given to our paper published on the same subject in the American Journal of Cardiology (1987; 60:857-67): "Despite the use of the largest single balloon available, hemodynamic improvements may remain insignificant and suboptimal."

Considering the nature of the paper of Miday et al, this seems to implicate that we found that the improvement is insignificant with a single balloon only. I would like to correct this. We found the improvement of the anatomy in calcified aortic stenosis following aortic valveplasty insignificant using either single or double balloons.

As we see it, the procedure provides minimal, if any, anatomic improvement regardless of the balloon technique used.

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**PA Catheter Measures in Lung Injury**

*To the Editor:*

I must applaud the investigative work of Henriquez (Chest 1988; 94:491-95) which looks at the value of pulmonary artery catheter measurements taken in different regions of the lung. Their results in a group of patients with well-established chronic lung disease demonstrated that regional local perfusion abnormalities may exist, thus altering the absolute pressure measurements monitored in that region. Their recommendation after considering the results of sequentially monitoring the various lung segments was to make serial measurements "...in the same location for all of the periods of observation". No mention was made as to how these different lung segments may have responded to a change in the patient's condition (eg, hypovolemia, cardiogenic failure or the addition of lung injury). Stated simply, the absolute values may be different but each lung segment measured may still trend accurately! This is exactly what was found in a recent study conducted using a unilateral lung injury model in sheep where both lung segments were simultaneously monitored throughout the experiment. I must admit that I am a bit surprised that so little investigative work has been done to confirm the accuracy of this unilateral lung monitor in different pathologic states, yet dogmatic clinical decisions are made daily with its continuing use.

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**REFERENCES**

1 Henriquez A, Schnojin F, Redondo J, Delorme N. Local variation of pulmonary arterial wedge pressure and wedge angiograms in patients with chronic lung disease. Chest 1988; 94:471-95

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**Reaction After Re-exposure to Trimethoprim-Sulfamethoxazole**

*To the Editor:*

We read with interest the recent report by Silvestri et al of two AIDS patients who developed pulmonary infiltrates and hypoxemia upon re-exposure to trimethoprim-sulfamethoxazole (TMP/SMX). Recently, we also cared for an AIDS patient with a similar presentation. In our case, a 23-year-old homosexual man with human immunodeficiency virus infection developed progressive exertional dyspnea associated with bilateral interstitial pulmonary infiltrates. Bronchoalveolar lavage revealed *Pneumocystis carinii*. The patient was started on oral TMP/SMX and promptly improved. However, after seven days he developed patchy erythema and hives over his trunk and arms. These resolved after one day off medication. He did well until dyspnea recurred on the day of admission, and in response took another dose of his prescribed TMP/SMX. He quickly became flushed, diaphoretic, more dyspneic, nauseated, and experienced vomiting and diarrhea. A bifrontal headache developed.

Upon presentation to our hospital, temperature was initially 38.9°C (oral) and rose to 40.5°C associated with shaking chills. Heart rate was 130 bpm, respiratory rate 40/min, blood pressure 93/60 mm Hg falling to 82 mm Hg systolic. No rales or wheezes were heard upon auscultation of the lungs. Cardiac examination revealed a 1/VI systolic murmur without gallops. Skin examination revealed blanching erythema without hives or blisters. Laboratory revealed Hct 38.3, WBC 6800/ml with 47 segs, 39 bands, 8 lymphocytes and no eosinophils.

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**FIGURE 1**