from water, are DNA, acid mucoprotein and fibrin. Sputum mucoprotein structure can crudely be shown as disulfide and hydrogen bond linked proteins joined by calcium sulfate and ammonium bonds to a column of saccharides with a molecule of fucose on one end and sialic acid molecule on the other end. By far, the largest part of this molecule is its protein moiety which also is the most treatable part in that its disulfide bonds are ruptured by acetylcysteine. Further illustration of the usefulness of attacking the protein moiety of the mucopolysaccharide is the rapid lysis and decrease of viscosity of viscous sputum brought about by trypsin digestion.

Because of the relative unsuitability of previous methods in the clinical measurement of sputum viscosity, we have developed the following method which has been highly satisfactory for us. The fluid consisto-viscosimeter was designed in our laboratory to measure the consistency of heterogenous semi-plastic materials such as sputum. The device consists of hollow stainless steel plunger with a perforated disc at its lower end which is driven by a constant infusion pump through a close fitting barrel filled with sputum. The bottom of the cylinder is fitted with a transducer which reflects the pressure generated as the sputum is forced through the perforated disc. This pressure is directly proportional to the sputum consistency, which is expressed in “consistency units.” The thicker the sputum, the greater the number of consistency units. One consistency unit is equivalent to 1,500 centistokes (the standard units of kinematic viscosity).

We have recently completed a double-blind crossover study of the effect of glyceryl guaiacolate on chronic bronchitis and have shown that it does not affect ventilatory capacity or the clinical course of the disease. Furthermore, glyceryl guaiacolate does not increase the ease of expectoration or alter the consistency or volume of the sputum. A similar double-blind crossover study, using potassium iodide, likewise showed no change in ventilatory capacity, nitrogen washout, or clinical course in patients with chronic bronchitis. Sputum consistency, sputum volume and ease of expectoration showed no consistent differences in patients given iodide or placebo. In summary, the most effective agents we have at this time for reducing sputum viscosity are N-acetylcysteine and water.

Dr. Salvaggio: Does N-acetylcysteine perhaps alter or destroy immunoglobulins in view of the fact that it ruptures disulfide bonds? Could it increase the chance for infection by altering “protective” secretory IgA for example?

Dr. Hirsch: I don’t know.

Bronchopulmonary Lavage in Bronchial Asthma*

Robert M. Rogers, M.D., John F. Shuman, M.D., and Alan B. Zubrow, B.A.

Airway obstruction in bronchial asthma during an acute attack is predominantly due to a bronchospasm. However, as the attack persists, edema of the bronchial mucosa and thick tenacious mucus in the airway lumen contribute to airway obstruction. The thick mucus may contribute to the morbidity and mortality of status asthmaticus by causing atelectasis which leads to severe hypoxemia. In addition, the mucus may serve as a depository of an antigen which continues to stimulate airways obstruction when it cannot be removed by cough or other means—ie, aspergillosis. The purpose of this report is to review our experience with bronchopulmonary lavage in seven asthmatic patients.

The technique of lavage has been described in detail elsewhere.1 Simply, it is accomplished by alternately filling and emptying one lung while the nonlavaged lung is used to maintain gas exchange.

Bronchopulmonary lavage in asthmatic patients offers several unique technical problems: 1) high airway resistance decreases flow of fluid so that the procedure takes considerably longer; 2) inhalation anesthesia induction is prolonged because of decreased gas exchange; 3) manipulation of the airway with a Carlenes tube may induce further bronchospasm which must be treated in the context of lavage; 4) ventilation of the nonlavaged lung may require high pressure making adequate alveolar ventilation difficult to achieve, so that CO2 retention during the procedure is common; 5) more fluid remains in the lung at the end of lavage in asthmatics (mean = 1340 ml) than in patients with alveolar proteinosis (mean = 740 ml).

To obviate some of these problems, we usually give IV aminophylline three hours prior to lavage, give IPPB with a bronchodilator immediately before induction of anesthesia, apply local anesthesia to vocal cords and trachea, give IV aminophylline.

*From the Departments of Medicine and Physiology, Hospital of the University of Pennsylvania, Philadelphia.

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or isoproterenol (Isuprel) during the procedure if bronchospasm worsens, use an anesthetic agent that is readily eliminated to avoid post-lavage CNS depression, use an IPPB machine to ventilate the nonlavaged lung during lavage, monitor blood gases carefully, and give ventilatory support post-lavage until the patient is fully alert.

