inferences of covariance are difficult. The design of this study did not control for environmental factors (e.g. antibiotics, steroids, and degree of activity) which proved to be significant variables affecting the outcome of the equipment culture. Even though the study sample was considered homogenous by pulmonary diagnosis, a difference between the control and experimental groups did exist.

Because of the untreated control group, a threat to internal validity is the selection-maturation process (Cook & Campbell, 1977). The experimental group, having more exposure to the investigator, may have gained knowledge at a greater rate than the control group. This increased exposure to the investigator could make a difference in the pre and posttest scores. Because only the investigator came into contact with both groups, the major threats to internal validity were restricted. Threat of instrumentation was controlled for by the investigator using the same "wording" in administering questionnaires and in obtaining the cultures.

The major threat to internal validity was the problem of morbidity. Several subjects were hospitalized while in the study and returned to complete the study. One subject was hospitalized three times before he was able to complete the study. The incidence of frequent hospitalizations directly influenced the outcome of the cultures since changes in the microflora of the

respiratory tract are caused by the hospital flora (Sander, Sander, & Harrowe, 1976; Matthay & Greene, 1980).

A final threat to internal validity is from the outside events (physician's orders, unwilliness to comply, increased severity of disease) which can directly affect compliance and which influenced the outcome of this study. Study limitations are discussed in Chapter five.

The generalizability of this study will be very limited due to the small 'n' of the study sample. The population of this study sample share similarities (age, disease history, limitation of activity, dependency upon equipment, etc.) in disease pathology and physical limitations. The sample is representative of the population of individuals who suffer from chronic lung disease. The participants in this study were fairly homogenous by social class and age grouping. A definite threat to the external validity is the small sample size, and its modest generalizability will be limited to those individuals sharing commonalities in disease and type of inhalation equipment.

Chapter Four

Introduction

Since the highest level of measurement for this data set is the nominal and/or the ordinal level (dichotomous), nonparametric statistical analyses were used.

A. Statistical Approaches

The data was coded for computer analysis and descriptive statistics of all the variables was performed. Crosstabulation of certain variables considered to be significant to the outcome of this study was conducted. A total of 136 variables were crosstabulated and results were reported using Kendall's Tau with one-tail P values. Variables crosstabulated between control and experimental group were: dependent variables (tubing contamination, nebulizer contamination, and type of bacteria) against impinging variables (corticosteroids, antimicrobials, hospitalizations, multiple organ disease, and the number of hours since equipment was last decontaminated before the culture was obtained).

The dichotomous variables of the experimental and control group were crosstabulated against the descriptive statistics. Pretest and posttest scores were compared according to dichotomous and ordinal variables. Results of the dichotomous variables were reported using the McNear test with two tailed p values; the ordinal variables were reported by the Wilcoxons matched pairs signed-rank test.

B. Statistical Analysis

The hypothesis that the equipment used by the treatment group would be less contaminated than that of the control group because of differences in the type and frequency of the cleaning procedure used on that equipment was rejected. The data analysis indicated the control and experimental groups had equal degrees of contamination. The difference in the degree of equipment

contamination between the two groups was not statistically significant. The lack of statistical significance could have been partially explained by the small sample size. Because of the general trend in the data towards acceptance of the hypothesis, the data will be reported in descriptive terminology.

The total sample study was comprised of eleven females (61.1%) and seven males (38.9%) with a mean age of 66.2 years. Table 6 presents the frequencies of general information about the study population. Only the variables considered to be significant to the outcome results will be discussed.

Table 6

Frequencies for Sample Demographic Variables

Variable	Experimental		Control	
	Female	Male	Female	Male
1. Sex	3	6	8	1
2. Age (mean) years	65.3	69.2	66.3	66.
3. Homebound	1	3	3	1
4. Permanent tubing	2	5	7	1
5. Disposable tubing	1	1	1	0
6. Purified water	0	1	1	0
7. Distilled water	2	2	6	1
8. Tap water	0	1	0	0
9. Sterile saline	1	2	1	0
10. Length Diagnosis (mean yrs.)	8.3	10.8	6.13	14
11. O_2 therapy - continuous	1	0	2	0
12. O_2 therapy - nocturnal	1	2	1	0
13. Marital status:				
Married	2	4	2	1
Single	0	0	0	0
Widow	1	0	1	0
Divorce	0	2	5	0
14. Ethnicity:				
Anglo-American	2	5	7	0
Afro-American	1	1	0	0
Mexican-American	0	0	1	0
Japanese	0	0	0	1

I. Study Findings

A. Tubing contamination

Contamination in the tubing and nebulizer were reported as 10^{o}-10^{1}, 10^{1}-10^{2} or greater than 10^{2}. Contamination was further ranked by the virulence of bacteria. The lowest value for virulence of bacteria was assigned a 2 value and the highest (most virulent) was assigned a 5 value. The value of 3 and 4 indicated moderate degrees of virulence and the value of 1 equalled no virulence. Virulence of bacteria was ranked according to its pathogenicity; thus the highest rank of five was considered the most likely to cause pulmonary infection. The same ranking was applied to the bacteria found in both the tubing or nebulizer. The specific bacteria and the rank assigned to each was:

Bacteria	Rank
Staphylococcus epidermidis	2
Neisseria species	2
Candida parapsilosis	3
Candida lipolytica	3
Acintetobacter cloaceticus	4
Moraxella specie	4
Enterobacter cloacae	5
Enterobacter aerogenes	5
Pseudomonas maltophilia	5
Klebsiella pneumonia	5

In the total sample study, post treatment contamination of the tubing was present in three subjects (16.7%). One experimental subject (11.1%) had tubing contamination in the 10^{0}-10^{1} degree of contamination; two control subjects (22.2%) had 10^{1}-10^{2} degree of tubing contamination. The bacteria found in the tubing of the experimental subject was the second rank of virulence (Neisseria specie); the control subjects had a dual ranking of five (Pseudomonas meltophilia, Enterobacter cloacae) for bacteria virulence. The higher degree of bacteria contamination and pathogenicity found in the control group tubing lends significance to the importance of equipment cleaning. The control subjects with tubing contamination last decontaminated (soaked in vinegar and water) the

equipment 98 hours prior to culturing. The vinegar and water dilutional strength used by both control subjects was considered "low" in effectiveness. This solution strength was comparable to a 10% acetic acid solution, rather than 0.25% recommended solution by Pierce and sanford (1973). One subject in the experimental group with tubing contamination decontaminated his eqiupment 48 hours before the culture was taken; the vinegar and water dilutional strength used was equal to 0.25% acetic acid in its effectiveness. A relationship appears to exist between when the equipment was last decontaminated (hours) and when the culture was obtained. Associated with this relationship is how strong (effective) of a dilution of vinegar and water solution the subject used in cleaning the equipment. Whether how long (10 to 30 minutes) the equipment was soaked in this solution contributed to the effectiveness of the decontamination process was not extrapolated from the analysis. The combination of these factors (vinegar and water solution and frequency of using the solution) may cause the contamination present in the tubing. Statistical significance was not present but the analysis did infer a relationship between these factors.

The gram-negative bacillus (Pseudomonas maltophilia) found in one of the tubings in the control group is considered a significant finding because of the known pathogenicity of this organism. Pseudomonas maltophilia multiplies in a warm, moist environment and is very difficult to eradicate. This singular finding is important to mention as Pseudomonas maltophilia is a problem pathogen for the home care patient. The subject was symptomatic of having a pulmonary infection (sputum was green and thick; bibasilar rales). Furthermore, this subject had a history of recurrent pulmonary infections refractory to treatment. Probably because the source of her pulmonary infections was not explored and the contaminated equipment was re-infecting her after each regime of antibiotic therapy.

The capability of gram-negative bacilli inhabitating the tubings, although not a frequent occurrence in either group, is a significant finding. These bacteria pose a threat of infection to the subject using inhalation equipment at home.

B. Nebulizer Contamination

Contamination of nebulizers was a significant problem for both the control and experimental group. Only 3 subjects (16.7%) of 18 had negative cultures. Eighty-three percent of study sample nebulizers tested were contaminated; the degree of contamination varied from 10^0-10^1 to greater than 10^2 colony counts. The experiemntal group had seven (77.7%) positive cultures while the control group had eight (88.8%) positive cultures (see Table 7). Both groups were considered to have an equal incidence of contamination but the pathogenicity of the organisms varied between groups. The bacteria isolated from the control group nebulizers were considered to be more virulent. The majority of nebulizers in both groups were contaminated and thus statistical significance was absent. The conclusion of the analysis indicated there was a 'no difference' in contamination between the two groups. As a result, the equal contamination of nebulizers in both groups supported the rejection of the hypothesis.

