Balloon Catheters

To the Editor:

We read the article of Eschenbacher and Gravelyn (Chest 1987; 92:105-09) and agree upon the potential widespread application in pulmonary research of their novel technique. We also tried (unpublished data) this technique with a single balloon-catheter (arterial embolectomy catheter 5 EMB 80, size 1.66, length 80 cm, Shiley Inc, Irvine, CA) passing through the vocal cords with fiberoptic bronchoscopic guidance. But we abandoned it as it was difficult—sometimes impossible—to pass the vocal cords, and we prefer to introduce the catheter in the channel of the Olympus IT-10 fiberoptic bronchoscope (inner diameter 2.6 mm).

In this way, we are able to reduce the time for bronchoscopy and consequent hypoxemia, and to do it more safely for the patients. Using this single-balloon catheter, we can also observe the bronchial lumen during the introduction and suction of the saline solution, so we obtain a good seal and more regular recovery of fluid.

Further changes are needed to improve our novel technique.

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Increased Mitral Valve Insufficiency During Precordial Whoop

To the Editor:

I enjoyed reading the interesting article "Increased mitral valve insufficiency during precordial whoop" by Shaikh et al (Chest 1988; 92:209-10). However, I was so frustrated by my effort to find the phonocardiograms referred to in Figure 1 that after ten minutes of meticulous search I gave up and merely blamed the copy editor of Chest for having inadvertently cropped off the phonocardiograms from either the top or the bottom of the two panels of the composite figure. Then, all of a sudden, my peripheral vision caught glimpses of those three unlabelled late systolic whoops, more noticeable in the first and second cycles than in the third, superimposed on the dense posterior-wall and pericardial echoes of the M-mode echocardiogram in the lower panel. Since other readers might have the same difficulty as I did, perhaps Figure 1 of this article should be reprinted with the late systolic whoops clearly labelled. Incidentally there is a typographical error in the legends of Figure 1: S = interventricular (not intraventricular) septum.

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Adult Onset Still’s Disease

To the Editor:

In their description of a patient with adult onset Still’s disease and severe restrictive pulmonary defect,1 Cantor et al (Chest 1987; 92:939) claim that respiratory muscle function abnormality could have contributed to the observed restrictive defect. This was based on the decreased maximum inspiratory mouth pressure (≈ 40 cm H2O). Although they mention that the patient was more dyspneic in the supine position, they failed to notice whether he had any paradoxical abdominal movement or whether the vital capacity fell with change of posture from upright to supine. These are simple clinical clues to suspect diaphragmatic weakness.2 Pleural and gastric pressure recordings during tidal breath and maximal static inspiratory effort could further substantiate this diagnosis.

Furthermore, they found only two reports of adult onset Still’s disease associated with impairment of pulmonary function. They overlooked our report3 of a similar patient in whom diaphragmatic weakness was clinically evident and was documented by transdiaphragmatic pressure measurement (Fig 1). Vital capacity was reduced to 45 percent of predicted in the sitting position and fell by 510 ml in the supine position. Unlike their patient, there was no radiologic pulmonary infiltrate which could have contributed to the restrictive defect. We thought that the diaphragmatic weakness was part of a generalized myopathy suggested by an elevated aldolase level (serum creatine kinase level was normal), abnormal electrocardiograms and muscle biopsy results. None of these test results were reported by Cantor et al. On the other hand, myocardial biopsy was done while hemodynamic measurements were normal.

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REFERENCES
2 Loh L, Goldman M, Davis JN. The assessment of diaphragmatic function. Medicine 1977; 56:165-69

To the Editor:

Dr. Braidy has brought to our attention another report of a patient with adult onset Still’s disease and an associated restrictive pulmonary defect. Unlike our patient, however, there was no pulmonary infiltrate and the initial residual volume was 130 percent of predicted. An elevated residual volume in association with a low total lung capacity in the absence of obstructive airways disease, as seen in the patient reported by Dr. Braidy, is suggestive of inspiratory and expiratory muscle weakness. The presence of respiratory muscle dysfunction is further supported by a reduction in the vital capacity from sitting to supine positions and the demonstration of decreased transdiaphragmatic pressure.

Although our patient did not demonstrate paradoxical abdominal movement with breathing, this does not rule out respiratory muscle dysfunction. Low maximal respiratory pressures and increased dyspnea while lying supine as observed in our patient may reflect respiratory muscle weakness. Unfortunately, the presence of pulmonary infiltrates confounds our ability to use the results of