marked in pulmonary embolism, but the “causal” conduction defect involves only one portion of the left bundle branch.5

George Nikolic, MBBS, FCCP
Intensive Care Unit, Woden Valley Hospital
Woden, Australia

REFERENCES
2 Spodick DH. Q-wave infarction versus S-T infarction. Am J
Cardiol 1983; 51:913-15
3 Walder LA, Spodick DH. Global T wave inversion. J Am Coll
Cardiol 1991; 17:1479-85
1982; 50:213-22
5 Scott RC. The S1Q3T3 (McGinn-White) pattern in acute cor
pulmonale: a form of transient left posterior hemiblock? Am
Heart J 1971; 82:135-39

Asthma Therapy Clarified

To the Editor:

I am writing to express our concern regarding a recent
editorial, “Asthma Therapy: What’s New and Is It Necessarily
Better?” (July 1997).1 In this editorial, Dr. Georgitis wrote: “Clearly, physicians should document the total microgram dose per day of inhaled corticosteroids for patients (ie, beclomethasone 336 μg ×4, triamcinolone 600 μg ×4, fluticasone 440 μg ×4, or budesonide 1000 μg ×4 per day).” While the author may have simply intended to suggest that clinicians document inhaled corticosteroid use in their patients, some readers may infer that this budesonide dosage regimen is acceptable. It is not. The maximum recommended adult dose of budesonide via Pulmicort Turbuhaler (Astra, Westborough, Mass) is 800 μg twice daily (1600 μg daily), not 4000 μg daily as the regimen given would suggest.

Robert P. Baker, PharmD
Medical and Drug Information Services
Astra USA, Inc.
Westborough, Massachusetts

REFERENCE
1 Georgitis JW. Asthma therapy: what’s new and is it necessarily
better? Chest 1997; 112:3-5

To the Editor:

In my editorial, dosages for the inhaled corticosteroids were cited as examples. The correct inhaled dosage is presented in the tables from the new asthma guidelines, expert panel report 2. This is important because the doses for children are much lower than those
for adults, and there are specific daily ranges for low, medium, and high doses. In the sentence noted by Dr. Baker, the “×4” should be deleted so that the sentence reads correctly in that total daily dose of the inhaled corticosteroid is documented in the patient record.

John W. Georgitis, MD, FCCP
Department of Pediatrics
Bowman Gray School of Medicine
Winston-Salem, North Carolina

REFERENCES
1 National Asthma Education and Prevention Program. Expert
panel report 2: guidelines for the diagnosis and treatment of
asthma. NIH publication 55-4051. Bethesda, Md: National
Institutes of Health, 1997

Pulmonary Hypertension and Leukemia

To the Editor:

I read with great interest the report by Rossoff and colleagues of a man with a CD8/T-cell large granulocyte leukemia and “primary” pulmonary hypertension ameliorated by treatment with cladribine (August 1997). Several features of the patient’s pulmonary disease are striking for their similarity to those of tumor microembolism, leading me to wonder if “primary tumor microangiopathy” might be a more apt diagnosis.

The multiple bilateral subpleural shallow perfusion defects seen on the perfusion scan sound much like the “contour mapping” classically seen with lymphangitic carcinomatosis and tumor microembolism. Similarly, the described pathologic changes of the vessels are akin to those that may occur consequent to tumor microembolism. These changes of thrombosis, organization, and especially intimal proliferation with luminal obliteration, termed pulmonary tumor microangiopathy by von Herbay et al., were first recognized a century ago. They have since been described in numerous patients with tumor embolism, as recently reviewed.

Pulmonary tumor microangiopathy occurs in reaction to circulating malignant cells, usually carcinomas, lodging within the pulmonary vasculature. Pathologically, the condition resembles plexogenic arteriopathy. Although malignant cells can usually be seen in the vessels, the vascular changes are sufficiently characteristic to justify suspicion of tumor microembolism—even when the tumor cells themselves are not visualized—in the appropriate clinical setting. I believe this case presents an appropriate setting: although the terms tumor and embolism don’t apply with leukemia, the key feature of circulating malignant cells impacting in the pulmonary circulation is analogous. I would argue that the reported case is not one of remitting “primary” pulmonary hypertension, but is instead the first reported case of pulmonary tumor microangiopathy occurring with leukemia. The remission of the patient’s pulmonary hypertension is particularly exciting viewed in this light, as it suggests that pulmonary tumor microangiopathy is a potentially reversible process. The question of why some circulating malignant cells should trigger such a vasculopathy in the lung, while the majority clearly do not, is intriguing. This case is a reminder that the diagnosis of “primary” pulmonary hypertension should be made with extreme caution in the presence of a malignancy—including, apparently, a hematologic one.

Susan Murin, MD, FCCP
Division of Pulmonary and Critical Care Medicine
Department of Internal Medicine
University of California, Davis, Medical Center

REFERENCES
1 Rossoff LJ, Genovese J, Coleman M, et al. Primary pulmonary
hypertension in a patient with a CD8/T-cell large granulocyte
leukemia: amelioration by cladribine therapy. Chest
1997; 112:551-53
2 Sostman HD, Brown M, Toole A, et al. Perfusion scan in
pulmonary vascular/lymphangitic carcinomatosis: the seg-
mental contour pattern. AJR Am J Roentgenol 1981; 137:
1072-74

Downloaded From: http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21761/ on 06/27/2017