Mechanical Ventilation for Pneumocystis carinii Pneumonia in AIDS

To the Editor:

Wachter et al\(^1\) provide valuable discussion regarding intensive care treatment of patients with AIDS. When the authors state that "no patient who survived mechanical ventilation has lived for more than one year after hospital discharge\(^1\)", they were presumably referring only to the studies cited. Unfortunately, that point is not clear. We fear that the impression that no patients survive beyond one year will be accepted and used in discussions of intensive therapy with AIDS patients. Two of our patients provide examples of improved outcome following mechanical ventilation for Pneumocystis pneumonia (PCP).

A 34-year-old man presented in September, 1986 with severe dyspnea and hypoxemia requiring intubation and mechanical ventilation. PCP was diagnosed by bronchosalveolar lavage (BAL), and the patient was treated with IV pentamidine. He was extubated five days later and received a 21-day course of pentamidine therapy. He received Zidovudine and was generally well for 19 months. He developed cryptococcal meningitis in April, 1988 and died of line-related sepsis in August, 1988.

A 30-year-old man presented in June, 1987 with HIV seropositivity, wasting and fevers. Zidovudine therapy was started in August, 1987 and one week later he developed PCP, diagnosed by BAL. He received trimethoprim-sulfamethoxazole (TMP-SMX) therapy, but hypoxemia progressed. Four days later the patient was intubated for respiratory failure. The patient was extubated after 18 days, and TMP-SMX was continued for prophylaxis. The patient's subsequent clinical course is demonstrated in Figure 1. He is alive 23 months following mechanical ventilation.

Reported mortality rates for AIDS and respiratory failure have ranged from 98 to 100 percent.\(^1\)\(^3\) Luce et al\(^1\) speculated that the improved survival in one study\(^2\) was in part due to short-lived, procedure-related respiratory failure which may have been avoided with the use of less invasive tests.\(^5\) However, the reported mortality of 58 percent may be more relevant to patients cared for in centers that continue to rely on bronchoscopy for diagnosis.

All physicians who counsel AIDS patients must carefully analyze mortality data before accepting a figure as applicable to their patients. Informing a patient that there are no long-term (\(>1\) year) survivors of AIDS and respiratory failure would be incorrect. In addition, physicians should recognize the limitations of early reports of intensive care outcome with AIDS. These reports often lacked important clinical information, and changes in therapeutic options and prognosis are rapidly occurring. We agree with El-Sadr and Spritz\(^4\) that many physicians are reluctant to recommend mechanical ventilation to any patient with AIDS based on reports of 98 percent mortality or higher mortality. This approach should be re-examined, as it does not take into account the diversity of patients with AIDS who develop respiratory failure.

Lisa Veach, M.D., and Jack T. Stapleton, M.D., Infectious Diseases, Department of Internal Medicine, University of Iowa, Iowa City

REFERENCES

3 Luce JM, Wachter RM, Hopewell PC. Intensive care of patients with the acquired immunodeficiency syndrome: time for a reassessment? Am Rev Respir Dis 1988; 137:1261-63

To the Editor:

We thank Drs. Veach and Stapleton for their comments. As they infer, our statement\(^1\) regarding long-term survival of patients who survived mechanical ventilation for Pneumocystis carinii pneumonia (PCP) referred only to those patients reported in the literature. At the end of the statement, we went on to say that "three (patients) were alive at the time their cases were reported\(^1\)\(^2\). In our original study on the outcome of PCP and respiratory failure,\(^1\) we reported 45 patients who received mechanical ventilation for PCP. Of these, 39 (87 percent) died in the hospital, and five others died in the next 11 months. In fact, the one patient who was alive at the time of that report remains alive 33 months later. Thus, we have also become aware of the possibility of long-term survival after intensive care for PCP and respiratory failure.
In the last three years, five centers (including our own) have documented the improving survival for PCP and respiratory failure. At San Francisco General Hospital, the survival rate has increased from 13 percent in 1981 through 1985 to 40 percent in 1986 to 1988. The reasons for the improvement are uncertain, since severity of illness and anti-PCP therapy were similar in both eras. Although steroid use was much more common in recent years, its use in individual patients was associated with only a mild improvement in outcome.

The decisions of competent patients regarding life-sustaining treatments should be respected. We agree with Veach and Stapleton that such patients should be informed of the most recent data, including the possibility of long-term survival. We suspect that increasing numbers of patients so informed will choose intubation and mechanical ventilation for PCP and respiratory failure. The ethical principle of justice requires that such patients be allowed the same degree of access to the ICU as non-AIDS patients with similar prognoses.