The effectiveness of the treatment (cleaning program) in the experimental group was difficult to ascertain because of variables that altered this treatment. Of the 7 contaminated cultures in the experimental group, 5 were of the 10^0-10^1 degree of contamination and 2 were greater than 10^2. The amount of time that had elapsed since they last decontaminated (vinegar and water soaks) prior to collection of the culture did not have a significant relationship with regards to contamination. This was in direct contrast to the tubing contamination which indicated a relationship. The average time span since the equipment was last cleaned (decontaminated) for the experimental group was 70 hours (range 48-98

Table 7

Comparison Between Experimental and Control Groups In Degree and Severity of Contamination

Contamination	Experimental										Control									
Subject Number:	1	2	3	4	5	6	7	8	9	mean	1	2	3	4	5	6	7	8	9	mean
Tubing contamination *a	0	1	0	0	0	0	0	0	0	0.11	0	0	0	2	0	0	2	0	0	.44
Tubing bacteria *b	0	2	0	0	0	0	0	0	0	.22	0	0	0	2	0	0	5	0	0	1.33
Nebulizer contamination	1	0	3	1	1	3	1	0	1	1.22	0	1	1	1	1	1	3	1	0	1.00
Nebulizer bacteria *b	+2	0	5	2	2	5	2	0	3+	3.0	0	4	3	2	3	2	5*	4	0	3.28
Hours - last Decontamination										70										7.43

*a

0 = no contamination

1 = 10^0-10^1

2 = 10^1-10^2

3 = greater than 10^2

\+ = Psuedomonas M.

* = Klebsiella P.

*b

2 = Staphylococcus epidermidis
Neissera species

3 = Candida P.
Candida L.

4 = Acinetobacter C.
Moraxella specie

5 = Enterobacter C.
Enterobacter A.
Pseudomonas M.
Klebsiella P.

hours) before the culture was taken (see Table 6) for those experimental subjects sharing the 10^0-10^1 degree of contamination. The two experimental subjects with greater than 10^2 degree of contamination had decontaminated the equipment at 98 and 72 hours before the culture was obtained (see Table 6). The time since the equipment was last cleaned did not seem to interact significantly with the degree of contamination. The strength of the decontamination solution was a controlled variable in the experimental group and thus the solution was considered effective as a decontaminant.

If the equipment would be cleaner if cleaned by a family member was only partially answered. More family members cleaned the equipment in the experimental group which was indicative of their physical debilitation. Only four subjects (44.4%) in the experimental group cleaned their own equipment. The remaining five subjects (55.5%) had their equipment cleaned by family members. The equipment cleaned by family members had a lower rank of bacteria and a lower degree of contamination. The control group, in comparison, all cleaned their own equipment ($p=0.0058$). The debilitated physical status of the experimental subjects was a factor in the non-comparability of the two groups.

The seven control subjects with contaminated nebulizers had degrees of contamination ranging from 10^0-10^1 (six nebulizers) to greater than 10^2 (one nebulizer). The control group did manifest a lesser degree of contamination, but not significantly less. What was significant was that the bacteria found in the control group was more pathogenic.

The last cleaning (decontamination) of the control group equipment averaged 74.3 hours before culture collection. Even though, this represented a 4.3% increase in hours from the experimental group it was not statistically significant. The strength of the vinegar and water solution varied widely within the control group. The majority (66.7%) used a 10% decontaminating solution

which was considered to have minimal effectiveness. One control subject did not even use vinegar and water solution to clean the equipment. Only two subjects (22.2%) used an adequate dilutional strength (0.20-0.25%) of vinegar and water. Of those who used vinegar and water soaking solution, the average soaking time was 10 to 20 minutes. A 30 minute soaking time was used by two control subjects. Whether a weak contaminating solution and a short soaking cycle affected contamination of equipment was not examined in this study. Statistical analysis did infer an existing relationship between these two factors.

There was a difference in the type of bacteria found in the nebulizers of the experimental and control group. Table 8 delineates these differences in bacteria species.

Table 8

Bacteria Isolated and Frequency

Bacteria	Frequency experimental/control		Rank
Staphylococcus epidermidis	3	0	2
Neisseria species	1	0	2
Candida parapsilosis	1	2	3
Candida lipolytica	1	0	3
Acinetobacter caloaceticus	0	1	4
Moraxella specie	0	1	4
Enterobacter cloacae	1	0	5
Enterobacter aerogenes	1	0	5
Klebsiella pneumonia	0	1	5

Ranked Bacteria: Incidence

Rank	Frequencies	
	Experimental	Control
2	3	0
3	2	2
4	0	2
5	2	1

In order to imply significance bacteria with a rank of three, four, or five were considered more pathogenic based on their ability to cause pulmonary infections. A slightly greater degree of pathogenicity of nebulizer bacteria in the control group is evident when rank 4 and 5 are combined. Three nebulizers in the control group were contaminated with bacteria in the 4 or 5 ranking. While two nebulizers in the experimental group were contaminated with bacteria in the same ranking. All of the bacteria in the four or five ranking were gram-negative microorganisms. Acientobacter c., Moraxella sp., and Klebsiella p. were the bacteria isolated from the nebulizers in the control group in the 4 or 5 ranking. All of these gram-negative bacilli are capable of causing pulmonary infection and Klebsiella p. which is difficult to treat is a problem pathogen for the COPD patient. The pathogens isolated from the experimental group included Enterobacter c. and Enterobacter aerogenes. Enterobacter cloacae is found readily in the "hospital flora" and this subject was hospitalized during the course of the study and returned to complete his participation. It was also this nebulizer that had the highest degree of contamination. What is significant is that the patient was hospitalized and returned to the home symptomatic of infection and on antibiotic therapy. These factors probably directly affected the results of contamination found in this subject's nebulizer.

The staphylococcus epidermidis (rank 2) was the only gram-positive microorganisms cultured. This bacillus was isolated from an experimental subject's nebulizer who was hospitalized during the course of the study. This is another common "hospital flora" which needs to be considered because of the recent hospitalization. Those bacteria ranked a three (Candida species) are yeast species and opportunistic microorganisms. Candida sp. can cause systemic infections in the COPD patient which are serious and difficult to resolve. All of the gram-negative bacteria found in this study are opportunistic microorganisms

and are well known to cause pulmonary infection in the COPD patient.

Even though the difference of incidence between species of bacteria was considered minor, the increasing virulence of bacteria in the control group's nebulizers combined with the slightly higher degree of increased virulence of bacteria in the control group's tubings indicate that the treatment might have had some effect on the outcome. How effective vinegar and water soaking solution (decontamination) was in eradicating the vegetative microorganisms found in inhalation equipment was not answered. However, because the experimental group had a modest difference in the virulence of the bacteria found in the tubings and nebulizers, the stronger solution of vinegar and water combined with more frequent cleaning may have contributed to this lower incidence. This does imply an association between less contamination and the cleaning/treatment and is mentioned because of an apparent relationship that was extrapolated from the analysis.

Summary

The purpose of the study was to evaluate the effectiveness of an entire cleaning schedule including vinegar and water as a decontaminating solution. Unfortunately, the study results did not support the hypothesis for several reasons. Originally, it was assumed the two groups would be comparable; but as it turned out the groups were not comparable and this had a major impact on the results of the study. The major reason the groups were dissimiliar was the fact that the experimental group was the "sicker", less stable group, and this greater instability affected the results of cultures. During the analysis certain variables were detailed as significant in influencing the outcome of the study. These variables warrant discussion because of the impact they have upon the patient using inhalation equipment at home. The cleanliness of the equipment is directly correlated to the impinging variables which will be discussed next.

II. Group Differences

Experimental Group: Six males and three females comprised this group and the mean age was 66.2 years. The majority of the group (66.7%) were married; seven subjects were caucasian and two subjects were of the black race. Seven (77.7%) of the subjects had been diagnosed as having pulmonary disease for over eight years and one had been diagnosed for over 25 years. The average length of time they had used inhalation equipment in the home was five years. Three subjects had used equipment at home for almost ten years; one subject had used equipment at home for eleven years. Use of oxygen therapy (continuous or twelve hours daily) and the degree of "home-boundness" was comparable between the two groups. Three of the experimental subjects had never smoked while six subjects had a history of smoking more than one-half packs a day. Of these six smokers, five had smoked for over twenty years. In the experimental group two subjects continued to smoke on a daily basis.

Control Group: This group was comprised of eight females and one male. The mean age of the groups was 65.7 years. Three of the subjects were married (33.3%) while six were either divorced or widowed. The majority (77.8%) of the subjects were caucasian; the two other subjects were of Japanese and Mexican-American descent. Five subjects (55.5%) had been diagnosed as having pulmonary disease for 8-25 years. Four were diagnosed in the last seven years and three of these four were diagnosed in the last three years. This is a distinct difference in pulmonary disease history compared to the experimental group. Only one experimental subject had been diagnosed in the last three years. A difference in the number of years they had used equipment in the home was also present. The control group averaged 5-10 years (eight subjects) whereas the experimental group averaged five years (5 subjects). Six of the control subjects had a history of smoking more than a pack of cigarettes a day for more than 20

years and three continued to smoke daily. The smoking history of the control group was longer than the experimental group.

