parietal thrombi centrad from the periphery had occurred. Super-
ific thrombophlebitides 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 
angitis.1 Wegener's granulomatosis, allergic angitis and granulo-
matosis, necrotizing sarcoid granulomatosis, lymphomatoide granu-
matosis, and bronchocentric granulomatosis. In the literature we 
found only one case report1 describing primary lymphocytic pul-
monary 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." 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 
results the transfer factor, but the transfer factor coefficient rises. In 
1977, I found no published data on the relationship of the coefficient 
to lung volume in normal persons; later Lipcomb 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. Gloyne,5 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 
pleuriae 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 craddle 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 independ-
ent investigations.1-1 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,1-1 we have previously shown1 that over 50 percent 
of individuals with pleural disease and normal-appearing paren-
chyma 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). Intermitting 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 PaO$_2$ 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 (servo-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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