Your observation that "advanced directives need to be developed before transferring patients from ICU to other areas offering lower levels of intensive care" is appropriate. Our article, however, focused on the need to develop criteria that can be applied to present situations to evaluate whether or not they would benefit from ICU, or if, in fact they would progress equally well or better in an alternative setting with less intensive levels of care.

As was evidenced by our review of the literature, low risk patients are admitted to ICU primarily for observation purposes, and, statistically, they do not require the high levels of care offered by their ICU experience. In this case, they probably would do equally well if admitted to an observation unit that offers the required monitoring of their condition without the high costs associated with an ICU stay.

As for your comment regarding "100 percent guarantee of good outcome before changing a lesser value of care," I don't believe that this is possible or practical in such a situation. What is possible is planning step-down levels of care that offer appropriate backup in the event additional support is required. This approach would offer the patient appropriate levels of security without incurring the high economic costs or elevated risk of iatrogenic illness possibly associated with ICU care.

I agree with your observation that the decision to offer maximum levels of care to patients with high risk of death or persistent vegetation state is a decision that the families and society must ultimately make for physicians to feel free to select care based on their best judgements. Until this occurs, physicians will continue to respond to high risk patients with little chance of survival or improved quality of life by giving them the highest level of care available to protect themselves against possible legal retaliation.

This may change, however, as the economics of healthcare will ultimately force caregivers to make decisions as to which patients will best respond to a shrinking pool of available healthcare resources.

Hopefully, more studies will produce tools that will enable physicians to make more informed decisions about the patients who will benefit the most from intensive care.

Roger C. Bone, M.D., F.C.C.P.
Medical College of Ohio, Toledo, Ohio

Wet Nebulizers vs Metered Dose Inhalers

To the Editor:

We read with interest the article by Colacone et al. that appeared in the September 1993 issue of Chest in which the effects of albuterol administered by either metered dose inhaler (MDI) with a holding chamber or wet nebulizer were compared in patients suffering of acute asthma. The authors support the hypothesis that a dose ratio of six in favor of MDI is needed to obtain equivalent bronchodilatation. We believe that several issues need to be addressed before a final conclusion can be drawn regarding the place of wet nebulizers for treatment of acute asthma.

First, the definition of acute asthma lacks the precision needed for a therapeutic study and can lead to controversial interpretations. As suggested in the guidelines for the diagnosis and management of asthma, asthma can be precisely classified in three groups by severity of disease: mild, moderate, and severe. The FEV₁ and blood gas analysis failed to estimate severity of patients disease. The fact that all patients but one could be discharged supports the hypothesis that they belonged to acute and moderate asthma. The only patient requiring hospital admission had to be treated by wet nebulization. Indeed, wet nebulizers are in our opinion valuable for treatment of acute and severe asthma. Often in such a situation, patients cannot participate in MDI treatment and require passive beta adrenergic therapy at a high dosage.

It was shown by Barnes et al. that beta-adrenoreceptors are located in the small bronchi. The preference of a nebulizer, the name of which is unfortunately in the study by Colacone et al. producing a 3.6 μm mass median aerodynamic diameter aerosol is therefore controversial. Use of a facial mask is also controversial as inhalation through a mouthpiece is known to limit nasopharyngeal impaction. These two points are particularly important considering that the effects of a wet nebulizer were compared with those induced by an MDI and a holding chamber producing a 1.3 μm mass median aerodynamic diameter aerosol inhaled through a mouthpiece. Moreover, the inhaled fraction, which is one of the most important criteria to assess effectiveness of a wet nebulizer, was not considered at all. The remaining volume of solution in the nebulizer at the end of the inhalation is high with some poor nebulizers, requiring a correction to estimate the actual amount of drug inhaled. Finally, the effects of a smaller dose of albuterol administered with the nebulizer was not assessed and the question of a smaller ratio to obtain an equivalent bronchodilatation in these conditions remains unsolved.

We believe that all studies comparing effects of nebulizers and MDIs have to be carefully analyzed to avoid misinterpretations. Therefore, we think that too much relevant data is lacking to recommend extensive use of MDI rather than wet nebulizers to treat acute and severe asthma in emergency rooms.

Patrice Diot, M.D., and Etienne Lemarie, M.D.,
Department Maladies Respiratoires,
Unite Evaluation Clinique, Tours, France

REFERENCES
1 Colacone A, Afilalo M, Wolkove N, Kreisman H. A comparison of albuterol administered by metered dose inhaler (and holding chamber) or wet nebulizer in acute asthma. Chest 1993; 104:835-41

Occupational Exposure and Pulmonary Function in Health Care Workers in an Aerosol Pentamidine Clinic

To The Editor:

In the August, 1983, issue of Chest, McDiarmid et al. reported adverse effects in healthcare workers (HCWs) administering aerosol pentamidine (AP) in an inpatient unit were engineering controls were not yet available.