Differences: In general, the experimental group was the sicker of the two groups and the greater debilitation in their physical status affected the outcome of the study results. They were considered "sicker" based on an increase use of antibiotics and steroids. Also, the fact that two experimental subjects were hospitalized during the course of the study reflected their physical instability. Because these three variables (antibiotics, steroids, hospitalization) were identified as significant to the outcome of the study results, they will be examined according to how they affected both the control and experimental group.

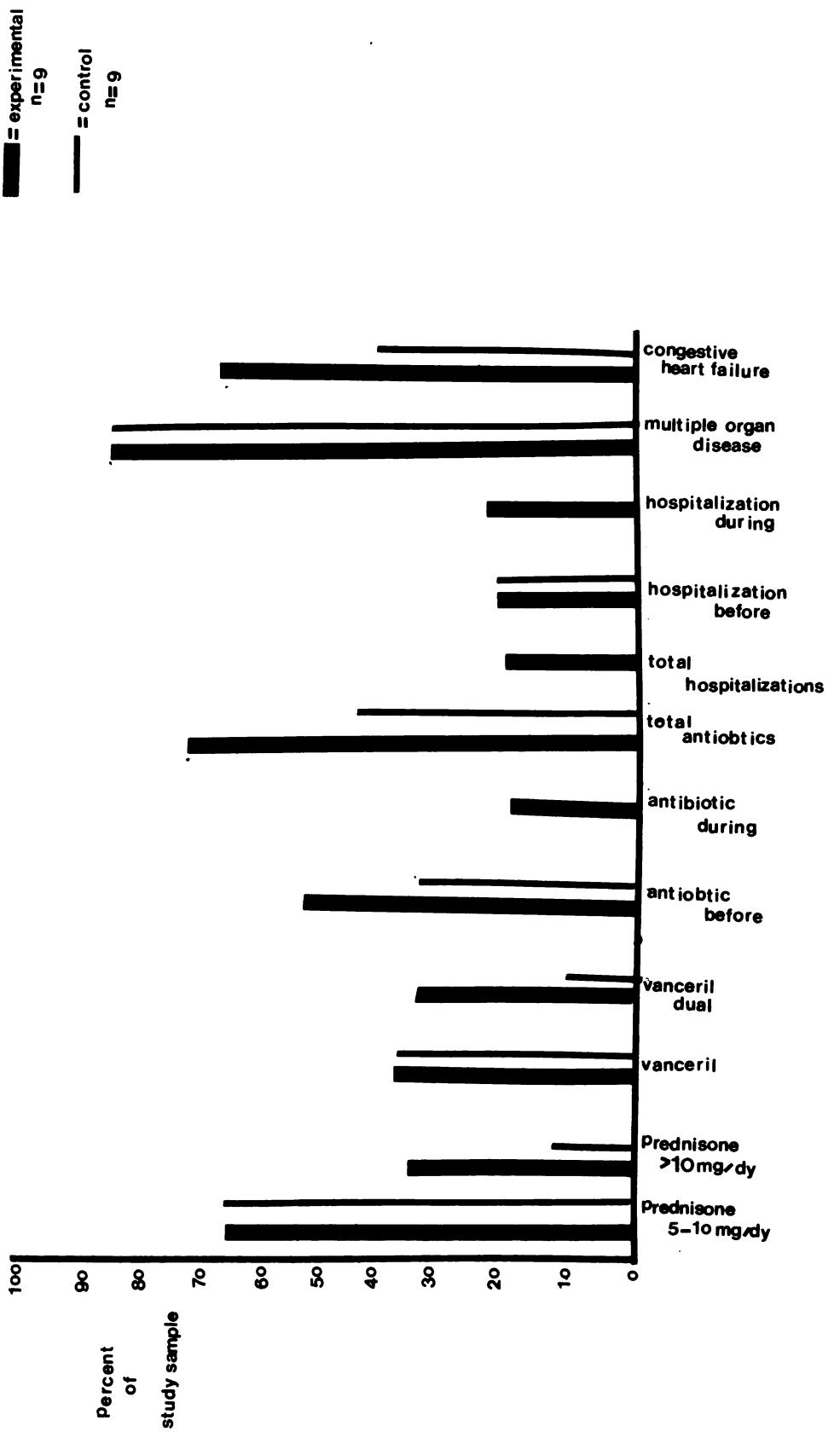
III. Impinging variables

The experimental group was considered "sicker" because they had a greater incidence of multiple organ disease, because they were hospitalized more frequently, and because they had greater use of antimicrobial and corticosteroid therapy. These four variables were delineated by the statistical analysis as having significant influences on the degree of contamination found in the nebulizers and tubings. Table 9 emphasizes how these differences contributed to the non-comparabilty of the two groups.

Contamination of the nebulizers in both the control and experimental group were comparable by the number of nebulizers contaminated in each group. The bacteria isolated from the nebulizers of the control group was more pathogenic even though the colony counts in some cases were lower. For any future researcher planning to examine equipment in the home controlling for the following impinging variables is imperative.

TABLE 9

CORTICOSTEROIDS, HOSPITALIZATIONS, & MULTIPLE ORGAN DISEASE



A. Multiple Organ Disease

The experimental and control group had an equal incidence in multiple organ disease of 88.8% (sixteen out of eighteen) (Table 9). The degree of incidence for congestive heart failure was significantly different between the two groups. In the experimental group six subjects (66.6%) had a history of congestive heart failure. In contrast, the control group had only four subjects (44.4%) with congestive heart failure. Table 8 provides a visual explanation. Congestive heart failure when associated with COPD indicates that pulmonary vascular pressures are elevated and the subject usually exhibits some impairment of heart function. COPD with coexisting congestive heart failure can be representative of long standing pulmonary disease. The increased incidence of congestive heart failure in the experimental group was of minor significance ($p=0.0793$).

The overall incidence of multiple organ disease was equal between the two groups but there was a difference in incidence of specific diseases. Multiple organ involvement was comprised of six sub-categories: congestive heart failure, rheumatic heart disease, gastric ulcer or gastric distress, diabetes mellitus, arteriosclerotic heart disease (angina included) and other (vascular problems, alcohol abuse). Because of the different categories for multiple organ involvement, a difference in the incidence of elevated blood pressure, and gastric distress was present in the experimetnal group. For instance, five subjects (55.5%) in the experimental group had elevated blood pressure compared to two subjects (22.2%) in the control group. The experimental group had more complaints of gastrointestinal distress than the control group. These complaints of gastric distress by the experimental group were reflective of subjective rather than objective organ involvement. In compiling the actual multiple organ involvement between the groups the incidence was equal. The sub-category

differences are mentioned because elevated blood pressures and gastric distress can reflect chronicity of their pulmonary disease. Multiple organ disease denotes progressive disease processes and a tendency for greater susceptibility to infection. These increases in subjective complaints in the experimental group is indicative that they were generally a "sicker" group of subjects.

B. Corticosteroids

All of the subjects (100%) in the experimental group used oral corticosteroids (prednisone) compared to seven (77.7%) subjects in the control group (Table 9, p.). The dosage of prednisone varied from 10 mg. daily (66%) to a maximum of 30 mg. daily (33%) for the experimental group. Comparatively, two subjects in the control group did not use any form of oral steroid therapy. Six control subjects (66.6%) were on 10 mg. daily dosage of prednisone and one subject (11.1%) was on 20 mg. of prednisone daily. This difference in use of oral prednisone between the two groups (experimental 100%/control 77%) was an important variable. Table 9 (p. 65a) provides a graphic explanation of steroid usage between the two groups.

There was also a difference between the two groups in use of inhaled steroids. Three of the experimental subjects (33.3%) were on Vanceril and all three were on oral corticosteroid therapy. Comparatively, three control subjects were on Vanceril and only one had dual coverage with the oral corticosteroid. One possible reason why the experimental group was on more corticosteroids could have been related to their pulmonary instability. Corticosteroid therapy does cause changes in the microflora of the respiratory tract and this suppression of normal flora can enhance invasion by the gram-negative and fungal microorganisms. What the true impact of the corticosteroid therapy had in affecting the contamination of nebulizers could not be delineated by statistical analysis. Rather a relationship or trend in the data suggested that subjects on

corticosteroids therapy tended to have contaminated nebulizers. This was true for both the experimental and control groups.

C. **Antimicrobials**

The overwhelming difference in use of antimicrobials between the control and experimental group is visually demonstrated by Table 9 (p.65a). Information of different forms of antimicrobials (ampicillin and tetracycline) was solicited from both groups. The overall incidence of antimicrobial therapy in the experimental group was 77.7% compared to 44.4% for the control group. The criteria for these results were that subjects had to have used an antibiotic in the last six weeks; the majority had used an antibiotic in the last month.

