


The Role of Aldosterone in Pulmonary Venous Hypertension

To the Editor:

We read with interest the recent article in CHEST (July 2009) by Robbins et al demonstrating an association between the metabolic syndrome and the existence of pulmonary venous hypertension. Recent research has also demonstrated an association between serum aldosterone, a known cause of left ventricular dysfunction and remodeling, and the metabolic syndrome. Thus, it is possible that increased circulating aldosterone might be independently associated with the risk for pulmonary venous hypertension. Do the authors have data examining aldosterone or renin activity in their subjects?

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REFERENCES

A Comparative Study of Two Different Metered-Dose Inhaler-Valved Holding Chambers in the Administration of Salbutamol

To the Editor:

Rapid-acting, inhaled β-2 agonists are frequently delivered from a pressurized, metered-dose inhaler (MDI) used with a valved holding chamber (VHC). Several studies have shown that VHCs enhance the efficacy of short-acting β-2 agonists in patients who have poor MDI technique and in children. However, different VHCs are available for inhaled therapy without information in the summaries of product characteristics and patient information leaflets on studies of efficacy and compatibility between drugs and

Response

To the Editor:

We thank Drs Farber, Walkey, and Alikhan for bringing to our attention the importance of aldosterone in the development of left ventricular (LV) diastolic dysfunction. The studies noted in their letter highlight the importance of metabolic derangements in the development of LV diastolic dysfunction, which can occur even in the absence of systemic hypertension or LV hypertrophy. We did not measure aldosterone levels in our study but plan to do so in future studies. There is increasing evidence that features of the metabolic syndrome are likely to contribute to the development of pulmonary hypertension in susceptible patients. This is an area that requires further investigation, and we were intrigued by the recent publications of Dr Farber and his colleagues about the potential role of adiponectin deficiency in the development of pulmonary hypertension.