4. What are the drivers for innovation and product development among manufacturers? Is there a search for engineering solutions that do correlate with patient comfort?

5. Does measurement of comfort lend itself to an euclidean quantitative relationship, of the type: if a > b and b > c, then a > c? That is, if a patient says that (s)he prefers ventilator b to ventilator a, and ventilator c to ventilator b, can we safely assume that ventilator c will be preferred to ventilator a in a direct comparison? The order of comparison may be a major confounding factor, as is often the case in market testing of consumer products. This possibility would require not only that all ventilators be tested, but also that each of them be tested against each other, an even more daunting task.

The current study offers us useful information: patient comfort (and, presumably, long-term compliance and therefore benefit) does vary among devices, and the perception of comfort is not correlated with either their physiologic effects or their impact on respiratory mechanics. The current study also concludes that long-term home NPPV should be tailored to the individual patient. In essence, this is another plea for patient-centered care and participation by the patient in relevant care decisions. If only we practiced in a system where that were even remotely possible . . . .

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Ventriculator-Associated Pneumonia

Is There Any Gold in These Standards?

Standard: something established by authority, custom or general consent as a model or example.

Middle English, from Old French estandard rallying point.

Ventriculator-associated pneumonia (VAP) is not only a common hospital-acquired infection but is said to be the most frequent infection in the ICU. A literature search also suggests it must be one of the most studied and written-about topics in critical care. There seems to be a relatively constant risk per day of developing pneumonia while receiving mechanical ventilation. It is a problem that every hospital must address. Regardless how common it is, and despite the voluminous literature already in existence, controversies remain in every aspect of VAP including epidemiology, diagnosis, treatment, prevention, and outcomes assessment.

In this issue of CHEST (see page 2115), Rello et al have defined several risk factors and examined clinical and economic outcomes data. This is the largest US study of its kind to date. The authors are to be congratulated for tackling and dissecting the MedQual Profile database. Employing a vast database gives more credence to their conclusions about length of stay and added cost of care. They also openly discuss the limitations of their study, including difficulties regarding the definition of VAP and assessment of other risk factors. These limitations, however, are not unique to the study of VAP.

The challenges involved in the evaluation of VAP really serve as a paradigm for other ICU research issues. A recent study in CHEST looked at difficulties encountered in studies involving the critically ill. Although the study by Rello et al was a retrospective matched cohort study, and the recent CHEST study referred to the design of randomized clinical trials, the issues are similar. How do we define diseases and syndromes? How do we account for heterogeneous groups of patients undergoing multiple interventions? Can the measured outcomes truly discriminate risks and benefits? For example, though mortality rate may be the most common and
easiest outcome to record, in the ICU it may be too insensitive to detect small benefits of certain interventions.³

Another study looked at several different definitions of VAP.⁵ Depending on criteria used, incidence of VAP varied from 4 to 48%. Furthermore, risk factors predicting VAP varied among the definitions. The particular basis for diagnosis may also account for differences in reported outcomes.⁶ Prior treatment with antibiotics may influence the predictive value of diagnostic techniques as well.

The Health and Science Policy Committee of the American College of Chest Physicians convened a group of experts to develop recommendations based on the literature.⁷ They suggested one of two management options. The first involved quantitative culture techniques, and the second used selection of antibiotics based on risk factors, local epidemiology, and resistance patterns. Both options are grade D, meaning no definite evidence or consensus opinion exists. This group also noted that the reported sensitivity of quantitative BAL and protected specimen brush (PSB) varied widely. These techniques are not well standardized, and there was no conclusive evidence or consensus indicating preference for one invasive test over another. Light⁸ suggested that BAL and PSB are simply variable dilutions of the endotracheal aspirate. He goes on to describe “the common sense notion that specimens obtained from locations only 5 to 15 cm apart along a widely patent airway in continuous motion are unlikely to have substantially different bacterial populations.” In any case, there is no high level evidence proving that any particular quantitative culture technique provides better clinical outcomes than empiric treatment.⁷

What should the clinician do when there is no “gold standard” approach to diagnosis, treatment, or outcomes? Rather than view it as a depressing controversy with no hope for resolution, I would suggest the study of VAP is ripe for further exploration. Study populations can be better defined and outcomes should be evidence based. When designing trials, researchers must decide philosophically if it is more important for the intervention to show benefit under ideal conditions or those encountered under usual practice circumstances.⁴ Despite the large volume of extant literature, many questions remain unanswered. Which patients benefit from continuous subglottic suctioning? Do certain surgical procedures predispose patients to VAP more than others? What is the relative risk for different types of trauma? I have wondered if the mode of mechanical ventilation itself, the inspiratory flow rate, or the inspiratory waveform in some way predisposes to or protects from the deposition of bacteria into the lower respiratory tract and thus affects the subsequent risk for VAP?

