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

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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.

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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.3 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 The authors chose 500 mmol/L as their optimal dose of ATP based on a preliminary (unpublished) dose-response study.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,1 I found that a lower dose of 100 mmol/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 mmol/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 cannulated 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.4 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.
They state that this solution contains calcium bicarbonate in a concentration of 10 mEq/L. The correct formulation of Plegisol includes calcium chloride in a concentration of 2.4 mEq/L. Sodium bicarbonate is added just prior to use to give a final concentration of 10 mEq/L.

The purpose of this study was to investigate whether exogenous ATP added to cardioplegic solution enhanced myocardial protection in the immature myocardium. I suspect that ATP is probably effective in the immature heart also, as it was previously shown to be in the adult heart in my study. Nevertheless, rather than just accept the positive conclusions of the authors, as we are often inclined to do when an article is published, I have chosen to raise questions regarding the methodology employed to generate these data and formulate their conclusions. The reader of the medical literature must critically assess all aspects of published studies, including methods, before results can be accepted.

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Evaluation of Cardiac Output by Thoracic Electrical Bioimpedance During Exercise in Normal Subjects

To the Editor:

I am writing to express my concern about the article by Moore et al., which appeared in the August 1992 issue of Chest. Contrary to their assertions, I do not believe that the authors have demonstrated that the thoracic electrical bioimpedance method can be used for determination of cardiac output during exercise in healthy subjects.

In the analysis of their data, they use strained logic to conclude that there is "acceptable" agreement between bioimpedance and CO2 rebreathing methods. Compensating for bias in the bioimpedance method at some work rates, but not others, is not a reasonable procedure. Are the authors suggesting that other users make work rate-dependent corrections in bioimpedance values in order to obtain acceptably correct results? Examination of their Figure 3 reveals that approximately 35 percent of determinations lie outside the rather liberal "acceptable" range of ±22 percent of the CO2 rebreathing determination.

Moreover, the authors claim that credence is lent to the bioimpedance technique because cardiac output measurements correlate significantly with VO2 over a range of work rates. These two variables could hardly fail to correlate significantly! Heart rate increase is the dominant cause of cardiac output increase during exercise, and heart rate is strongly correlated with VO2. The stroke volume measurements of the bioimpedance device could have a very large percentage of random inaccuracy and calculated cardiac output would still correlate quite well with VO2.

Thoracic electrical bioimpedance has a weak theoretical foundation, which has not been substantially improved by recent modifications in the calculational algorithm. Basic assumptions include the following: (1) At the beginning of systolic ejection, blood flow into the aorta is maximal. (2) There is no outflow from the aortic segment at the time of maximal inflow. (3) The inflow into the aorta continues at a constant rate throughout ventricular ejection. (4) The ventricular ejection time can be accurately measured from the impedance signal. (5) The fluctuation in thoracic impedance is due solely to impedance of the thoracic aorta. None of these assumptions is on firm ground, and some have been explicitly refuted.

Based on both theoretical considerations and the experimental data obtained to date, I remain convinced that this bioimpedance method is of no use in evaluating hemodynamics during exercise.

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Aspiration of Potpourri

To the Editor:

Aspiration of foreign bodies into the tracheobronchial tree is a relatively common occurrence in children. Waring lists "indiscreet curiosity" as one of the hallmarks of pediatric respiratory disease and foreign body aspiration as one of its consequences. We recently cared for a child who aspirated a fragment of potpourri, which is an aromatic mixture of dried flower petals and spices, sometimes treated with oil-based perfumes.

The child was a previously healthy 10-month-old boy who was found to be coughing with a mouthful of potpourri air freshener. The father removed this material, but within 1 h the infant developed coughing and audible wheezing. He was seen by his pediatrician and was thought to have a normal physical examination and a normal chest x-ray film. The wheezing and coughing persisted overnight, prompting his pediatrician to transfer him to Tulane Medical Center. During transport, the patient coughed up an object that appeared to be a small, dried leaf. He continued to cough, although the coughing was markedly decreased. On arrival at Tulane Medical Center, his physical examination was remarkable for tachypnea (respiratory rate of 60 breaths per minute) and coarse inspiratory crackles over the right middle and right lower lobes. A chest radiograph revealed an infiltrate in the right middle lobe. Normal movement of both hemidiaphragms was noted on fluoroscopy. Rigid bronchoscopy showed a moderate amount of white secretions in the right mainstem bronchus, while the left bronchial tree appeared normal. He was treated with aerosolized racemic