low us to get more informative results, because a comparison of dyspnea sensation with or without the treatment program in the same subjects is more trustworthy than that in the different patient groups. However, we basically agree with the authors that the dyspnea treatment program without comprehensive exercise training do not always produce beneficial effects for COPD. Because the small reduction of dyspnea in the subjects of this study did not produce the improvement of exercise capacity as indexed by walk distance, the dyspnea management program alone may not be sufficient to contribute a better quality of life. However, the accompanying article in the journal showed that the effectiveness of pulmonary rehabilitation on exercise performance in COPD patients. The similar observation has also been reported by others. Taken together, there is a possibility that a combination of the dyspnea treatment program with the exercise training may be a more powerful modality to produce a better quality of daily living in COPD than the single treatment program, since an increased exercise capacity and a reduced sensation of dyspnea may dependently or independently contribute to improvement of some aspects of quality of life.

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REFERENCES
1 Stevens SS. Issues in psychophysical measurement. Psychol Rev 1971; 78:426-50

To the Editor:

We believe that the comments by Drs. Teramoto and Fukuchi agree with the basic conclusions from our study on the treatment of dyspnea in COPD. They suggest that exercise training may be an important component of pulmonary rehabilitation activities and that dyspnea treatment strategies alone are not sufficient to produce significant changes in symptoms or function for these patients.

With regard to our use of the Borg scale, we recognize that the Borg scale was derived from psychophysical properties. Our use of this scale in conjunction with exercise tests, such as the 6-min walk, is consistent with commonly accepted practice and with recommended use of the scale. In some applications the comparisons within subjects over time may have reduced variability with use of a standardized stimulus. However, a timed distance walk test like the 6-min walk performed in our study does not provide a standard stimulus since subjects are encouraged to exercise maximally and their walking speed and distance vary. Teramoto and Fukuchi suggest that our conclusions might have been different if we had standardized dyspnea ratings by walking distance. We performed the calculations as suggested using individual subject data to confirm that the results would still not be statistically significant and would not change the conclusions. The minimal differences in results between the raw and "standardized" dyspnea scores would be expected since the distance walked did not change significantly in the trial.

Regarding the comment about the use of a crossover experimental design, we do not believe that such a design would have worked well in this study. The control group was not a true no intervention group. The general health education program was used to control for attention and social interaction and support that might have influenced changes in symptoms or function. If we had crossed subjects over, we would have had to start everyone as a control to avoid any residual treatment effect before a subsequent control period after the experimental intervention. Since we also wanted to look at longer-term effects (6 months), we would have had to wait 6 months before interventions. This would have prolonged the study and made it much more difficult to enroll subjects.

In conclusion, studies now substantiate the benefits of pulmonary rehabilitation programs that include exercise conditioning for patients with chronic lung diseases. As suggested in the letter, we do not believe that dyspnea control strategies alone without exercise training are sufficient to induce significant changes in these patients.

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Fulminant Malignant Arrhythmia and Multiorgan Failure in Acute Arsenic Poisoning

To the Editor:

We had the opportunity to care for a patient developing early and fatal ventricular arrhythmias and multiorgan failure after the accidental ingestion of arsenic. The patient, a healthy 21-year-old man, accidentally ingested 21 g (300 mg/kg) of sodium arsenate. He was admitted to our hospital 30 min after the ingestion. Initial examination revealed a confused patient in moderate respiratory distress, complaining of abdominal tenderness and burning throat. On admission, blood pressure was 100/60 mm Hg, CVP 0 cm H2O, cardiac rhythm showed an atrial fibrillation at 110 bpm and anuria was observed. Arterial blood gases on mask at 50% oxygen were PO2 158, Pco2 24 mm Hg, pH 7.27, HCO3 11 mM. Glucose was 148, BUN 23, creatinine 1.4 mg/dl, Na 145 and K 3.4 mM, CPK 156 and LDH 293 IU/L, Hb 17 g/L, WBC 3,400 cells/mm3; other measurements included normal coagulation studies. Serum arsenic level was 300 µg/L. Despite immediate management with supplemental oxygen, aggressive volume expansion, correction of metabolic disturbances, forced diuresis, gastric lavage, administration of activated charcoal, and treatment with dimercaprol (250 mg IM), the patient developed progressive hypotension and refractory ventricular fibrillation with

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