4 Allison DJ, Stanbrook HS. A radiologic investigation into hypoxic pulmonary vasoconstriction in the dog. Invest Radiol 1980; 15:178-90

To the Editor:

We thank Dr. Morrell for pointing out his data on the lack of effects of captopril and losartan on acute hypoxic pulmonary vasoconstriction (AHPV) in conscious rats. The likelihood is that there are species differences, and this may explain why nonselective angiotensin II receptor blockade with saralasin was found to exhibit activity in humans in terms of attenuating AHPV. Indeed, we have also shown that as occurs with saralasin treatment, AHPV is attenuated by type I angiotensin II receptor blockade with losartan in humans.

In the context of cor pulmonale, the increase in the pulmonary vascular resistance has a dynamic component due to hypoxic vasoconstriction, as well as a more fixed component due to established pulmonary vascular remodeling. We have evaluated patients with hypoxemic cor pulmonale, in whom a 50-ng oral dose of losartan produced a significant fall in both mean pulmonary arterial pressure (mPAP) (13%) and total pulmonary vascular resistance (TPVR) (16%) compared with placebo. Taken together, these studies with saralasin and losartan in healthy volunteers and patients with cor pulmonale show that angiotensin II receptor blockade may have an important effect in modifying the AHPV response in man.

We realize that there are limitations to the measurement of Doppler-derived mPAP using the pulmonary acceleration time (PAT). However, we have shown with our own hands in previous studies that there is in fact little variability between simultaneous measurements in a given individual, with coefficients of variability for PAT, reported as 1.7%4 and 1.1%5 in healthy volunteers, and values of 3.2% and 1.9%5 in patients with cor pulmonale. We have also shown a highly significant correlation between Doppler PAT and catheter mPAP over a range of pulmonary arterial pressures. The main problem with Doppler-derived mPAP is that it does not distinguish between pre- and post-capillary vascular resistance, as it is not possible to measure wedge pressure with this technique.

Dr. Morrell also inferred that changes in cardiac contractility may explain the effects of angiotensin II blockade on the PAT. In this respect, we have recently shown in a dose-ranging study, with infusion of endothelin 1 in healthy volunteers, that marked increases in Doppler mPAP and TPVR occurred in concert with negative inotropic and lusitropic activity. Since angiotensin II also has a similar profile in terms of inducing pulmonary vasoconstriction in the presence of negative inotropic-lusitropic effects, it is highly unlikely that effects of angiotensin II receptor blockade on the myocardium would explain the associated fall in mPAP. Indeed, if anything, one could argue that antagonizing the myocardial effects of angiotensin II would, if anything, tend to underestimte the effects of angiotensin blockade in terms of attenuating AHPV.

We therefore remain firmly of the opinion that angiotensin II receptor blockade may be implicated in the pathophysiology of acute pulmonary vasoconstriction and that this may be reliably measured using Doppler assessment of pulmonary arterial flow.

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REFERENCES
1 Kiely DG, Cargill RI, Lipworth BJ. Angiotensin II receptor blockade and effects on pulmonary hemodynamics and hypoxic pulmonary vasoconstriction in humans. Chest 1996; 110:698-703
2 Kiely DG, Cargill RI, Lipworth BJ. Acute hypoxic pulmonary vasoconstriction in man is attenuated by type I angiotensin II receptor blockade. Cardiovase Res 1995; 30:575-80

Noninvasive Measurement of Pulmonary Arterial Blood Velocity
Can it Replace Right Heart Catheterization?

To the Editor:

A recent publication1 has concluded that right heart catheterization using pulmonary artery flotation catheters is “associated with increased mortality and increased utilization of resources.” Methods of magnetic resonance imaging (MRI) of large vessels provide morphologic and dynamic flow-related information completely noninvasively and with high spatial and temporal resolution. In the decade since Singer and Crooks2 proposed the measurement of blood flow with such techniques, MRI velocity mapping has been shown to give accurate estimates of total blood flow and velocity profiles.3,4 Five normal adult volunteers were positioned supine in a 1.5-Tesla Signa imager (GE Medical Systems, Milwaukee, Wis) and a series of scout images of the upper torso of each subject was obtained. An optimal transverse plane was chosen which contained a representative cross-section of a side branch of the pulmonary artery. For blood flow velocity measurement, an ECG-gated phase-sensitive imaging protocol was used which provided velocity sensitivities of 0.7 to 5.0 rad/s/mm, and which resulted in five to eight velocity measurements spanning two