The Nose and Obstructive Sleep Apnea

The nose is properly considered a part of the upper airway. The nasal alae demonstrate inspiratory bursts of activity resulting in nasal flaring, which is precisely timed to precede diaphragm activity.1 Rhinomanometry has been successfully employed to study nasal airflow. Using this, Bridger and Procter2 showed that almost all of the nasal resistance to airflow is produced in the anterior 2 to 3 cm, and that reducing resistance at the nostril has beneficial effects on airflow. Conversely, when nasal resistance is high, a higher total pressure drop is required,3 and collapse of the upper airway, specifically the pharyngeal airway, is facilitated. Just as pathologic narrowing of the pharyngeal airway may induce sleep-related impaired breathing,4 impairment of nasal breathing has been noted to result in obstructive sleep apnea,4 and treatment of nasal pathology in some instances has been beneficial.4,5

Although rhinomanometry has been used to study nasal pathology, Shepard and Burger6 introduced a more clinically useful method—nasal flow volume loops (FVLs). They showed that the nose behaved like a variable resistor and that the area under the nasal FVLs contributed to the prediction of severity of obstructive sleep apnea. From this he suggested that limitations to ventilation via the nasopharynx may significantly influence the frequency of sleep-disordered breathing.

From these observations it is reasonable to speculate that mechanical dilatation of the anterior nares might have an impact on nasal resistance and airflow, and consequently on sleep apnea. A nasal dilator that increases the cross-sectional area of the nasal valve region has been shown in a group of healthy volunteers to improve nasal airflow.7 In another study,8 the benefit was seen only in a subgroup, specifically, those with a nasal FVL that exhibited a pattern of variable extra-thoracic obstruction, while those with a fixed pattern of obstruction had no improvement in flows. Subsequently, this device was shown to decrease the frequency and severity of obstructed breathing events and snoring in a group of patients with obstructive sleep apnea,9 and was used to quiet the world’s loudest snorer (London Sunday Express, February 16, 1991 p1). This individual had been reported in the Guinness Book of World Records to produce a sound level of “87.5 decibels at a distance of one meter from the recorder,” and it was noted that his “wife Julie is deaf in one ear.”10 In addition, external rhinoplasty with elevation of the nose tip has been used to increase the cross-sectional area of the nares with subsequent improvement in respiratory disturbance during sleep.11

The importance of the anterior nasal airway in the development or modification of sleep apnea is highlighted by these experiences. The convenience of recording FVLs to assess nasal flow makes them particularly valuable, though, surprisingly, to our knowledge no additional data have been published. Since the problem of snoring, and frequently of accompanying sleep apnea, is significant, we believe that more attention could usefully be paid to the evaluation and improvement of nasal airflow.

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Mortality and Ventilator-associated Pneumonia

The Best Antibiotics May Be the Least Antibiotics

Ventilator-associated pneumonia (VAP) is considered to increase the risk of death in mechanically ventilated patients. Although multiple studies demonstrate greater mortality rates in patients with VAP than in those without,1,2 the only investigation using multivariate analysis found that VAP was not an independent risk factor for death in mechanically ventilated patients.3,4

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ventilated patients.\textsuperscript{1} Despite this, the gravity of complications from VAP is considered worrisome enough to warrant expensive and elaborate efforts aimed at its prevention. Although immunotherapy and a variety of other techniques have been utilized, prophylactic antibiotics, given either parenterally,\textsuperscript{4} topically,\textsuperscript{5,6} or in combination,\textsuperscript{7-10} have been the most extensively investigated.

Much of the information on VAP is compromised by the assumption that all cases of VAP can be combined, as if they had similar risk factors, pathogenesis, and prognosis. Langer and colleagues\textsuperscript{11} tried to demonstrate that at least one subgroup of VAP exists, which they designated early-onset pneumonia. This subgroup is considered to have a different pathogenesis (impaired airway reflexes) and a different etiologic spectrum (normal oropharyngeal flora, especially anaerobes, rather than Gram-negative bacilli) than VAP occurring more than 72 h after intubation. Application of this concept led to the logical, albeit unsuccessful, strategy of prevention with prophylactic systemic antibiotics directed at anaerobes and other "normal" oral flora.\textsuperscript{4}

The report by Rello and colleagues in this issue of Chest (see page 1230) illustrates a different subgroup of VAP patients with a significant difference in mortality, namely VAP occurring after the use of antibiotics. Multivariate analysis of factors possibly predictive of mortality in 129 cases of VAP demonstrated that prior antibiotic therapy was the only significant independent risk factor for death. Repeat multivariate analysis in the subgroup of patients with an established etiologic agent demonstrated that only "high-risk" causative organisms remained a significant risk factor, suggesting that the effect of prior antibiotic therapy was to select an etiologic agent with a greater associated mortality. Their results confirm those of previous studies\textsuperscript{12,13} and conventional wisdom that infection with certain organisms is more lethal than that with other organisms. Clearly, VAP due to Pseudomonas has the greatest associated mortality. Other organisms associated with a grave prognosis include other nonfermenting Gram-negative bacilli, such as Acinetobacter\textsuperscript{2} and Xanthomonas, methicillin-resistant Staphylococcus aureus,\textsuperscript{3} and, possibly, multidrug-resistant Enterobacteriaceae.\textsuperscript{5} The feature common to infection with these organisms is the competitive advantage afforded to these organisms by broad-spectrum antibiotic therapy.\textsuperscript{3}

Determination of the factors important in a fatal outcome from VAP is complex. In general, mortality reflects the balance between the virulence of the pathogen and the combination of host defenses and appropriateness of antibiotic support. Prior antibiotic therapy influences this balance both by selecting more virulent pathogens and by potentially compromising the effectiveness of antibiotic therapy due to development of multiple drug resistance.

