capnia. In our judgment, this still remains a useful point in the management of patients with COPD who may have concomitant emboli.

**Michael L. Lippmann, M.D., F.C.C.P., Pulmonary Critical Care Division, Albert Einstein Medical Center, Philadelphia; and Alan M. Fein, M.D., F.C.C.P., Pulmonary Critical Care Division,Winthrop University Hospital, Mineola, New York**

**References**

To the Editor:

We respect the observations of Drs. Lippmann and Fein but recognize that their experience was limited to three patients. Our observations showed inconsistent changes in the arterial Pco₂, but our data were also limited. Perhaps with the acquisition of more data, a more definitive answer will be forthcoming.

**Barry A. Lesser, M.D., F.C.C.P., Grace Hospital; and Paul D. Stein, M.D., F.C.C.P., Henry Ford Hospital, Detroit**

**Adenosine Triphosphate Enhancement of Postischemic Function in Immature Myocardium**

To the Editor:

I read with interest in the August 1992 issue of Chest the article by Muralidharan and associates, describing their finding of enhanced postischemic recovery of function in immature myocardium with adenosine triphosphate (ATP) added to St. Thomas' Hospital cardioplegic solution. I was surprised that the authors failed to mention in their literature review any of the multiple prior published studies that also used ATP to protect the myocardium during ischemia. This body of literature is well summarized in an article from my laboratory. Because of some differences found between adult and immature myocardium, many investigators too often assume that all studies in adult models of ischemia (including the prior ATP articles) are irrelevant and therefore ignore them.

The primary questions about this study center on methodology. The authors chose 500 μmol/L as their optimal dose of ATP based on a preliminary (unpublished) dose-response study. When I performed my original dose-response work with ATP in St. Thomas' Hospital cardioplegic solution in adult hearts, I found that a lower dose of 100 μmol/L (0.1 mmol/L) was optimal (Fig 1). However, when ATP is added to solution, especially at higher doses, the pH of the mixture declines, thus requiring the addition of base (dilute sodium hydroxide in our study) to maintain a stable pH in the cardioplegic solution. The authors do not mention pH adjustment when ATP was added in their study; and probably their resultant solution was more acidic. In a recent study by Iannettoni and associates, postischemic recovery of function by neonatal hearts receiving St. Thomas' Hospital cardioplegic solution was best if the solution was made acidic. Consequently, the presumed ideal dose of ATP used by Muralidharan and associates was probably incorrect due to inadvertent acidification of the solution. A lower optimal dose (similar to the optimal adult dose of 100 μmol/L) might have been found in immature hearts if the pH had been appropriately adjusted.

The second methodologic concern involves the isolated working heart model used in this article. As they described it, "a canulated balloon was positioned into the left ventricular (LV) cavity via the mitral valve through the left atrium." The balloon was inflated to a steady preload pressure of 10 cm H₂O, and the rate of rise of LV pressure was measured. They are, therefore, describing the classic isovolumic isolated working heart model. Yet the authors also state that they have quantitated LV flow with a flowmeter connected to the LV afterload system. In the isovolumic heart preparation that they are using, significant LV flow does not reliably occur; therefore, this measurement has no validity.

Finally, in the "Materials and Methods" section, the composition of St. Thomas' Hospital cardioplegic solution (Plegisol) is incorrect.