We have reported similar studies from our ambulatory central AP clinic in Toronto, Canada, which has provided over 38,000 individual treatments (60 mg pentamidine via Fisonex)....
though we agree that the most common adverse effect in HCWs is ocular irritation, we found no significant differences in cross shift, peak expiratory flow rates (PEFR), forced expiratory volume in one second (FEV₁), or long-term pulmonary function.4,5

McDiarmid et al.1 also focused on the potential for AP to cause changes in the diffusion capacity for carbon monoxide (DLCO), which we find difficult to explain. Serial pulmonary function tests (PFTs) have been performed on our patients and HCWs over the last 3 years and there has been no significant change in DLCO. All PFTs at our center were measured by a single pulmonary function technician using a computerized system (Collins model number 08000, WE Collins, Raintree, Mass.) with the standard American Thoracic Society guidelines, and DLCO was corrected for hemoglobin. The mean baseline DLCO of the six HCWs was 107% ± 21 pred, after 3 years this was 102% ± 5 (p=0.4).

In addition, we have identified a subgroup of 52 patients attending our AP clinic regularly for more than 3 years who have received more than 82 treatments, (4,920 mg) of AP. Surely if AP were to have an effect on DLCO it would be detected in this group of individuals. Baseline DLCO was 89% ± 17 pred, after 3 years this was 91% ± 16 (p=0.6).

We have also detected pentamidine in ambient air samples from our clinic.6 Serial urinary pentamidine assessments were performed on all HCWs at the start and end of random working weeks, analyzed blinded by collaborator G Smaldone,7 all samples were negative for pentamidine, although in a previous study one HCW did have a trace amount of pentamidine detected.8

In conclusion, we could detect no adverse effects on the respiratory function of HCWs in a high volume outpatient clinic. Although cough, wheeze, and bronchospasm occurs in up to 30 percent of patients, we have detected no acute effects on our HCWs. Long-term pulmonary function in both our HCWs, along with patients directly exposed to large cumulative doses of AP show no alteration in DLCO.

R. Andrew McIvor, M.B., Leslie R. Lee-Pack, and Charles K. Chan, M.D., F.C.C.P., Department of Respirology, The Wellesley Hospital, University of Toronto, Toronto, Ontario, Canada

REFERENCES

Hyalin Degeneration
Present in Heart Infarction & Implicated in Pathogenesis of Heart Rupture

To the Editor:

Hyalin degeneration in the myocardium leads to necrosis manifesting features that have not been described in the generally accepted classification of cardiac cell death according to Baroldi.2 For example, relatively large areas of the myocardium, both myofibers and interstitial tissue, may undergo hyaline degeneration and form "pseudovascular ectasias,"1 which imitate sinusoids, venous lakes, hematomas, and thrombi, particularly when the hyalinized eosinophilic tissue starts to fragment into "eosinophilic droplets" similar to red cells.3 Hyalin degeneration may also affect interstitial tissue with the periphery of adjacent cardiomyocytes preferentially, leading to a formation of reticular "intermyocyte hyalinized structures."1 When these structures undergo fragmentation into eosinophilic droplets, their similarity to interstitial hemorrhage becomes startling.4

Reviewing published articles concerning this subject, I have been intrigued by the observation of Lunseth and Ruwaldt5 who consider dissecting hematomas to be the cause of heart ruptures. "We see what we know," Goethe said, and indeed, having our previous observations of hyalin degeneration in mind,1,3 I have looked at Figures 2 to 5 in the article by Lunseth and Ruwaldt5 with different eyes than the authors. Instead of dissecting hematomas with red cells, I have seen intermyocyte hyalinized structures (Fig 2, Lt side) and pseudovascular ectasias fragmenting into eosinophilic droplets (Fig 2, rt side). In Figure 4, the alleged hematoma contains linear structures incompatible with blood and fusing with the surrounding myocardium (there is no question of a laminated thrombus). The alleged hematoma in Figure 5 contains the structures that are characteristic of highly organized tissue, eg, in the upper right corner and in the middle of the right side, clearly distinguishable borders of hyalinized cardiomyocytes are still visible.

If my interpretation is correct, hyalin degeneration is also present in acute myocardial infarction and contributes decisively to the pathogenesis of infarction heart ruptures by a weakening of infarcted and noninfarcted myocardia. In this case, the hyalinized tissue is eliminated by fragmentation or by enzymatic digestion, leading to a formation of tissues accessible to the blood coming from the interstitium or from the heart cavity.

Jiri T. Beranek, M.D.,
Columbia, Missouri

REFERENCES