parietal thrombi central from the periphery had occurred. Superficial thomboemboli of all extremities were observed without general vasculitis. No causal relation between chronic myelocytic leukemia and pulmonary arteritis was obvious.

This case does not fit into the common categories of pulmonary angiitis:1 Wegener's granulomatosis, allergic angiitis and granulomatosis, necrotizing sarcoid granulomatosis, lymphomatomoid granulomatosis, and bronchocentric granulomatosis. In the literature we found only one case report2 describing primary lymphocytic pulmonary arteritis with thrombosis. Thus, we believe that our patient represents a very rare case of primary pulmonary arteritis.

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Pleural Determinants of Restrictive Lung Function and Respiratory Symptoms in an Asbestos-exposed Population

To the Editor:

Pleasure at seeing one's papers cited is lessened when the citation appears to give one's support to a rejected argument. The article by Broderick and colleagues1 that appeared in the March 1992 issue of Chest contains the following statement: "...there is a reduction in the diffusing capacity of carbon monoxide in patients with pleural disease and normal-appearing parenchyma on the chest x-ray film, ...a finding that suggests the presence of underlying parenchymal abnormalities." 1 I helped write one of the references2 cited by Broderick et al in support of that statement; although we did report the finding mentioned, we did not arrive at the same conclusion. As Dr. Broderick says earlier in her paper, impaired chest expansion reduces the transfer factor, but the transfer coefficient rises. In 1977, I found no published data on the relationship of the coefficient to lung volume in normal persons; later Lipscomb and colleagues3 showed that it is curvilinear and published limits for the values in healthy subjects. The results in our patients fell within those limits, so we did not have to suggest parenchymal disease to explain our results, which could all have been due to pleural restriction of lung expansion.

Of course pleural and lung changes occur together. Some of the original cases of Burton Wood and Page4 had pleural changes, confirmed at postmortem. Glynne,6 discussing the pathology of asbestosis, said, "Old tough adhesions are common...they may be extensive on the one side and almost absent on the other...Apical adhesions are...not uncommon, and the interlobar fissures may be completely closed. In advanced cases complete symphysis pleurae is found...Towards the bases the pleura becomes thicker...in the portions showing more advanced disease the thickening becomes sufficiently great to give a ground-glass look to the pleura, and in the thickest part of all it becomes stiff, yellow, almost horn-like in appearance."

Cases seen now may have no cracks or finger clubbing, minor breathlessness, and radiographic shadowing and lung function abnormalities entirely explicable by pleural thickening. We showed that in such cases computed tomography can show normal lungs. Our evidence (confirmed by others) led to the prescription of diffuse bilateral asbestos-induced pleural thickening as an industrial disease in the United Kingdom separate from asbestosis. It would be far less satisfactory to diagnose asbestosis in such cases because "10 to 15 percent of patients with histologic evidence of interstitial fibrosis have been reported to have normal-appearing parenchyma on the chest x-ray film," as Broderick et al suggest; the overwhelming majority of patients diagnosed and compensated this way would not have asbestosis.

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To the Editor:

We appreciate the comments of Dr. Wright and apologize for the perceived misinterpretation of his article.1 In general, we agree with Dr. Wright. We, too, believe that asbestos-induced pleural fibrosis is a disease independently associated with restrictive lung function. This strong association is supported by several independent investigations.1-4 However, the mechanisms underlying this association have not been clearly identified. We believe that this is very likely a multifactorial process. Individuals with pleural fibrosis are more likely to have asbestosis than those with normal pleura, and individuals with pleural fibrosis may have limited expansion of their chest wall simply due to the scarring of the parietal and/or visceral pleura. Although previous studies have shown that 10 to 15 percent of patients with histologic evidence of interstitial fibrosis have been reported to have normal-appearing parenchyma on the chest x-ray film,1 we have previously shown1 that over 50 percent of individuals with pleural disease and normal-appearing parenchyma on the chest x-ray film have parenchymal abnormalities on a high-resolution computed tomographic scan that are consistent with asbestosis. Alternatively, given the extensive chest wall scarring in some individuals with asbestos-induced pleural fibrosis, restricted chest wall motion must account for some of the reduced lung volume among individuals with asbestos-induced pleural disease.