Seven patients underwent lavage, four because of intractable status asthmaticus unresponsive to conventional therapy, and three because of chronic asthma which was only controlled by high doses of steroids for prolonged periods of time. The only major complication was laryngeal edema which developed in one patient two days postlavage and required tracheostomy. Pulmonary infiltrates and fever developed in two patients and responded promptly to antibiotic therapy. Most patients were able to be extubated immediately following the procedure; however, in four of 12 lavages, intubation and mechanical ventilation were required for 12 to 24 hours postlavage. Mucus plugs were removed in all instances, and these varied from the size of a segmental bronchus to that of a respiratory bronchiole. These mucus plugs contained alveolar macrophages, eosinophils and Charcot-Leyden crystals, and the smaller ones fitted the description of Curschmann's spirals. There was usually an initial decline in vital capacity and FEV₁ in the first 12-24 hours, with subsequent improvement accompanied by a decrease or absence of wheezes on the lavaged side, and persistence of wheezes on the nonlavaged side, a decrease in peak pressure on the ventilator and rapid weaning from same, and in some instances, rapid reduction in the amount of medication required to control the wheezing. The improvement of one patient is documented in Figure 1. After lavage of each lung, the wheezes in that lung disappeared and the patient was off all medication for ten months, at which point he developed another attack requiring hospitalization. Steroids were withheld and lavage was performed on one lung with subsequent significant clinical deterioration which responded to steroids. He has remained on steroids since that time. In other instances, high doses of steroids were reduced to more manageable levels, and the number of hospitalizations for acute attacks was reduced. In the chronic asthmatics, there were no noticeable changes in their overall clinical picture.

We conclude that bronchopulmonary lavage in asthma has a definite, although limited, role in the treatment of status asthmaticus which is unresponsive to the conventional forms of therapy. Its role in chronic airways obstruction is yet to be determined, and will require carefully controlled studies.

**Reference**

Discussion

Dr. Long: Dr. Rogers, would you also visualize lavage as a vehicle for drug administration?

Dr. Rogers: We use lavage only as a lung "enema." The addition of drugs to the protocol would add another parameter and thereby further confuse the value of lavage as a treatment by itself.

Dr. Ellis: Dr. Hirsch, what is known about the efficacy of the mist tents? Recent studies have shown that most water produced by these tents ends up in the pharynx or the stomach and almost none in the lower airway.

Dr. Hirsch: Usually I don't use a mist tent because it doesn't help acute asthmatics breathe easier. One of the problems associated with the inhalation of water mists is that the absorption of water by mucus results in an increased volume before its consistency decreases and this can transiently lead to increased obstruction of the upper bronchi. The prevention of this situation is to first increase the size of the bronchial lumen with bronchodilators and then to use water.

Dr. Lyons: Dr. Rogers, can lung lavage decrease plasma urea and affect electrolytes to a significant degree? Do hemorrhagic changes occur in the lung as in lavaged lungs of rats?

Dr. Rogers: Lung lavage has been shown to be relatively ineffective in removing urea. We have found no hemorrhage in the lungs of our patients. The patients with alveolar proteinosis who were lavaged did show a loss of surfactant activity, although this was only transient. Much more surfactant would be removed as bubbles present in the lavage solution so we attempt to avoid them.

Dr. Nadel: Dr. Hirsch, why is hydration effective as a treatment if it causes such a small change in sputum consistency?

Dr. Hirsch: Water is effective mainly because it can be used parenterally or by inhalation in relatively large amounts.

Dr. Townley: Is N-acetylcysteine as effective with nonpurulent sputum as it is with purulent sputum?

Dr. Hirsch: Evaluation of sputum viscosity with the fluid consisto-viscosimeter has shown N-acetylcysteine to be equally effective in reducing the viscosity of purulent and mucoid sputum.

Dr. Chodosh: I cannot let this section end with the impression that glycercyl guaiacolate is inactive. Glycercyl guaiacolate is not a useless drug. In our study, patients who have been on a large dose for seven days did show improvement in the ventilatory capacity, their subjective clinical status and in the physical properties of their sputum.

SESSION XI: FEATURED PAPERS

Asthma without Wheezing*

Richard S. Farr, M.D.; Michael T. Kopetzky, M.D.; Sheldon L. Spector, M.D.; and David S. Hurewitz, M.D.

Asthma can be defined as reversible obstructive airway disease. When patients are symptomatic auscultation of the chest usually reveals expiratory and sometimes inspiratory wheezing or rhonchi. In addition, during an attack they usually have a reduced forced vital capacity (FVC), and the one-second forced expiratory volume (FEV1) is less than 75-80 percent of the FVC. If the attack is severe enough, blood gas determinations will reveal hypoxia.

CASE REPORT

We have studied an unusual 19-year-old woman who had intermittent typical bouts of asthma associated with respiratory tract infections, but between attacks she complained of tightness of the chest relieved by oral or aerosolized bronchodilators. The tightness of the chest occurred at times when her FVC, FEV1, blood gases, and physical examination of her chest were normal. Although this case simulated bouts of hysteria between actual asthma attacks, whole body plethysmograph data revealed some remarkable values. She had an essentially normal FVC, FEV1, inspiratory capacity (IC), expiratory reserve volume (ERV), residual volume (RV), and total lung capacity (TLC). However, her true total lung capacity (TTLC) was increased because of a trapped air volume (TAV) as high as 2.65 liters. Her airway resistance (Rw) was also elevated to as high as 14.0 cm H2O/L/sec. These abnormal findings were largely reversible following the inhalation of aerosolized bronchodilators, including atropine.

METHODS

The subject was 158 cm tall, weighing 67 kg (ideal weight: 53 kg). VC, IC, and ERV were determined by...