readily available to clinicians. This was also a few years before we began to use mechanical ventilation for conditions other than poliomyelitis, spinal cord injury, and kyphoscoliosis. Many surgeons could not duplicate Brantigan’s mortality and morbidity. Although spirometry was slowly coming into general use, the clinical pulmonary function measurement was still in its early days and Brantigan presented no objective preoperative and postoperative data. Finally, the pulmonologists of the day were busy controlling the epidemic of tuberculosis and learning how to do so in the outpatient setting as sanitarium beds were closed apiece. Thoracice surgeons were busy resecting residual “open negative” tuberculous cavities and solid caseous foci.

I did not mention Brantigan’s name in my recent article in CHEST (1996; 109:540-48), because Brantigan’s contribution was to have the insight to recognize that reduction pneumoplasty would work for emphysema in the absence of a giant bullae. His formulation of the physiologic basis for operating on emphysematous patients in the absence of bullae is slowly being confirmed as physiologic studies on lung volume reduction surgery are published. I have reviewed Brantigan’s seminal contribution to reduction pneumoplasty in another article.1 Since my review was on reduction pneumoplasty for bullous disease, I thought it inappropriate to present it there. 

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REFERENCE


Determining the Pathogenetic Significance of Cytomegalovirus in Patients With AIDS

To the Editor:

Despite extensive research into the significance of identifying cytomegalovirus (CMV) in bronchoalveolar lavage fluid of patients infected with HIV, a clear understanding of the pathogenetic significance of CMV is still beyond our grasp. There are many reasons for this, foremost among which is the lack of a uniform definition of CMV pneumonitis: should it be based on evidence of CMV infection (culture positivity) or disease (cytologic changes)? The latter seems to result in a lower diagnostic yield, as seen in a study by Miles et al.1 in which, of 62 patients proved to have CMV infection by culture and immunofluorescence stain, only 5 had positive cytology for CMV. In a recent issue of CHEST, Hayner and colleagues2 found that the odds ratios for predicting mortality based on BAL evidence for CMV were comparable high whether CMV positivity was defined by culture or cytology. What criteria, therefore, should we be using?

The high prevalence (28 to 52%)3,4 of CMV in patients with HIV potentially obscures its contribution to the high morbidity and mortality associated with HIV-related pulmonary complications. The added difficulty of distinguishing CMV disease from coinfection is highlighted by the results of an autopsy series,4 which demonstrated that 94% of 31 patients with CMV pneumonitis (positive culture and cytology) had coexisting pulmonary disease.

The advent of steroid therapy for severe Pneumocystis carinii pneumonia has added a twist to this dilemma, which may result in a better understanding of the pathophysiology of CMV pneumonitis. In a recent article in CHEST, by Jensen et al.,3 patients with severe Pneumocystis carinii pneumonia treated with corticosteroids, who were also CMV positive, had a two times higher 3-month mortality than those who were CMV negative (p=0.08). This was not accounted for by differences in HIV helper cell count, PO2, duration of AIDS, or age. There are two possible interpretations for this finding. Steroids may further immunocompromise these HIV patients and allow a “colonizer” to become pathogenic. Alternatively, CMV infection may simply be a prognostic marker, which identifies patients likely to do poorly. In the study by Hayner et al.,2 the retrieval of CMV was also associated with significantly greater 3- and 6-month overall mortality.

To clarify the pathogenetic role of CMV in HIV-infected patients, what is needed, therefore, is a prospective intervention trial. A cohort of HIV patients infected with CMV, based on BAL findings, would be randomized to receive ganciclovir therapy or placebo for 6 months. (Since we could expect to see differences in mortality by 6 months, as per the study by Hayner et al.,2 this would seem to be an adequate treatment duration.) The lowest effective dose should be used to avoid drug toxicity. If there were a significant difference in observed mortality, then CMV infection is worth treating. If, on the contrary, no difference were seen, then CMV would seem to be more of a marker of poor advanced immunosuppression state and thus an adverse prognostic marker.

It is clearly of great importance to define the significance of CMV in this population, as larger proportions of AIDS-afflicted patients are surviving to later stages and one would expect a larger percentage of patients infected with CMV.

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Do the Methods Justify the Conclusion?

Dopexamine and Splanchnic Blood Flow

To the Editor:

In a recent article in the journal, Maynard et al. (CHEST 1995; 108:1458-54) describe the effects of dopexamine and dopamine on gastric intramucosal pH, monoethylglycinexilidide formation from lido-caine, and indocyanine green clearances in mechanically ventilated critically ill patients. The authors conclude that low-dose dopexamine increases splanchnic blood flow as measured by gastric intramucosal pH, monooethylglycinexilidide formation from lidocaine, and indocyanine green clearances. This conclusion was based on data as obtained from three comparable groups of patients: a group treated with dopamine (n=10), a group with dopexamine (n=10), and 1 control group treated with saline solution (n=5). We would like to make a comment on the methodology, for it remains...