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Nasal Mask Ventilation in Acute Respiratory Failure

To the Editor:

I read with great interest in the September 1992 issue of Chest the data reported by Benhamou and colleagues concerning nasal mask ventilation (NMV) in acute respiratory failure (ARF). Intermitent positive-pressure ventilation by nasal mask was assessed as first-line therapy in order to avoid endotracheal intubation in very elderly patients or as the therapeutic last resort. The patients were ventilated with a volumetric respirator, and artificial ventilation was performed in the controlled mode (volume-cycled ventilation). Although reported data seem relatively encouraging, several points have to be discussed. Nine of 30 patients had an immediate deterioration, resulting in 5 deaths. Cases of poor tolerance were related to poor adaptation to the ventilatory mode. Correction of hypercapnia required several hours (and was probably related to the severity of buccal leaks), whereas PaO₂ improved early. Finally, conjunctivitis was observed in poorly adapted patients as a consequence of air leaks.

Merely using a nasal mask is not sufficient to ensure optimal tolerance of artificial ventilation. The second crucial condition is the use of a ventilatory mode allowing synchronization between the patient and the machine, by using a specific respirator for noninvasive ventilation. Recently published data have shown that using a flow-triggering system (vs a pressure-triggering system) with a decelerated inspiratory flow (vs a constant inspiratory flow) appears to be the best way to decrease the work load during assisted ventilation. Decelerated inspiratory flow is obtained by application of a constant positive airway pressure synchronized with spontaneous inspiration (pressure support). Initiation of inspiratory flow being a major determinant of respiratory work, early application of pressure and stable inspiratory airway pressure (inspiratory positive airway pressure [IPAP]) provides optimal matching of the patient's needs. Assessing the effects of noninvasive partial ventilatory assistance with constant positive pressure, either one-level (ie, IPAP) or two-level (ie, bilevel positive airway pressure [Bi-PAP]), in ARF, these recent studies have shown a good clinical tolerance with better patient comfort, reduction of respiratory rate, and rapid improvement of blood gas values.

Although several recent studies have reported successful use of noninvasive pressure-support ventilation via facial mask (less potential risk of air leaks than with a nasal mask) and conventional respirator, it is probable that optimal noninvasive ventilation requires a specific respirator with an inspiratory flow-triggering system, early and stable inspiratory pressure support (sorvico-controlled loop), a compensatory system of air leaks, an expiratory flow-triggering system (eg, triggered by a percentage of inspiratory peak flow), and passive exhalation through a low-resistance device (eg, Whisper Swivel, Respiromics, Murrysville, Pa). This spontaneous mode of ventilation is flow-cycled and includes a security system relative to the switch from inspiration to expiration (detection of expiratory effort, duration of inspiration), so that the patient never exhales against the pressure support. Furthermore, with a compensatory system of air leaks, specific ventilators allow speech and feeding without removal of the nasal mask. Using a conventional ventilator with a nasal mask may be difficult because of the absence of a compensatory system for air leaks and the potential importance of extra work involved in breathing.

Ideal NMV in ARF involves the maintenance of spontaneous breathing, with the machine supporting the patient's efforts by an appropriate mode of assistance. The clinical tolerance is the crucial point, and as Benhamou et al themselves said: "This good general tolerance . . . is the only factor that was found to have a prognostic value in our study." It is possible that Benhamou et al are using a "good wheel" (the nasal mask) but not a "good motor" (the ventilatory mode) and that clinical tolerance (with fewer air leaks and earlier correction of hypercapnia) could be better yet with a synchronized mode of ventilation.

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