Humidification of the Airways Adequate for Function and Integrity?

The upper respiratory tract serves as an air conditioning system; particulate matter is entrapped while inspired air is warmed and humidified prior to entry into the major airways. Turbulent flow through the nose and pharynx propels solid particles towards the surface, enhances convective transfer of heat from the mucosa, and increases the apparent surface area of exposure for evaporation of water vapor into the air stream. Because the nasal mucosa becomes cooled in the process, exhaled gas, which is fully humidified at body temperature, gives up water vapor to the cooler surface and the effluent gas temperature is reduced to 32-34°C. Fully saturated air at this temperature contains 10.2 mg/L less water vapor than alveolar gas. Water and heat recapture by this mechanism conserves 20-25 percent of insensible losses during normal breathing in temperate atmospheric conditions. The administration of relatively dry anesthetic gases and oxygen-enriched mixtures via an endotracheal tube obliges the lower respiratory tract to perform these functions. The potential hazard of greater heat and water losses, as well as structural damage was appreciated early in tracheotomized patients, and external application of humidity was demonstrated to alleviate excess mucus production and encrustation of secretions.

Much subsequent study has revealed the sensitivity of the tracheobronchial tree to incompletely humidified gas. Cessation of ciliary beating occurs within ten minutes of exposure of the trachea to 50 percent relative humidity. Tracheal mucus velocity, studied in intact dogs, was reduced by half after one hour of breathing desiccated room air and completely ceased after three hours. Subsequent humidification of the airway rapidly restored tracheal mucus velocity, but histologic evidence demonstrated that extensive inflammation and sloughing of ciliated epithelium in the trachea and bronchi were present despite the functional restoration observed.

Cytologic studies in anesthetized human subjects confirmed that damage to the tracheal epithelium occurs within two hours of administration of dry gases through an endotracheal tube, whereas gases at 60 percent relative humidity produce no damage. However, air at 60 percent relative humidity delivered through an endotracheal tube reaches only 83 percent of full humidification at body temperature at the carina, and is not fully saturated until it reaches 10 cm further below this point. Thus, although confirmation is uniform that relative humidities of 60-70 percent do not affect ciliary function for up to three hours, and that the lower airways continue to warm and humidify inspired gas, the structural response of the peripheral airways during prolonged periods of incomplete humidification remains unknown.

The hygroscopic condenser humidifier reproduces the action of the nasopharynx to partially recapture exhaled heat and humidity and donates this retained moisture to the next inspired tidal volume. The nasopharynx increases its efficiency under colder and drier conditions, maintaining full humidification of inspired gas despite ambient temperatures down to $-100^\circ$C. By contrast, delivery of larger tidal volumes and higher inspired oxygen concentrations through the hygroscopic condenser humidifier reduces inspired humidity.

The study presented by MacIntyre and co-authors (see page 560) is a valuable clinical evaluation of this device and demonstrates that mucociliary activity is preserved for periods longer than have been evaluated previously. Further histologic investigation must ensure that the potential for histologic damage to subcari-
was that first reported by Timpe and Runyon who presented a tentative grouping of "atypical" organisms with a preliminary analysis of their relationship to human pulmonary disease. Their classification utilized pigment production, colony morphology and growth rates as differentiating features. While withstanding the test of time, the Runyon classification remains complex, involved, and nonclinical. It is descriptive, but is of limited relevance, especially to the clinician caring for the patient. Other classifications utilizing newer methods of identification (lipid analysis and serotypes) have been proposed, but these too are technical and not clinical.

The classification proposed by Dr. William Bailey in this issue of Chest (see page 625), approaches the numerous mycobacterial pathogens in a strictly clinical manner, based upon their treatment characteristics. This classification system divides the pathogens into broad classes: (1) nonpathogens; (2) easy to treat, standard mycobacterial therapy; and (3) difficult to treat, nonstandard mycobacterial therapy. (Nonpathogens, hard-to-treat pathogens, and easy-to-treat pathogens are distributed fairly evenly throughout the Runyon system, since their growth characteristics are not necessarily related to clinical characteristics.)

It is obvious that the Bailey classification itself is simple, reasonable, and directly imparts clinical information about these bacteria. With the increasing inclusion of the nonspecialist in the care and control of these diseases, the Bailey classification may prove to be an important tool in enhancing treatment of disease caused by these mycobacteria.

While we are on the subject of classification, a sorely needed next obvious step is to look at the entire family of mycobacteria. Use of "nontuberculous mycobacteria," "mycobacteria other than tuberculosis" or "atypical mycobacteria" is inconsistent, inaccurate and grossly displeasing. Can the physicians and scientists who have shortened to a period of six to nine months the treatment of diseases caused by several of these organisms get together with a name for the major groups? Francis and Abrahams suggestion of four groups consisting of: tubercle bacilli; tuberculoid bacilli; saprophytic mycobacteria; and leprosy might be a place to start.

ACKNOWLEDGMENT: The authors wish to thank Dr. David Glasser for calling our attention to the Francis and Abrahams classification.

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A New Classification for Some Mycobacteria

All classifications are ephemeral; they are useful only as long as they serve their purpose.1

In the last several years, two major developments have occurred in tuberculosis care and control. One has been the movement of tuberculosis into the mainstream of medicine.8 It is no longer uncommon for routine care of patients with tuberculosis to rest with the primary care physician with consultative advice from a specialist.9 The other has been the gradual fall of rates of disease due to M tuberculosis. Consequently, significant disease caused by other mycobacteria has gained in importance. More recently, attention has been drawn to the prevalence of disease caused by other mycobacteria within the spectrum of the acquired immune deficiency syndrome (AIDS).4

When one looks at and puts these two developments together, one realizes that pulmonary physicians and infective disease specialists are not the only physicians treating mycobacterial disease. With the consideration that mycobacterial diseases are being diagnosed and treated by an increasing number of nonpulmonary and noninfectious disease specialists comes the inescapable fact that in order to communicate about these conditions, a clear, functional way of describing them (ie, classification) is needed.

Until now, the most frequently used classification

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