Robert M. Wachter, M.D. F.C.C.P.; John M. Luce, M.D. F.C.C.P.; Bernard Lo, M.D. F.C.C.P.; and Thomas A. Raffin, M.D. F.C.C.P.
San Francisco

REFERENCES
1 Wachter RM, Luce JM, Lo B, Raffin TA. Life-sustaining treatment for patients with AIDS. Chest 1989; 95:647-52
5 Rogers PL, Lane HC, Henderson DK, Parrillo J, Masur H. Admission of AIDS patients to a medical intensive care unit: causes and outcome. Crit Care Med 1989; 17:113-17
8 Wachter RM, Russ MC, Hopewell PC, Luce JM. The improving survival rate after intensive care for P carinii pneumonia and respiratory failure. Fifth International Conference on AIDS, Montreal, Canada, 1989

Cytology of Bronchoalveolar Lavage Fluid in Bacterial Pneumonia

To the Editor:

We have read with interest the recently published paper of Hopkins et al about neutrophil chemotactic factors in bacterial pneumonia. Several lung diseases characteristically show an increased number of neutrophils in the fluid recovered from bronchoalveolar lavage (BAL). We studied the cytology of BAL fluid from mechanically ventilated subjects without lung disease, and in patients with pneumonia, sarcoidosis, pulmonary fibrosis and adult respiratory distress syndrome (ARDS). Over a period of one year we analyzed 24 samples of BAL fluid from nine intubated and mechanically ventilated patients fulfilling all the criteria for ARDS. The normal group consisted of 20 subjects, seven of whom were mechanically ventilated for neurologic problems with normal gas exchange breathing air. The sarcoidosis group consisted of 37 patients (15 stage I, and 22 stage II) diagnosed by lung biopsy. Thirty patients with pathologically-proven diffuse idiopathic pulmonary fibrosis represented the third control group. The pneumonia group consisted of 27 critically ill patients with pneumonia diagnosed by clinical, radiologic and microbiologic (positive quantitative culture of BAL fluid or protected endobronchial brushing) criteria without ARDS.

BAL was performed within 24 h of establishing the diagnosis of ARDS and repeated at 72-h intervals during the first 12 days of diffuse alveolar damage. We defined as early ARDS the results obtained during the first five days of evolution.

In patients with ARDS and mechanically-ventilated control patients, a flexible fiberoptic bronchoscope was passed through the endotracheal tube, inserted and wedged into a subsegment of the lingula or right middle lobe. In each patient, three 50 ml aliquots of buffered 0.9 percent saline solution at room temperature were instilled and immediately aspirated by gentle suction applied after each infusion until at least 45 percent of the volume injected was recovered. The first sample, corresponding to the first 50 ml lavage, was not used for cytologic analysis because of the small volume and disproportionate amount of bronchial airway material. Second and third samples were pooled, passed through gauze and immediately centrifuged to separate the cellular and noncellular components of BAL fluid.

A differential count was made from at least three air-dried cytospin-stained cell preparations stained with a modified Wright-Giemsa stain. All preparations were analyzed by the same observer using a standard light microscope and employing objectives of 25 and 40. A minimum of 300 to 500 cells were counted by the random field method.

Patients in control groups without mechanical ventilation underwent the same lavage procedure except that the bronchoscope was passed through the upper airway following local anesthesia with 4 percent lidocaine spray. Endobronchial protected brushing was performed in all ARDS and pneumonia patients at the more

### Table 1 - Results of BAL Cytology

<table>
<thead>
<tr>
<th>Clinical group</th>
<th>Number of BALs</th>
<th>%MCP</th>
<th>%PMN</th>
<th>%LYM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal subjects</td>
<td>20</td>
<td>91 ± 1*</td>
<td>5 ± 1*</td>
<td>5 ± 1*</td>
</tr>
<tr>
<td>Sarcoidosis</td>
<td>37</td>
<td>60 ± 3*</td>
<td>5 ± 1*</td>
<td>26 ± 3</td>
</tr>
<tr>
<td>Pulmonary fibrosis</td>
<td>30</td>
<td>66 ± 3*</td>
<td>26 ± 3*</td>
<td>6 ± 1*</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>27</td>
<td>69 ± 3*</td>
<td>16 ± 3*</td>
<td>14 ± 2*</td>
</tr>
<tr>
<td>Early ARDS</td>
<td>14</td>
<td>31 ± 8</td>
<td>64 ± 8</td>
<td>4 ± 2*</td>
</tr>
<tr>
<td>Late ARDS</td>
<td>9</td>
<td>68 ± 10*</td>
<td>25 ± 10*</td>
<td>5 ± 3*</td>
</tr>
</tbody>
</table>

*Control groups vs ARDS, p<0.001 (one-way ANOVA).
†All groups vs sarcoidosis, p<0.001 (one-way ANOVA).
‡Late ARDS vs early ARDS, p<0.001 (ANOVA RM).

BAL cytology in early ARDS (less than five days evolution); late ARDS (more than five days); sarcoidosis, pulmonary fibrosis and pneumonia. (MCP = macrophages; PMN = polymorphonuclear leukocytes; LYM = lymphocytes.)