ature change from transpulmonary transit of indicator. Although the author did not provide a time scale on the Figure displayed, the time interval between the peaks of the thermodilution curves appears to be much too short to be explained by the transit of indicator through the pulmonary circulation. This fact alone would preclude consideration of an intracardiac shunt to explain the tracings displayed.

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To the Editor:

I thank Dr. Cole for focusing on a figure I recently published in CHEST (August 2004). However, I totally disagree with his comments. Dr. Cole claims that the time interval between the two peaks of the so-called camel transpulmonary thermodilution curve (with two humps) that was presented in my article is “much too short” to mimic a right-to-left intracardiac shunt.

When the camel curve is due to a right-to-left intracardiac shunt, the time interval between the two peaks of the so-called camel transpulmonary thermodilution curve (with two humps) represents the blood transit time between the right and left atrium through the pulmonary circulation. When the camel curve is due to a cross-talk phenomenon, the time interval between the two peaks is necessarily longer (and not shorter!), since it represents the blood transit time between the femoral vein (cold indicator injection) and the femoral artery (a longer distance means a longer time interval).

Moreover and more importantly, in both cases the time interval between the two peaks is highly dependent on cardiac output and, for instance, will be three times shorter in a patient with a cardiac output of 9 L/min than if the cardiac output is only 3 L/min. Therefore, I still believe that the “eyeball” inspection of a transpulmonary thermodilution curve does not allow the discrimination between a cross-talk phenomenon and a right-to-left intracardiac shunt. The camel curve represented in my article is now depicted in Figure 1 with a different time scale and really looks like a camel curve due to a right-to-left intracardiac shunt.²

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References


Effects of Leukotriene Receptor Antagonists on Vascular Endothelial Growth Factor Levels in Asthma

To the Editor:

Kanazawa et al.¹ have reported an interesting reduction in airway permeability with pranlukast that correlated with a reduction in airway vascular endothelial growth factor (VEGF) levels. However, no such reduction in VEGF levels was observed in steroid-treated asthmatics. In contrast, a clinical study² has shown a further benefit in symptoms and lung function in the combination of leukotriene receptor antagonists and inhaled steroids. It is therefore perhaps surprising that no further reduction in airway VEGF occurred with pranlukast added to the steroid-treated patients.

Other questions remain. What is the mechanism of the reduction in measured airway VEGF level in asthma? Is there a VEGF receptor up-regulation in the airway or an increase in free VEGF? Could this reduction be secondary to cellular changes in the airway epithelium, a known source of VEGF. Are there other significant sources of VEGF in asthma?

One limitation of the VEGF enzyme-linked immunosorbent assay is its inability to measure the more cell-associated VEGF isoforms (VEGF₁₅₁ and VEGF₂₅₁) that still have bioactivity. Analysis of their expression would increase understanding of the changes in VEGF bioactivity in asthma.

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References

2 Christian Virchow J, Prasse A, Naya I, et al. Zafirlukast improves asthma control in patients receiving high-dose
Effects of Pranlukast on Vascular Endothelial Growth Factor Levels in Asthma

To the Editor:

We thank Dr. Medford for an interest regarding our study.1 There has been a clinical interest in the use of leukotriene receptor antagonists (LTRAs) in asthmatic patients who have already been treated with inhaled corticosteroids. However, a further benefit in symptoms and lung function from therapy combining LTRAs and inhaled corticosteroids is contrary to this finding. In this study, we found that pranlukast administration added little efficacy to inhaled corticosteroid therapy for reduction in vascular endothelial growth factor (VEGF) levels in induced sputum from asthmatic patients. However, it is possible that the trends toward reduction in airway VEGF levels after pranlukast administration in steroid-treated asthmatic patients might have reached statistical significance if more patients had been included in our study.

The mechanism of the reduction in airway VEGF levels in asthma induced by pranlukast administration is unclear. One report2 has indicated that asthmatic patients exhibited a greater expression of VEGF receptors (flt-1 and flk-1) in the airway mucosa. Moreover, increased VEGF expression in asthmatic patients were identified by infiltrating inflammatory cells in the submucosa in order of abundance from the American College of Chest Physicians (e-mail: permissions@chestnet.org). Reproduction of this article is prohibited without written permission.

1 Kanazawa H, Nomura S, Yoshikawa J. Role of microvascular permeability on physiologic differences in asthma and eosinophilic bronchitis. Am J Respir Crit Care Med 2004; 169: 1125–1130

3 Kanazawa H, Nomura S, Yoshikawa J. Role of microvascular permeability on physiologic differences in asthma and eosinophilic bronchitis. Am J Respir Crit Care Med 2004; 169: 1125–1130

Atrial Fibrillation, Atrial Flutter, or Both After Pulmonary Transplantation

To the Editor:

We read with interest the recent report of Nielsen et al (August 2004).1 This is an important study that addressed the prevalence and predictors for atrial arrhythmias after pulmonary transplantation (PT). Nevertheless, some points should be reviewed due to possible misinterpretation.

Atrial fibrillation and atrial flutter were grouped together and termed “AF” in the results and analysis in the present study. However, it should be noted that they are mechanistically and therapeutically different entities, which should not be grouped under the same term.2 Atrial flutter is defined as macroreentry around one or more atrial anatomic obstacles (commonly, the tricuspid annulus or surgical incisions), which can be abolished by catheter ablation.3 A high incidence of atrial flutter has been reported in patients with PT.4,5 Atypical atrial flutter following PT is thought to be secondary to macroreentry around the anastomosis between the left atrium and pulmonary veins.6 On the other hand, the final etiologic mechanism of atrial fibrillation is unknown, and the best treatment for it is unclear. Electrical isolation of the pulmonary veins and the surrounding left atrium myocardium leads to atrial fibrillation abolishment in many patients.7 A similar electrical situation occurs in PT, in which the donor pulmonary veins and the surrounding left atrium tissue are not electrically connected to the recipient’s atria.

Therefore, current scientific evidence makes atrial flutter rather than atrial fibrillation more likely to occur after PT. Thus, separate descriptions and analyses of the incidence and predictive factors of these two different arrhythmias should have been performed in this study, and this warrants further investigation.

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References