Finally, what should we at the bedside do today? I would argue for a practical and common sense approach. When possible, patients receiving ventilation should be positioned at a 45° head-up angle to decrease the risk for aspiration of gastric contents. Pick a definition of VAP for your institution and apply it consistently. Work closely with your hospital infection control committee to track cases. In hospitals with surveillance programs, nosocomial infections decreased almost a third over a 5-year period.⁹ Know your local antibiotic resistance patterns and apply this information when choosing empiric therapy and awaiting culture results. Review data with your medical staff, and when you make changes in ICU practices observe for differences in whatever outcome variable you choose.

Most editorials like this usually end with a plea for multicenter randomized prospective controlled trials. While this represents a laudable goal, it simply is not a feasible approach to answer every clinical question we encounter daily. Medicine has always been part art and part science. We have no reason to believe that the study of VAP should be any different. There remains an important role for clinical judgment. And . . . don’t forget to wash your hands!

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Women and Mesothelioma

In this issue of CHEST (see page 2224), Metintas and coworkers compared the relative risk of women vs men for a malignant pleural mesothelioma (MPM) due to environmental amphibole asbestos exposure. The relative risk was higher for women than for men: 159.8 per 100,000 vs 114.8 per 100,000, respectively. A previous study from a different area of Turkey called Karain Village, where there had been environmental exposure to a highly carcinogenic fibrous zeolite called erionite, also demonstrated a female sex predilection for MPM with a risk ratio of 440.9 per 100,000 for women vs 298.1 per 100,000 for men. North American, Australian, and European cohorts have always shown a much lower risk for women.

First of all, we do not know if this difference is simply due to higher exposures in women. In many of these small Turkish villages, the whitewashing of homes is done by women, which is the procedure associated with the highest levels of exposure. Women spend more time in the home than men, who may work in an area with lower exposure. The authors have evaluated their cohort and cannot find any significant difference in exposure levels between men and women! Traditional work-associated cohorts are predominately men since many trades commonly associated with asbestos exposure historically excluded women. This in turn means that men are overrepresented in mesothelioma cohorts. I am unaware of any North American, Australian, South African, or European cohort where sufficient numbers of women were equally exposed to the same asbestos fiber type at the same intensity for sufficient periods to evaluate the relative risk of MPM for women. Could there be a physiologic explanation for this difference?

Deposition patterns may vary by mouth vs nose breathing, or deeper slower respirations vs shallow respirations. Lung volume influences fiber deposition and retention, particularly at low functional residual capacity, such as pregnancy. Generally, fiber deposition by impaction, sedimentation, and interception increases in major airways based on lung size. People who are taller and have longer tracheas and larger lungs have more deposition in the ciliated airways than shorter, smaller people who tend to have greater alveolar deposition at the same level of exposure. The role of body size needs to be further studied but could explain an increased female risk for mesothelioma. The effect of lung size on fiber retention might suggest that children would retain more asbestos fibers at the same exposure level than adults, but this is unproven and only speculative at this time.

Women seem also to be more susceptible to malignant peritoneal mesothelioma than men. In men, the risk increases up to fivefold with increasing exposure. In women, the relative risk for spontaneous malignant peritoneal mesothelioma is increased. Asbestos-related malignant peritoneal mesothelioma did not increase above moderate exposure in one study as opposed to men, where the risk increases continually with dose. Of course the reasonable question is: Is peritoneal mesothelioma misdiagnosed in women? Both ovarian cancer and malignant peritoneal mesothelioma derive from the same tissue, the coelomic epithelium from which the ovarian surface epithelium is derived. Pathologists, in the past, have had difficulty distinguishing histologically between epithelioid malignant mesothelioma of the peritoneum and serous adenocarcinoma of the ovary or extraovarian tissues. An association between peritoneal talc usage and MPM was made in 1982 when these women were exposed to older talc preparations containing tremolite asbestos. In older cohorts prior to modern tissue immunohistochemical staining and electron microscopic techniques, accurate diagnosis may have been a problem, but with current histopathologic techniques this is not a significant problem today. The environmental pathology research group in Great Britain concluded on review of 177 cases of both pleural and peritoneal mesotheliomas in women that 98% had an elevated fiber burden to amphibole asbestos. The high percentage of women with elevated amphibole asbestos in this British cohort reflects a selection bias since the opposite has been the American experience, where only a minority of women with a mesothelioma of any type have a history of asbestos exposure. The pathology of malignant mesothelioma is the same in men and women.

The other important issue raised by this study of Metintas and coworkers is the relationship between asbestos dose and latency. The latency time was the same for both sexes and was 59.2 years. Early reviews of the effect of asbestos dose and latency suggested that latency was not greatly affected by the degree of exposure. Previous reviews have noted a decline in mesothelioma incidence rates after age 70 years, thought to be due to death from competing causes and the death of those individuals with the highest exposures. More recent cohorts have shown longer latency times since first exposure, frequently > 50


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