Two of the experimental subjects were very unstable and as a result were on antimicrobial therapy during their participation in the study. Both were started on antibiotics by physician order due to sputum changes indicative of pulmonary infection. The other five experimental subjects had either just completed antibiotic therapy before beginning the study or had been on an antibiotic a month earlier.

The control group was considered a more stable group because none of the subjects required antimicrobials during their participation. The four control subjects (44.4%) who had used antimicrobials had done so the month prior to inclusion into the study. This lack of antimicrobial therapy within the control group is striking when compared with the experimental group.

Crosstabulation of antimicrobial therapy and contamination of nebulizers showed a trend towards contamination by less virulent organisms and use of antibiotics. Subjects who had used antibiotics in the last month or during the study had nebulizer contamination with less virulent microorganisms. Table 9 (p.65 a) demonstrates the relationship between virulence of the microorganisms found in the nebulizers and the lack of antimicrobial therapy. Two of the control subjects

(22.2%) who had not used any antibiotics before the study had nebulizer contamination with rank 4 bacteria (Moraxella species and Acinetobacter caloaceticus). It is known that antimicrobial therapy encourages growth of certain microorganisms (gram-positive) and provides a protection against colonization of respiratory tree with certain gram-negative bacillus.

The extensive use of antimicrobials by the experimental group resulted in the inability to clearly evaluate the treatment (cleaning program) outcome. It was assumed that because antimicrobials suppress the normal respiratory flora an underlying pulmonary infection might be present, thus the effectiveness of the cleaning program would be masked. The one experimental subject with a negative nebulizer culture had not been on antibiotics in the last month and did not use oral corticosteroids. Because the experimental group experienced more pulmonary infections during the study, this affected both the outcome results and the comparability of the two groups. A definite relationship exists between antimicrobial therapy, corticosteroid therapy, and equipment contamination.

D. Hospitalizations

The last factor that implied the experimental group was a more unstable and "sicker" group of subjects was the frequency of hospitalizations. Three fourths of all the subjects (13 out of 18) had been hospitalized in the last 6 months. Three of these thirteen had been hospitalized in the month prior to their involvement in the study. This is a significant incidence of hospitalization but frequent exacerbations are not uncommon for the individual with chronic lung disease.

The frequency of hospitalizations in the control group and experimental group are graphed in Table 9 (p.65a). The percentages represent the combined incidence of hospitalizations before and during the study. The experimental

group had a combined total of 44.4% compared to the control group of 22.2%. This percentage for the control group represents hospitalizations occurring before the study as none of the control group were hospitalized during the study. Two of the experimental subjects were hospitalized during the study and this was of significance (p=0.07). All of these hospitalizations were for pulmonary complications. The protocol the investigator implemented to integrate the subject back into the study once hospitalized involved a three week waiting period before continuing the study. The three week time period was selected because of the known changes that occur in respiratory flora because of the "hospital flora". These changes by 'hospital flora' could directly impact on the culture results.

Of the two experimental subjects hospitalized during the study, one subject was hospitalized a total of three times before he completed his participation. Interestingly, one of these subjects had the highest degree of nebulizer contamination (greater than 10^2; colony count of 500) in the total sample study. The microorganism isolated from his nebulizer (Enterobacter cloacae) is considered pathogenic and was ranked a five (highest rank) in the statistical analysis. The other subject had a gram-positive microorganism (staphylococcus epidermidis) isolated from his nebulizer which might have been indicative of the recent hospitalization. As a result of the two experimental subjects being hospitalized during the study, the results of the cleaning/treatment were skewed because of the excessive contamination present in their nebulizers. Exclusion of these two subjects from the statistical analysis would have added greater support to the study hypothesis.

Only two subjects (22.2%) in the control group were hospitalized and both incidents occurred prior to beginning the study. Table 10 (p.70a) illustrates the striking difference in hospitalizations between the two groups. This greater

Table 10

Impinging Variables and Type of Bacteria

Variables	Experimental									Control								
	1	2	3	4	5	6	7	8	9	1	2	3	4	5	6	7	8	9
Neb. Contamination	1*	1	3	1	1	3	1	0	1	0	1	1	1	1	1	3	1	0
Neb. Bacteria	2**	2	5	2	2	5	2	0	3	0	4	3	2	3	2	5	4	0
Hospitalizations	0	0	D	0	D	B	0	0	B	B	0	0	0	0	0	0	0	B
Antimicrobials	B	B	D	0	D	B	B	0	B	B	0	0	0	0	B	B	0	B
Steroids P.O.	+	+	+	+	+	+	+	+	+	0	+	+	+	+	0	+	+	+
Vancerial	0	0	+	0	0	0	+	0	+	+	0	0	+	0	+	0	0	0
Multiple organ dx	+	+	+	+	0	+	+	+	+	+	+	+	0	+	+	+	+	+
CHF	+	0	+	0	0	+	+	+	+	+	0	0	0	+	+	+	0	0
Gastrointestinal	0	0	+	+	+	0	+	0	+	0	+	0	0	0	+	0	0	0

Scale

*0 - 3 = degree of contamination

**0 - 5 = virulence of bacteria

D = During study

B = Before study

+ = on therapy/or a positive disease history

incidence (by 20%) of hospitalizations for the experimental group forced the study results towards rejecting the hypothesis. The interrelationships between these variables (multiple organ disease, corticosteroids, antimicrobials, hospitalizations) and how they impacted on the study results and equipment contamination cannot be overlooked.

Summary

The group differences according to these impinging variables are significant findings and reiteration of the results will clarify the interrelationships. Multiple organ involvement was of equal incidence (88.8%) in both groups and congestive heart failure was reported in 66.6% of the experimental subjects and 44.4% of the control subjects. Antimicrobial therapy was a significant finding with 77.7% of the experimental subjects reporting use of antibiotics in the last month compared to 44.4% for the control group in the same time period. Another significant finding was the greater use of corticosteroid therapy by the experimental group which was 100% whereas the control group had an incidence of 66.6%. The last significant variable was the frequency of hospitalizations within the experiemental group; a percentage total of 22.2% were hospitalized before the study and 22.2% during the study (total 44.4%). In comparison, the control group consisted of only 22.2% (2 subjects) who had been hospitalized prior to beginning the study. All of these variables point to the greater instability within the experimental group and this supports the conclusion that they were a "sicker" group. Furthermore, all of these factors affected the degree of contamination found in the inhalation equipment and the effectivness of the decontamination method.

IV. Equipment Contamination

The small sample size did not lend itself to statistical significance but certain variables were definitely associated with a greater degree of

contamination. What these interrelationships or trends were are valuable in assessing contamination of home equipment and the role of cleaning.

The degree of contamination found in the tubings and nebulizers and the type of bacteria were crosstabulated against antimicrobials, corticosteroids, multiple organ disease, and hospitalizations. The following conclusions were extrapolated from the crosstabulations and are not distinguished by groups.

Seven subjects who had used Ampicillin prior to inclusion into the study all had negative tubing cultures. This was statistically significant at a p-value of 0.07. This protective mechanism of antimicrobial therapy (mainly ampicillin) was indicated at another point in the data analysis (see Table 10, p. 70a). In evaluating antimicrobial therapy in the month prior to inclusion in the study, the analysis showed a positive relationship to 'clean' equipment. For example, three subjects who had used ampicillin in the last month had negative nebulizer cultures (p=0.0046). But, it should be emphasized that three other subjects also also had used antimicrobials (excluding ampicillin) in the last month had positive cultures. Significantly, two (out of the three) subjects were in the experimental group and were the same subjects who had been hospitalized prior to the study.

Associated with nebulizer contamination was corticosteroid therapy. Daily prednisone therapy (both low and moderate dose) was associated with contamination of equipment nebulizers for all of the subjects. When daily prednisone therapy was correlated with the inhaled steroids it indicated a relationship to contamination of the nebulizers. Six of the subjects in the total sample study used Vanceral and five of the six had positive nebulizer cultures. When the type of bacteria found in the nebulizers was correlated by the form of corticosteroid the growth of bacteria in the two to four rank revealed a p-value of 0.02. The combination of steroid therapy and recent hospitalization were associated with equipment contamination.

The frequency of hospitalizations impinged upon the results of nebulizer cultures and to a lesser degree on tubing contamination. Whether the actual hospitalization and the resultant changes in the resident flora of the respiratory tract were responsible for the incidence of nebulizer contamination was not delineated by the analysis. However, recent hospitalizations could imply instability of the pulmonary status which enhanced colonization of the respiratory tract by gram-negative microorganisms. If the patient was on steroid therapy and recently hospitalized, the potential for having contaminated equipment was almost assured. (Table 10, p.70a).

Crosstabulation of the data revealed that multiple organ disease was associated with contamination of inhalation equipment. Subjects with congestive heart failure usually had contaminated nebulizers. Chronic gastrointestional disorders and nebulizer contamination were also associated by rank of bacteria. The analysis indicated that a subject with gastrointestional problems had more pathogenic microorganisms inhabiting their nebulizers. Understanding the exact relationship between these two variables is beyond the scope of the investigation. The analysis did show a trend and relationship of these variables (multiple organ disease, antimicrobials, corticosteroids, hospitalizations) to equipment contamination but the actual etiology was not extrapolated.

