To the Editor:

We appreciate the comments from Dr. Wang and colleagues on our article (CHEST 1995; 108:670-76) based on their experience with a case of unilateral pulmonary artery agenesis (UPAA) in an adult.

Our statement that "many patients with UPAA had a benign clinical course" does not conflict with their observations. It is well known that patients with UPAA may develop pulmonary hypertension later in life as their patient did. However, by no means can it be stated that all adult patients with UPAA will have pulmonary hypertension at initial diagnosis or develop it later. The main objective of our article was to emphasize that UPAA is frequently misdiagnosed in adulthood and is often not considered in the differential diagnosis of the unilateral hyperlucent lung. Clinicians and radiologists should be aware of the possibility of undiagnosed cases in adults, with many atypical characteristics as our patients had.

It is not true that we did not mention whether there were any physical signs indicating the presence of pulmonary hypertension. A careful reading of the "Clinical Findings" section will reveal that we stated that "the remainder of clinical examination was unremarkable" in all patients. Our patient with mild exertional dyspnea and cardiac abnormalities had repeat clinical and transesophageal echocardiography (ECHO) examinations, and no signs of pulmonary hypertension were found.

Since transesophageal ECHO is a noninvasive technique and has been shown to be sensitive in detecting pulmonary hypertension, one could suggest it for follow-up of these patients instead of serial cardiac catheterization which is an invasive technique with possible complications.

In conclusion, we still believe that not all patients with UPAA will have pulmonary hypertension at the time of their initial diagnosis as none of our patients had. Whether or not they will develop pulmonary hypertension later in life is a matter for careful follow-up. Cardiac catheterization could be replaced by newer techniques for evaluation of this complication.

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Pulmonary Thromboembolism and Duplication of Inferior Vena Cava

To the Editor:

We read with interest the paper by Kouroukis and Leclerc (April 1996) on pulmonary embolism with duplicated inferior vena cava. Among the few reports on this topic, the authors should have mentioned our letter, which reported a fatal case of pulmonary thromboembolism in a young woman with stenosis and duplication of the inferior vena cava.

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REFERENCES

To the Editor:

We thank Dr. Dottorini for his interest in our paper. It is true that his letter should have been mentioned in our paper. We searched the literature under combinations of inferior vena cava, thrombosis, congenital anomalies, duplication, and pulmonary embolism. Unfortunately, his case report did not appear in our searches.

Thanks to Dr. Dottorini for pointing out our omission.

C. Kouroukis, MD Hamilton, Ontario Canada

REFERENCE

Pulmonary Involvement in a Case of Plasmodium vivax Malaria

To the Editor:

Complications involving the lung, kidney, peripheral blood, and CNS have been reported frequently in cases of Plasmodium falciparum infection, but they are extremely rare in other types of malaria. As far as we are aware, this is the first case of acute pulmonary interstitial pneumonia caused by Plasmodium vivax.

A 38-year-old Pakistani man was admitted to our hospital with a 1-week history of intermittent fever (35.5°C), pleuritic pain in the right side of the chest, cough, and mucus expectoration. Previously, a general practitioner had prescribed a 7-day course of erythromycin therapy and the respiratory symptoms disappeared, although intermittent fever, headache, diffuse arthralgia, and malaise persisted.

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