Understanding that all cases of VAP are not the same may explain the disappointing results of recent studies of selective decontamination of the digestive tract (SDD).\textsuperscript{5,7-10} One of the recurring findings in many of the studies is that while VAP may be prevented by SDD, mortality in the SDD group is not different from that of the control group. One possible conclusion is that SDD may prevent some cases of VAP, but not those causing death. Pseudomonas VAP is an important case in point, particularly since Pseudomonas has been demonstrated to colonize the trachea without prior colonization of the oropharynx,\textsuperscript{13} the intended target level of prevention with SDD. The pathogenesis of Acinetobacter and methicillin-resistant S. aureus VAP likewise may not include oropharyngeal colonization. Alternatively, SDD may prevent VAP due to many organisms but actually select for breakthrough VAP due to drug-resistant strains, with corresponding greater mortality rates, similar to the effect observed with the use of prophylactic aerosolized antibiotics.\textsuperscript{5}

A more cost-effective prevention strategy than SDD would be to heed the recommendation of Rello et al that avoidance of unnecessary antibiotics should be an important component of the management of mechanically ventilated patients. Although some antibiotic courses cannot be avoided, others, such as inappropriate prolongation of postoperative prophylaxis, can be. As in other aspects of the care of mechanically ventilated patients, the best antibiotics may be the least antibiotics.

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Trending of Oxygen Utilization in the Critically Ill
Is There a Reliable Method?

Goals in treating the critically ill patient are to maintain hemodynamic stability and to provide adequate oxygen transport commensurate with peripheral tissue oxygen requirements. Oxygen transport is calculated from the product of cardiac output (Qt) and arterial oxygen content, where Qt has been monitored traditionally by intermittent measurements using the principle of thermodilution with a pulmonary artery catheter. Although long considered a clinical standard of assessing Qt, the thermodilution method is time-consuming and user-frustrating and lacks the ability to provide continuous trending. In addition, it is an unreliable indicator of the interaction between oxygen transport and peripheral tissue oxygen utilization.

The Fick equation relates Qt indirectly to venous oxygen saturation (SVO2) and directly to oxygen consumption (VO2) when hemoglobin (Hb) and arterial oxygen saturation are relatively constant. Recent work has discredited the use of SVO2 to predict cardiac output and has led investigators to consider the metabolic rate or VO2 to be a more important component of the Fick equation. However, VO2 can vary significantly over short periods of time in the critically ill patient, and its measurement becomes less reliable in patients using a high fraction of inspired oxygen (FiO2). Because of these shortcomings, a modified Fick method6,7 has been employed, whereby VO2 is indirectly determined by measuring the production of carbon dioxide (VCO2) and dividing by an assumed respiratory quotient. The attraction of this method is that the measurement of VCO2 is technically simple and more reliable under conditions of a high or fluctuating FiO2.

In this issue of Chest (see page 1236), Mahutte et al elaborately examine the single variable relationships between thermodilution Qt and metabolic indices (VO2 and VCO2) as well as between thermodilution Qt and SV02, in 28 patients from medical and surgical intensive care units. Their results demonstrated that thermodilution Qt could not be predicted solely from any of the variables but correlated most closely with Qt calculated by the Fick method (ie, VO2 divided by arteriovenous oxygen content difference). The calculated Qt was not affected by saturations obtained from either direct CO-oximetry or an oximetric pulmonary artery catheter. Using receiver operating characteristic curve analysis, the authors found that changes in Qt were best predicted by changes in VCO2.

The findings raise some important issues. First, the direct measurement of metabolic indices avoids mathematical coupling of variables during analysis, which occurs when VO2 is determined indirectly from Qt and arteriovenous oxygen content difference. In addition, the measurement of metabolic indices, especially VO2, is subject to error since metabolic rate has been shown to vary considerably during resting conditions in critically ill patients. The short collection time for each patient and the lack of a mixing chamber for metabolic measurements in this study are factors that impact on the measurements more than the authors discuss. The finding that changes in thermodilution Qt are more closely related to changes in VCO2 reflects the ability to measure VCO2 with less error than with other indices. However, using VCO2 to trend oxygen utilization may be unreliable since changes in carbon dioxide elimination may not parallel oxygen uptake under dynamic conditions.

Second, data analysis must account for the inverse relationship between Qt and SV02 explicitly described by the Fick equation. The authors’ data showed that the inverse of thermodilution Qt could not be predicted by SV02 when determined from saturations using either direct CO-oximetry or an oximetric pulmonary arterial catheter. As discussed, this was attributed to the nonhomogeneity of the other Fick variables, namely, Hb and VO2. The authors were able to demonstrate that thermodilution Qt most closely