A. Pretest and Posttest Scores

Another verification that the experimental group was "sicker" and that this could have facilitated equipment contamination was the results of the pretest and posttest scores. The posttest revealed that the experimental group exhibited more severity of shortness of breath at the conclusion of the study ($p=0.05$). The difference in scores between the groups showed that the experimental group was slightly more symptomatic at the conclusion of the study (increased shortness of breath, sputum change, increase in rales). The control group scores remained

essentially unchanged. The difference in the scores for the experimental group indicated some degree of instability and this might be a limited indicator for assessing pulmonary infection.

Table 11

Comparison of Pretest and Posttest Scores

	Pretest		Posttest
	Mean		Mean
Experimental			
Sputum production	2.50		3.13
Shortness of breath	1.50	1 = absence	2.25
		2 = mild	
Control		3 = moderate	
		4 = severe	
Sputum production	0.0		0.0
Shortness of breath	0.0		1.0

There was a significance in the presence of rales in the experimental group at the closure of the study (posttest) and the p-value was 0.0326. The pretest and posttest did not show wide ranges in scores as had thought might occur.

Conclusion

These results infer that certain variables (antimicrobials, corticosteroids, hospitalizations, and multiple organ disease) were associated with contamination of inhalation equipment. The combination of steroids, hospitalizations, and multiple organ disease increases the probability of contaminated equipment regardless of how thoroughly the individual cleans it. Ampicillin appeared to exert some residual protective mechanism against colonization by the gram-negative bacilli while encouraging colonization of the gram-positive bacilli. The tetracyclines were difficult to correlate with any changes.

It would be impossible to elicit which variable was of major significance since they did not appear in isolation; therefore they were all considered significant. Even if the equipment is thoroughly decontaminated, this may not be

enough protection for these patients at home because of these impinging variables. The problem of the experimental group utilizing more corticosteroids and having frequent hospitalizations greatly interfered with the study results. The inability to validate if the cleaning was effective resulted from the non-comparability of the two groups. The cleaning program did provide some benefit and the pathogenicity of the microorganisms cultured from both tubings and nebulizers in the experimental group was slightly reduced although not significantly. This factor may be an indicator that the stronger solution of vinegar and water used more frequently did provide some degree of protection for these individuals even though they were sicker. Nevertheless, they are prone to contaminated home equipment.

V. Significance of the Different Bacteria

The most significantly pathogenic bacteria isolated from the nebulizers in both groups were Klebsiella pneumonia and Enterobacter species. The degree of contamination for each of these species was greater than 10^2 which was considered gross contamination. The degree of contamination and the associated virulence of these particular gram-negative bacteria make them capable of causing pulmonary infections. When comparing the two groups (experimental and control), the control group had more pathogenic microorganisms isolated in the four and five rank categories (see Table 10).

Table 10 provides a comparison between the type of bacteria isolated, antimicrobials, corticosteroids, hospitalizations, and multiple organ disease. This information was then correlated with individual members in order to assess interrelationships. The two experimental subjects who had rank 5 bacteria isolated from their nebulizers were on steroids, antibiotics, and had multiple organ disease. The highest colony count was greater than 500 colonies and was

obtained from an experimental subject who had been hospitalized during the study. Not only did this subject have an excessive degree of contamination but the bacilli isolated was very pathogenic (Enterobacter specie). Withdrawing this subjects data from the experimental group would have added validity to the effectiveness of the cleaning/treatment program. Excluding this data the remaining bacteria and their ranking (one culture of rank 3 and five cultures of rank 2) are of lower virulence than the control group. Those bacteria labeled rank 2 (Staphylococcus epidermidis and Neisseria species) rarely cause pulmonary infections in the COPD individual. When this bacteria does cause pneumonia it is usually associated with an immune suppressed process (e.g. leukemia). These findings in the experimental group emphasize that their debilitated physical status combined with the impinging variables increase their suscpetibility to pulmonary infections.

In contrast, bacteria found in the control group consisted of one culture in rank 5 and two cultures in rank 4 (Table 10, p.71). . The three control subjects with these results were on steroids and had multiple organ disease. They did not have an associated use of antimicrobials during this time and overall steroid usage was reduced (66% compared to 100% for the experimental group). Rank 2 bacteria was isolated from only two nebulizers in the control group. In general, the control group had more nebulizers in the 3,4, and 5 ranking of bacteria (5 compared to 3 in the experimental group) and it is likely this resulted from several factors. For instance, the cleaning/treatment program of a stronger dilution and more frequent cleaning could have been beneficial in reducing the virulence of bacteria found in the experimental group. The impinging variables and the fact the experimental group was "sicker" was another consideration in equipment contamination.

Conclusion

The major value derived from the study was that relationships were found to exist among the impinging variables and equipment contamination. The study results indicated equipment contamination is influenced by the following variables: corticosteroids, antimicrobials, multiple organ involvement and hospitalizations. The non-comparability of the groups may explain the rejection of the hypothesis; but the observed relationships are valuable findings and future research in this area should focus on these aspects.

Chapter Five

A. Significance

The fact that inhalation equipment at home can become contaminated emphasizes that cleaning of this equipment may be an important preventive measure. Since this equipment has been proven to be a significant source of gram-negative bacilli, evaluating how patients clean this equipment at home will hopefully take on new dimensions of concern. Furthermore, the fact that this study did not document vinegar and water soaking solution (0.25% acetic acid) as an effective decontaminate adds another dimension. The most significant outcome of this study was that cleaning of the equipment is very important since the analysis indicated that contamination of equipment occurs on a fairly frequent basis. These results are singularly helpful because minimal information had previously been available on whether contamination of home equipment was indeed a problem. The finding that contamination of home inhalation equipment is a problem substantiates the purpose of the study in re-evaluating a cleaning method for this equipment.

Because minimal knowledge existed about the cleaning of this equipment at home, the study results will provide valuable information. The increased awareness that cleaning of this equipment is important in preventing contamination may encourage health professionals to scrutinize and study further how home inhalation equipment is cleaned. The question whether inhalation equipment in the home could be a receptacle for the growth of gram-negative bacilli has been confirmed by this study. The fact that the treatment (cleaning program) resulted in inconclusive evidence to support the effectiveness of vinegar and water as a decontaminate warrants attention. Evaluating the efficacy of vinegar and water as a decontaminating solution when isolated from the other variables would be desirable. The present cleaning protocols (vinegar

and water soaking solution) in view of the questionable effectiveness may be inappropriate especially if the potential to prevent pulmonary infections can be reduced by utilization of other methods of cleaning.

The value of this study for the individual patient with COPD is unclear. Facilitating the understanding that keeping equipment clean at home may provide some degree of protection against pulmonary infections is of interest. The study may stimulate interest and concern that patients using dirty equipment at home have a high probability of infecting themselves simply from using the contaminated equipment. The more information that is available on cleaning of inhalation equipment at home can only benefit those patients whose care depends upon its use. It has been unclear if equipment at home could be responsible for infecting the patient. This study implies a clear association is present warranting evaluation and scrutiny.

This study suggests that cleaning home equipment is important. Factors which impinge upon the efficacy of the cleaning program were outlined such as: corticosteroids, antimicrobials, hospitalizations, and multiple organ disease. This study may increase interest in devising new approaches to clean equipment at home and to broaden the present protocols so as to ensure increased efficacy of decontamination. The implications surrounding the use and cleaning of inhalation equipment at home needs further exploration in order to resolve and prevent equipment contamination.

B. Limitations

The non-comparability of the two groups (control and experimental) biased the sample and was the major limitation of the study. This non-comparability of the two groups was related to the selection criteria which failed to account for variables that should have been controlled. By not controlling for certain variables (corticosteroids, antimicrobials, hospitalizations, and multiple organ

disease) the actual results were biased to disfavor the hypothesis. Not knowing beforehand that these variables could directly impact on the results of the study contributed to the lack of significant findings. Because the experimental group was "sicker" and utilized corticosteroid therapy to a greater degree than the control group, was a definite limitation. If the selection criteria had been more specific and limited subjects from enrolling into the study who were on extensive antimicrobial and corticosteroid therapy, a more comparable group may have resulted. The ability to control for these variables are necessary to evaluate the efficacy of cleaning this equipment. Furthermore, enrolling subjects who were not on corticosteroid therapy or used antimicrobials would reflect a healthier group than those that participated in this study. As it turned out, the sample study had moderate to severe pulmonary disease which was another limitation to the treatment outcome.

