Hydralazine and chronic cor pulmonale
Corriveau ML, Minh Vu-Dinh, Dolan GF. (The Geriatric Research Centre, Education and Clinical Centre, St Louis Veterans Administration Medical Centre and St Louis University School of Medicine, St Louis, Missouri and University of California School of Medicine at Irvine, Irvine, California.) Long term effects of hydralazine on ventilation and blood gas values in patients with chronic obstructive pulmonary disease and pulmonary hypertension. Am J Med 1987;83:886–92.

ABSTRACT
The effect of long term oral hydralazine therapy (in doses ranging from 40–200 mg/day) was evaluated on pulmonary ventilation and gas exchange in 10 patients suffering from chronic obstructive airways disease (COAD) with pulmonary hypertension. All these patients had earlier shown a significant decrease in Paco2 and increase in Pao2 and minute ventilation, both at rest and during exercise, following 24 hours of treatment with hydralazine. These changes were maintained when the subjects were restudied after 6–18 months of chronic therapy. The minute ventilation and resting Pao2 were greater by 2.2 L/min and 5.8 mm Hg respectively, and resting Paco2 lower by 5.8 mm Hg as compared with pre-treatment values. Ten age and sex matched control patients exhibited no significant changes in ventilation or blood gases when restudied after 6–18 months. These results suggest that treatment with hydralazine may benefit patients with chronic cor pulmonale with COAD.

COMMENTS
The outlook for patients with valvular heart disease or coronary atherosclerosis has considerably improved during the past two decades but the prognosis of patients with COAD and chronic cor pulmonale continues to be poor. The only major progress in the management of these patients has been the use of long term oxygen therapy for 15 hours per day. This results in the disappearance of congestive heart failure, and amelioration of polycythaemia and pulmonary arterial hypertension. Unfortunately, this form of oxygen therapy is expensive and not readily available to Indian patients.

The pharmacological approach to the management of pulmonary hypertension has not been satisfactory. Vasodilators such as captopril, nifedipine, nitrates, prazosin or hydralazine have been reported to have variable effects on pulmonary artery pressure. Even if the pulmonary vascular resistance is decreased, pulmonary artery pressure does not fall because of a concomitant increase in the cardiac output. While evaluating the acute effect of hydralazine on arterial blood gases in patients with COAD, several investigators have reported an improvement in those with respiratory failure. This is surprising as some vasodilators, such as nitrates and nifedipine, worsen the arterial oxygen tension because they inhibit hypoxic vasoconstriction to the underperfused units of the lung. The observed beneficial effect has been attributed to a central respiratory stimulant effect of hydralazine. Moreover, the V/Q of underperfused units is apparently not reduced, although the pulmonary vascular resistance does fall as observed in the present study. Unfortunately, pulmonary artery pressure does not fall due to an increase in cardiac output. However, even a modest improvement in blood gas values of patients with chronic respiratory failure is likely to have a beneficial effect on the quality of life and long term survival. The findings of the authors that the improvement in ventilation and blood gas measurements is maintained after chronic administration of hydralazine are significant. It is possible that even pulmonary artery pressure may fall on long term therapy if cardiac output returns to pre-treatment values. The fall in pulmonary vascular resistance must be partly attributable to the amelioration of blood gas tensions.

Hydralazine in this study has been shown to have a mild respiratory stimulant action like medroxyprogesterone or almitrine. Lack of haemodynamic improvement is disappointing. The search for an effective oral pulmonary vasodilator must continue for the management of patients with pulmonary arterial hypertension secondary to chronic lung disease.

REFERENCES

J. N. PANDE