Blowing Soap Bubbles: Teaching Pursed-Lip Breathing

To the Editor:

A number of different techniques have been used to teach patients pursed-lip breathing. These techniques emphasize a prolonged expiration through pursed lips. This provides an external resistance to expiration that increases airway pressure.1 Pursed-lip breathing has been shown to decrease dyspnea when it is utilized with activities that produce tachypnea, which leads to progressive air trapping.2 Frequently, a candle analogy is used, that is, one should “bend the flame of the candle.” However, this metaphor is cumbersome in teaching patients pursed-lip breathing.

Using a child’s soap bubble wand and blowing one big soap bubble, patients will experience a perfect pursed-lip exhalation. Other benefits of blowing soap bubbles are: (1) measurable means of visualizing a pursed-lip exhalation; (2) immediate patient feedback; (3) noticeable relaxation of patients’ upper bodies and decreased use of accessory breathing muscles; and (4) an enjoyable group activity.

Patients should practice blowing soap bubbles at home when they are clinically well and in control of their breathing so that they will be comfortable utilizing the bubble-blowing, pursed-lip exhalation technique when they need to control dyspnea. Patients report that they experience better control of their dyspnea and fewer panic attacks when practicing this technique.

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REFERENCES

Primary Pulmonary Hypertension or Portopulmonary Hypertension?

To the Editor:

Rosoff and coworkers (August 1997)1 present a fascinating case of a patient with CDS/T-cell large granulocyte leukemia who showed regression of primary pulmonary hypertension (PPH) following treatment with cladribine. The authors also present an interesting and carefully thought-out hypothesis concerning possible interaction between the endothelial cell and the CDS/T-cell in the pathogenesis of pulmonary hypertension in this patient, and perhaps also in HIV-infected patients.

Although the authors stated that “our patient demonstrated severe pulmonary hypertension with all diagnostic studies highly characteristic of PPH,” the cardiac output and pulmonary vascular resistance in their patient were uncharacteristic of PPH. The PPH registry2 reported hemodynamic data on 187 patients with PPH and found a mean pulmonary artery pressure of 60 mm Hg, pulmonary vascular resistance of 2,080 dyne·sec·cm⁻⁵, and a cardiac index of 2.3 L/min/m². Although the authors’ patient had a comparable pulmonary artery pressure of 59 mm Hg, his pulmonary vascular resistance was only 369 dyne·sec·cm⁻⁵, and his cardiac output was a whopping 10.6 L/min. These values are much more consistent with the hemodynamic findings in portopulmonary hypertension (which may also show plexogenic pathology) and raise the question of whether the patient had leukemic infiltration of his liver and portal hypertension.3 Although a liver biopsy 15 years prior to the development of leukemia was reported to be normal, I wonder whether the authors have any data concerning liver function at the time of diagnosis of pulmonary hypertension. If leukemia involved the liver, it is possible that treatment with cladribine may have resulted in regression of portopulmonary hypertension. This possibility is further supported by a concomitant fall in their patient’s cardiac output—a pattern that would be typical for improvement in portopulmonary hypertension. In contrast, improvement in PPH is typically associated with an increase in cardiac output.4

The sustained regression of pulmonary hypertension is certainly impressive regardless of its etiology. Although cladribine treatment may have caused regression of pulmonary hypertension, I believe it is equally possible (from the data presented) that long-term treatment with nifedipine may have been at least partially responsible. Sustained regression of pulmonary artery pressure in PPH has been documented with calcium channel blockers and with continuous infusion of prostacyclin.5 At the time of the initial diagnosis of pulmonary hypertension in the authors’ patient, nifedipine resulted in a 31% decrease in mean pulmonary artery pressure and a 42% decrease in pulmonary vascular resistance. These decreases are similar to those reported by Rich and coworkers,6 who showed that patients with PPH who respond quickly to calcium channel blockers may have sustained hemodynamic benefit and regression of right ventricular hypertrophy.7 The authors’ patient may have had a response to calcium channel blockers that persisted despite discontinuation of these agents. If so, it may be possible to wean long-term vasodilator therapy in selected patients with PPH who show complete or...