As stated, the number of hospitalizations within the experimental group was a limitation that was not accounted for in the sample selection. The frequent hospitalizations within the experimental group directly impacted on the evaluation of the treatment program and encouraged the inconclusiveness surrounding the treatment results. One of the subjects in the experimental group was hospitalized during the study expired a month after concluding his participation in the study. This degree of end stage disease and instability reflected in part the inadequacy of the selection criteria in controlling for these specific variables. The fact that the experimental group was "sicker" than the control group created the major difficulty and limitation of this study.

Culturing of different sources in contact with the equipment (nebulizing solution, bronchodilators, tap water, faucets, ambient air) would have provided more definite information on the sources of contamination in the home. Culturing of the equipment only at the conclusion of the subjects' participation in

the study may have been a limitation. Logically, obtaining an initial culture of the equipment would have provided direct information on before and after results of the cleaning/treatment program. This was not done due to financial limitations of the investigator but if it had been possible it would have been very beneficial.

C. Implications for Nursing

The implications for nursing that teaching COPD patients how to clean their inhalation equipment in the home continues to be a necessary and integral part of educating these individuals. For although this specific study failed to demonstrate the effectiveness of one cleaning procedure, the study clearly documented the extensive equipment contamination in the home. Encouraging the development of new cleaning techniques for home inhalation equipment may be advised for nursing practice. For nurses to coordinate and pilot a specific cleaning program is desirable due to the present question about the efficacy of vinegar and water as a decontaminant. The involvement of nurses in teaching COPD patients on how to clean and maintain this equipment at home is preventive care and within the realm of nursing practice. The actual cleaning of inhalation equipment; consideration of other variables (corticosteroids, pulmonary instability, antimicrobials) must be acknowledged in the teaching program. The more knowledgeable the patient is on how to clean and use the inhalation equipment at home the safer the environment and, hopefully, the less contaminated the equipment. The potential for preventing contamination of equipment and resultant pulmonary infections is realistic and most desirable.

Further research on cleaning of inhalation equipment at home is needed and nursing practice should be involved in such a project. The ability to validate by research what is viewed as a problem and to come to a solution is essential for all health providers and especially for nursing practice.

D. Future Research

Suggestions for future studies would be to control for use of steroids, use of antimicrobials, and frequency of hospitalizaiton. The selection criteria for a future study would have to be more restrictive in who could be enrolled into the study. A larger sample size would be desirable but with this group of patients their survival rate is poor and this does inhibit obtaining the large number. Future studies comparing different cleaning methods for home inhalation equipment may provide data on a "good" way to clean this equipment. The users of this equipment have the most to gain by discovering appropriate home cleaning techniques.

REFERENCES

Allen, J. Infection and the Compromised Host. Baltimore: Williams and Wilkins Company, 1976.

Asmundsson, T., Johnson, R., Kilburn, K., & Goodrich, J. Efficiency of nebulizers for depositing saline in human lung. American Review of Respiratory Disease, 1973, 108, 506-512.

Avery, W. Unpublished study: Investigating contamination in aerosol generating devices in the home. Personal Communication, 1981.

Barofsky, I. Medication Compliance. Boston: Charles B. Slack, Inc., 1977.

Bates, D.D.V., Macklem, P.T., Christie, R.V. Respiratory function in Disease. Philadelphia. W.B. Saunders, Co., 1971, 2nd Ed.

Block, S. Disinfection, Sterilization and Preservation., (2nd Ed.) Philadelphia: Lea & Febiger, 1977.

Busey, J., Grzybowski, S., Kilburn, K., Matthews, L., Newman, M., Organick, A., Simpson, D., & Corpe, R. Principles of Respiratory Care. A Statement by the Committee on Therapy. American Review of Respiratory Disease, 1967, 95, 327-337.

Campbell, D.T. & Stanley, J.C. Experimental and Quasi-Experimental Design for Research. Chicago: Rand McNally College Publishing Co., 1963.

Chaves, A.D., Brummer, D.L., Dickie, H.A., et al. Cleaning and Sterilization of Inhalation Equipment. A Statement by the American Thoracic Society. American Review of Respiratory Disease, 1968, 98, 521-522.

Cherniack, R. & Svanhill, E. Long term use of I.P.P.B. in C.O.P.D. American Review of Respiratory Disease, 1976, 113, 721-728.

Cook, T.D. & Campbell, D.T. Quasi-Experimentation: Design and Analysis Issues for Field Settings. Chicago. Rand McNally, 1979.

Craddock, C.G., Longmire, R., & McMillian, R. Lymphocytes and the immune response (part I). The New England Journal of Medicine, 1971, 285, No. 6, 324-329.

Craddock, C.G., Longmire, R., & McMillian, R. Lymphocytes and the immune response (part II). The New England Journal of Medicine, 1971, 285, No. 7, 378-384.

Dumas, R.J. Pseudomonas and water, with a new twist. Annals of Internal Medicine, 1972, 76, No. 3, 506-507.

Dyer, E.D. & Peterson, D.E. How are do bacteria travel from the exhalation valve of I.P.P.B. equipment. Anesthesia and Analgesia, 1972, 51, 516-519.

Dyer, E.D., Maxwell, J.G., Peterson, D.E., & Mitchell, C.R. Disposable fiberblass filter to counter bacterial contamination of I.P.P.B. equipment. Anesthesia and Analgesia, 1970, 49, 140-147.

Eickhoff, T.C. Pulmonary Infections in Surgical Patients. Surgical Clinics of North America. 1980, 60, No. 1, 175-183.

Emirgil, Sobol, Normal, Moskowitz, Goyal, & Wadhwani. A study of long-term effect of therapy in C.O.P.D. American Journal of Medicine, 1969, 47, 367-377.

Farca, A., Maher, G., & Miller, A. Pulmonary alveolar proteinosis: Home treatment with I.P.P.B. Journal of American Medical Association, 1973, 224, 1283-1285.

Favero, M.S. Proposed microbiologic guidelines for respiratory therapy equipment and materials. Health Laboratory Science, 1978, 15, 177-179.

Fouts, J. Intermittent positive - pressure breathing. Postgraduate Medicine, 1976, 59, No. 5, 103-107.

Frobisher, M. Fundamentals of Microbiology. Philadelphia: W.B. Saunders Co., 1963, 7th Ed.

Gallucci, B.B. & Rebeis, C.E. Infection, nutrition, and the compromised patient. Topics in Clinical Nursing, 1979, 1, No. 2, 21-28.

Green, G. In defense of the lung. American Review of Respiratory Disease, 1970, 102, 691-703.

Green, G., Jakab, G., Low, R., & Davis, G. Defense mechanisms of the respiratory membrane. American Review of Respiratory Disease, 1977, 115, 479-484.

Greico, M. Infections in the Abnormal Host, New York, York Medical Books, 1980.

Grieble, H., Colton, M., Bird, T, Toigo, A., & Griffith, L. Fine particle humidifiers: Source of Pseudomonas aeruginosa infections in a respiratory unit, New England Journal of Medicine, 1970, 282, No. 10, 531-534.

Goodman, L.S. & Gilman, A. The Pharmacological Basis of Therapeutics. New York: MacMillan Publishing Co., 1975.

Guyton, A.C. Textbook of Physiology. Philadelphia, W.B. Saunders Company, 5th Ed., 1976.

Haas, H., Morris, J., Samson, S., Kilburn, J. & Kim, P. Bacterial flora of the respiratory tract in chronic bronchitis: Comparison transtracheal fiberbochscopic and oropharngeal sampling methods. American Review of Respiratory Disease, 1977, 116, No. 1, 41-47.

Harris, T., Richards, W. & Blake, J. An evaluation of bacterial contamination of ventilator humidifying systems. Chest, 1973, 63, No. 3, 922-925.

Hinshaw, H., & Murray, J. Diseases of the Chest. Philadelphia: W.B. Saunders Company, 1980.

Hoeprich, P. Infectious Disease. Hagerstown, Maryland: Harper and Row Publishers, 1972, 2nd Ed. 1977.

Hodgkin, J., Balchum, O., Koss, I., Glaser, E., Miller, W., Haas, A., Shaw, D., Kimbel, P., & Petty, T. Chronic obstructive airway disease: Current concepts in diagnosis and comprehensive care. Journal of American Medical Association, 1975, 332, No. 12, 1243-1258.

Hudson, L., Tyler, M., & Petty, T. Hospitalization needs during an outpatient rehabilitation program for severe C.O.P.D. Chest, 1976, 70, 606-610.

Hyde, E.A., Moore, D., & Higgins, B.D. A survey of fungal flora in respiratory equipment. Respiratory Care, 1979, 24, 921-927.

Jackson, A.E., Southern, P.M., Pierce, A.K., Fallis, B.D., & Sanford, J.P. Pulmonary clearance of gram-negative bacilli. Journal of Laboratory and Clinical Medicine, 1967, 69, 833-841.

Johanson, W.G., & Gould, K.G. Lung defense mechanisms, Basics of Respiratory Disease, 1977, 6, No. 2, 66-71.

Johanson, W.G., Pierce, A.K., Sanford, J.P., Thomas, G.D. Nosocomial respiratory infections with gram-negative bacilli. Annals of Internal Medicine, 1972, 77, 701-706.

Johanson, W.G., Pierce, A.K., Sanford, J.P. Changing pharyngeal bacterial flora of hospitalized patients. The New England Journal of Medicine, 1969, 281, No. 21, 1137-1140.

Karr, R.M. Mechanisms of Immune Injury. Chest, 198, 78, No. 2, 388-392.

Kelsen, S.G., McGuckin, M., Kelsen, D.P., & Cherniack, N. Airborne contamination of fine-particle nebulizers. Journal of American Medical Association, 1977, 237, 2311-2314.

Koss, J.A., Conine, T.A., Eitzen, H.E., & LoSasso, A.M. Bacterial contamination potential of sterile, prefilled humidifiers and nebulizer reservoirs. Heart and Lung, 1979, 8, No. 6, 1117-1121.

Laforce, F., Hopkins, J., Trow, R., & Wong, W. Human oral defenses against gram-negative rods. American Review of Respiratory Disease, 1976, 114, 929-935.

Lefcoe, N. & Patterson, N. Adjunct therapy in chronic obstrucive pulmonary disease. American Jouranl of Medicine, 1973, 54, 343-350.

Litsky, W., Botko, G.J. & Litsky, B.Y. Evaluation of decontamination procedures and subsequent contamination of I.P.P.B. setups. Respiratory Care, 1975, 20, 137-144.

Lepper, M.H. Opportunistic gram-negative rod pulmonary infections. Diseases of the Chest, 1963, 44, 18-25.

Lertzman, M., & Cherniack, R. Rehabilitation of patients with chronic obstructive pulmonary disease: State of the art, American Review of Respiratory Disease, 1976, 114, 1145-1165.

MacDonnell, K. & Segal, M. Predispositions to pulmonary infections. Current Respiratory Care, 1977, 77, 169-190.

MacKowiak, P., Martin, R., Jones, S. & Smith, J. Pharngeal colonization by gram-negative bacilli in aspiration prone persons. Archives of Internal Medicine, 1978, 138, 1224-1227.

Matthay, R.A., Greene, W.H. Pulmonary infections in the immunocompromised patient. Medical Clinics of North America, 1980, 64, No. 3, 529-551.

Mays, E. Inhalation therapy bacteria: Study of decontamination by trained technicians versus ward personnel. Respiratory Care. 1973, 138, No. 4, 219-221.

Miller, W.F. Aerosol therapy in acute and chronic respiratory disease. Archives of Internal Medicine, 1973, 131, 148-155.

Muir, D.C. Physiological aspects of ultrasonic aerosols. Respiration, 1970, 27, Suppl., 185-190.

Murray, J.E. The Normal Lung: The Basis for Diagnosis and Treatment of Pulmonary Disease. Philadelphia: W.B. Saunders Company, 1976.

Nazemi, M.M., Musher, D.M. & Martin, R.R. A practical method for monitoring bacterial contamiantion of inhalation therapy machines. American Review of Respiratory Disease, 1972, 106, 920-922.

Paez, P.N. Decontamination of respiratory therapy equipment. Journal of American Medical Association, 1977, 238, No. 4, 303.

Parmley, J., Tahir, A., Dascomb, H. & Adriani, J. Disposable versus reusable breathing circuits: Advantages, disadvantages, hazards and bacteriologic studies. Anesthesia and Analgesia, 1972, 51, 888-894.

Parker, R., & Hoeprich, P. In vitro effect of buffered solutions of acetic acid, triclobisonium chloride, chlorohexidine digluconate on urinary tract pathogenes, Antimicrobial Agents and Chemotherapy, 1972, 26-34.

Pavia, D., Thomson, M., Clarke, S. Enhanced clearance of secretions from the human lung after the administration of hypertonic saline aerosol. American Review of Respiratory Disease, 1978, 117, 199-203.

Pavia, D., Thomson, M., Clarke, S., & Shannon, H. Effect of lung function and mode of inhalation on penetration of aerosol into the human lung. Thorax, 1977, 32, 194-197.

Peterson, N.J., Carson, L.A., Favero, M.S., Marshall, J.H., Bond, W.S. Microbial contamination of mist therapy units on six pediatric wards. Health Laboratory Science, 1975, 12, 41-46.

Phillips, I., & Spencer, G. Pseudomonas aeruginosa cross-infection due to contaminated respiratory apparatus. The Lancet, 1965, 1325-1327.

Pierce, A.K. and Sanford, J.P. Bacterial contamination of airesole. Archives of Internal Medicine, 1973, 131, 156-159.

Pierce, A.K., Sanford, J.P., Thomas, G.D., & Leonard, J.S. Long-term evaluation of decontamination of inhalation therapy equipment and the occurrence of necrotizing pneumonia. New England Journal of Medicine, 1970, 282, No. 10, 528-531.

Pierce, A.K., Edmonson, E.B., McGee, G., Ketchersid, J., Loudon, R.G., & Sanford, J.P. An analysis of factors predisposing to gram-negative bacillary necrotizing pneumonia. American Review of Respiratory Disease, 1966, 94, 309-315.

Petty, T. Does treatment for severe emphysema and chronic bronchitis really help? Chest, 1974, 65, 124-127.

Petty, T.L. Reduced infection hazards during mechanical ventilation. Chest, 1970, 58, No. 4, 317-318.

Reinarz, J.A. Gram-negative pneumonia and inhalation therapy. Clinical Symposia, 1978, 30, No. 6, 16-19.

Reinarz, J.A., Pierce, A.K., Mays, B.B., & Sanford, J.P. The potential role of inhalation therapy equipment in nosocomial pulmonary infections. Journal of Clinical Investigation, 1965, 44, 831-839.

Rippon, J.W. Pathogenesis and epidemiology of opportunistic mycotic infections: A review. American Journal of Medical Technology, 1977, 43, No. 3, 226-228.

Roberts, J., Cockcroft, W.H., Jonson, E., & Fishwick, T. The infection hazard of contaminated nebulizers. Canadian Medical Association Journal, 1973, 108, 53-56.

Roberts-Thomson, I.C., Whittingham, S., Youngchaiyud, U., & MacKay, I. Ageing, immune response, and mortality. The Lancet, 1974, 2, 368-370.

Ryan, K.J. & Mihalyi, S.F. Evaluation of a simple device for bacteriological sampling of respiratory-generated aeresols. Journal of Clinical Microbiology, 1977, 5, 178-183.

Sanders, D., Luby, J., Johanson, W., Barnett, J., & Sanford, J.P. Serratia Marcescens infectious from inhalation therapy medications: Nosocomial breakout. Annals of Internal Medicine, 1970, 73, 15-21.

Sanders, C., Sanders, W., & Harrowe, D. Bacterial interference: Effects of oral antibiotics on the normal flora and its ability to interfere with Group A Streptococci, Infection and Immunity, 1976, 13, No. 3, 808-812.

Sanford, J.P. Disinfectants that don't. Annals of Internal Medicine, 1970, 72, No. 2, 282-283.

Solomon, W.R. Fungus aerosols arising from cold-mist vaporizers. Journal of Allergy Clinical Immunology, 1974, 54, No. 4, 222-228.

Statement American Thoraic Society: American Review of Respiratory Disease, 1977, 115, No. 5, 893-895.

Stark, C. Sterilization by chemical agents. International Anesthesia Clinics, 1972, 10, No. 2, 49-65.

Sykes, M.K. Sterilization of ventilators: Infections and sterilization problems. International Anesthesia Clinics, 1972, 10, No. 2, 131-145.

Valenti, W.M., Trudell, R.G., Bentley, D.W. Factors predispose to orophryngeal colonization with gram-negative bacilli in the aged. New England Journal of Medicine, 1978, 298, No. 20, 1108-1111.

Warr, G., Martin, R., Sharp, P., & Rossen, R. Normal human bronchial immunoglobulins and proteins - effects of cigarrete smoking. American Review of Respiratory Disease, 1977, 116, No. 1, 25-30.

Weksler, M. & Hutteroth, T. Impaired lymphocyte function in aged humans. The Journal of Clinical Investigation, 1974, 53, 99-104.

Welch, M., Shapiro, B., Mercurio, P., Wagner, W., & Hirayama, G. Methods of intermittent positive pressure breathing. Chest, 1980, 78, No. 3, 463-467.

Williams, D., Kirek, J., Remington. Pulmonary infection in the compromised host. American Review of Respiratory Disease, 1976, 114, No. 2, 359-394.

Zeluff, G., Deaton, W., & Jackson, D. What good is respiratory therapy. Heart and Lung, 1978, 7, No. 4, 659-666.

Patient:_____1_____

Date:_____

Demographic Profile

1. Age_____
2. Sex_____
3. Disease_____
4. Length of diagnosis_____
5. Medications_____

_____

_____

_____

6. Antibiotics: Most recent antibiotic_____Duration_____

Prior antibiotic_____Duration_____

7. Most recent Hospitalization_____
8. How do you clean your equipment_____

frequency_____

solutions_____

tubings_____

disposalbe/nondisposable tubings_____

9. Do you clean your own equipment_____
10. What is the name of the machine you use_____-_____
11. How often do you take treatments in a day_____
12. Do you use tap water or saline in the nebulizer_____
13. How long have you used your machine at home_____

Patient: 11

Date: _____

Patient Symptom History

Cough:

a. None - I do not have a cough.

b. Mild - I cough only in the morning and have little difficulty with coughing during the day.

c. Moderate - I cough in the morning with episodes of coughing during the day requiring rest and interfering with daily activities.

d. Severe - I cough throughout the day as well as at night Coughing may cause me to have chest pain, dizziness, or unsteadiness.

Sputum:

a. None - I do not produce sputum.

b. Mild - I produce sputum mostly in the morning usually less than ¼ cup.

c. Moderate - I produce sputum throughout the day and it is usually ½ to ¼ cup per day.

d. Severe - I produce sputum throughout the day greater than ½ cup per day.

Shortness of Breath:

a. None - I have no restrictions of normal activities.

b. Mild - I have shortness of breath when walking stairs or on an incline, but not on level ground.

c. Moderate - I get short of breath when walking/minimal exertion and with routine daily activities.

d. Severe - I am short of breath at rest as well as with any activity.

Wheezing:

a. None - I never wheeze.

b. Mild - I have no wheezing at rest, but I occasionally wheeze with moderate exercise, or wheeze at night.

c. Moderate - I wheeze with most daily activities and with minimal exercise.

d. Severe - I wheeze at rest.

Patient: iia

Date: _____

Patient Symptom History

Fluid Retention:

a. None - My ankles never swell.

b. Mild - My ankles swell after I stand or sit for a long time.

c. Moderate - My ankles swell when I stand or sit and get worse as the day goes on. The swelling goes away at night.

d. Severe - My ankles get more swollen as the day goes on, and the swelling usually does not completely go away at night.

Color of Sputum:

a. Clear - It is clear and thin.

b. White - It is white and thick to spit out.

c. Yellow - It is thick, sticky and is even harder to spit out.

D. Green - It is very thick and sticky and I have trouble expectorating it and I have more of it.

Exercise:

a. None - My breathing is the same.

b. Mild - I am having more trouble breathing when I do my activities of daily living.

c. Moderate - I become short of breath with the slightest movement and it is hard for me to do my activities of daily living.

d. Severe - I am short of breath at rest and have to sit up at night in order to breath.

Cleaning Record

Subject _____

Date	Morning	Noon	Afternoon	Evening

Instructions: **Write out exactly what parts of the equipment you cleaned and how you did it. Name the solution you used and when you washed the tubings. For those times when you do not clean any parts of the equipment place a zero under the appropriate time.**

POSTTEST

Patient: _____

Date: _____

Symptom History:	none	mild	mod.	sev.
1. Cough	____	____	____	____
2. Sputum	____	____	____	____
3. Shortness of breath	____	____	____	____
4. Wheezing	____	____	____	____
5. Fluid retention	____	____	____	____

6. How many cigarettes does the patient usually smoke per day? _____

Physical Examination:

1. Blood pressure (with patient standing) (mmHg)
   Systolic _____
   Diastolic _____
2. Respiratory rate/min _____
3. Apical rate/min _____

Pulmonary:	No	Yes
4. Does the patient use the accessory neck muscles for quiet breathing?	___	___
5. Does the patient have rales?	___	____
If yes, are they localized?	___	____
6. Does the patient have wheezes on quiet breathing?	____	____
7. Does the patient have decreased breath sounds?	____	____
Cardiac:		
8. Is the rhythm regular?	____	____

Other:

1. Has the color of your sputum changed _____

   color _____

2. Do you have shortness of breath with exercise _____

UNIVERSITY OF CALIFORNIA, SAN FRANCISCO

Consent to be a Research Subject

A) Grace Hardie, a master's student in the cardio-pulmonary nurse specialist program at UCSF, is doing a study about the relationship between the way people clean their inhalation equipment and how clean the equipment actually is. It is hoped this study may help discover a more effective and simpler method of cleaning inhalation equipment than is presently used. Because I take inhalation treatments in my home, I have been asked to participate in this study.

B) If I agree to be in the study, the following will happen.

1) I will be randomly assigned to one of two study groups. This means I have a 50/50 chance of being in either group. Group A will receive a teaching cleaning program while Group B will continue to clean their equipment as they have been doing prior to joining the study.

2) I will be interviewed for about one hour, complete a brief questionnaire, and answer questions about my lung problem. This will be done once at the beginning of the study, and once at the end (10-14 days later).

3) I will be asked to keep a log of my equipment cleaning procedure during this time.

4) A sample of the air from the large tubing on my inhalation equipment will be taken to study the cleanliness of my equipment. This will take about 15 minutes, and will be done once or twice, in my home.

5) During the visit(s) to take the air sample, Grace Hardie will also listen to my heart and lungs.

C) Being in this study may have some risks such as assignment to the group that is later shown to be less effective (have less effective cleaning techniques), or to the group that is no more effective but involves more time and inconvenience on my part. However, this information will not be known until the study is over.

D) The study may have the benefits of showing a better way of cleaning home inhalation equipment, and thus avoid getting infections from contaminated equipment. This could help me and other people who use inhalation equipment at home in the future.

E) I have had the opportunity to talk with Grace Hardie about this study. If I have further questions, I may call her at 654-1620.

F) I have received a copy of this form and of the Experimental Subject's Bill of Rights to keep.

G) Participation in research is voluntary. I have the right to refuse to participate or to withdraw from the study at any time without jeopardy to my continued treatment. I just have to say so.

_____ _____

Date Subject's Signature

Human Subjects Protocol No. 940601-01

7/30/80

UNIVERSITY OF CALIFORNIA, SAN FRANCISCO

EXPERIMENTAL SUBJECT'S BILL OF RIGHTS

The rights below are the rights of every person who is asked to be in a research study. As an experimental subject I have the following rights:

1) **To be told what the study is trying to find out,**

2) **To be told what will happen to me and whether any of the procedures, drugs, or devices is different from what would be used in standard practice,**

3) **To be told about the frequent and/or important risks, side effects or discomforts of the things that will happen to me for research purposes,**

4) **To be told if I can expect any benefit from participating and, if so, what the benefit might be,**

5) **To be told the other choices I have and how they may be better or worse than being in the study,**

6) **To be allowed to ask any questions concerning the study both before agreeing to be involved and during the course of the study,**

7) **To be told what sort of medical treatment is available if any complications arise,**

8) **To refuse to participate at all or to change my mind about participation after the study is started. This decision will not affect my right to receive the care I would receive if I were not in the study.**

9) **To receive a copy of the signed and dated consent form,**

10) **To be free of pressure when considering whether I wish to agree to be in the study.**

If I have other questions I should ask the researcher or the research assistant. In addition, I may contact the Committee on Human Research, which is concerned with protection of volunteers in research projects. I may reach the committee office by calling: (415) 666-1814 from 8:00 AM to 5:00 PM, Monday to Friday, or by writing to the Committee on Human Research, University of California, San Francisco, CA 94143.

Call X1814 for information on translations.

Cleaning Methods Prior to Study

Sample study undifferentiated by groups:

1. Type of dishsoap used in cleaning:

   A. Tap water rinse used by 27.6% of study sample
   B. Ivory dishsoap used by 27.8% of study sample
   C. Joy dishsoap used by 38.9%.

2. Frequency of cleaning tubings.

   A. Had never washed the tubings —61.1%.
   B. Washed the tubings every month—11.1%
   C. Washed the tubings every 1 or 2 weeks—16.7%
   D. Washed the tubings every week—5.6%
   E. Washed the tubings at least twice a week—5.6%

3. Used vinegar and water before study.

   A. Had used it before the study—83.3%
   B. Had never used the solution—16.7% (used as a rinse)

4. Strength of dilution of vinegar and water.

   A. Low effectiveness (less than .10% solution)—77.8%
   B. Moderate (adequate) 0.20-0.25% solution—16.7%
   C. Did not use any solution—5.6%

5. Length of soaking in the vinegar and water solution.

   A. Not at all—5.6%
   B. Rinse only—11.1%
   C. A 10 minute soaking—22.2%
   D. A 20 minute soaking—38.9%
   E. A 30 minute soaking—22.2%

6. Allowed the equipment to drip dry.

   A. Allowed it to drip dry—83.3%
   B. Toweled dried it—16.7%

7. Dismantle the manifold and tubings before cleaning.

   A. Did the dismantling—33.3%
   B. Did not dismantle any of the equipment—66.7%

8. Frequency of washing nebulizer.

   A. Washed the nebulizer at least once a day—88.9%
   B. Did not wash nebulizer daily—11.1%.