Table 1 in the article by Mathru et al1 shows that when the pericardium was opened, the cardiac index fell from 2.9 to 2.8, coupled with a rise in heart rate from 63 to 64. This should cause a fall in stroke volume of 5.0 percent, not the 1.7 percent rise (from 50 to 60 ml) claimed in Table 2 in that article. Likewise, Table 3 shows no change in cardiac index. However, that cardiac index value coupled with an increase in heart rate from 66.9 to 69.5 together should cause a fall in stroke volume of 3.7 percent, not the 16.3 percent increase (from 66.1 to 76.9 ml) shown in Table 4.

The total errors of 6.7 percent in group 1 and 20.0 percent in group 2 imply a large enough error in stroke volume and RVEF to call into question the authors' conclusions that the "right ventricle enlarges after opening the pericardium," and that the RVEF decreased in group 1 but not in group 2. Also, with such large differences between RVEF (39.1 percent and 36.8 percent) and ejection fraction area (55 percent and 61 percent) in group 2, studies are needed to determine whether technique, RVEF thermodilution or echocardiography, is accurate.

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Pulmonary Alveolar Microlithiasis

To the Editor:

In the July 1990 issue of Chest, Pant et al4 reported a case of typical pulmonary alveolar microlithiasis (PAM). In that communication they stated that peripheral white lines are rarely reported on chest roentgenogram in PAM and suggested that those white lines are the result of associated pleural calcification.

From 1965 to 1984 I myself studied eight cases of PAM. Each case had been authenticated by lung biopsy after thoracotomy. Among those eight cases, five were found to have pencil-thin, sharp, dense white lines surrounding (more or less) the lungs, the heart, or the mediastinum. In describing four of those cases in 1970, Lahreche2 stated that peripheral pulmonary miliary disseminated densities associated with white lines are almost pathognomonic for PAM. Since in each case a thoracotomy was performed, it was possible to see and study directly the pleura. We never saw calcification, but only the pleura thickened by a fibrohyaline process.

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References
2 Lahreche H. La microlithiase alvéolaire pulmonaire: à propos de 4 nouveaux cas authentifiés découverts en Algérie (thesis). University of Algiers, 1970

To the Editor:

In almost all reported cases of PAM, the lesion is restricted to the lungs. The presence of pleural calcification and nephrolithiasis makes our case unique, rather than "typical," especially since it raises, along with a few other reports of multiorgan calcification, the possibility of PAM being a systemic disorder. Winzelberg et al5 have also reported pleural calcification depicted on computed tomographic scans of a patient who underwent thoracotomy. In the more than 200 cases reported in the English-language literature, peripheral white lines have been reported only twice before.6,7 Furthermore, this feature is not mentioned in standard English-language texts of pulmonology8 and chest roentgenology.9 In fact, Dr Felson10 considers the black pleural line diagnostic of PAM. Unfortunately, we had no access to the report of Dr Petit's cases, which was published in French. In view of his observation, the presence of these lines in PAM may also represent fibrohyalineization of the pleura.

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Upper Airway Obstruction: Anatomic-Functional Relationship

To the Editor:

In the July 1990 issue of Chest, Shepard et al11 discussed cross-sectional area changes in obstructive sleep apnea (OSA) detected with computed tomography (CT). The dimensions of the upper airways have, indeed, often been evaluated in OSA, and they have also been compared with airflow resistance measurements.12 It is amazing that in upper airway obstruction (UAO) at the level of the larynx and especially of the trachea, studies correlating functional data with diameters or cross-sectional area during quiet breathing and/or forced maneuvers are almost lacking. Miller and Hyatt13 obtained data in experimental conditions in which they made healthy subjects breathe through added resistances with internal diameters varying between 4 and 13 mm and measured the corresponding changes in PE F, PEF, and MEFw (Fig 1). The question is, however, to what extent these results may be applied to UAO.

As a first approach to this problem, we investigated whether the relationship of functional indices in UAO was similar to that in the experimental model of Miller and Hyatt13 (Fig 1). In 18 patients with UAO14 (11 with variable extrathoracic UAO, five with fixed UAO, and two with variable intrathoracic UAO), we measured PE F, PEF, and MEFw and found that the relationships were similar to those in the model of Miller and Hyatt.13 It is, therefore, tempting to conclude that this indicates that in UAO the diameter at the level of the obstruction during forced expiration may be estimated from the superposition of the functional results on the data of Miller and Hyatt.13

References
2 Balkan JP, Fuleihan FJD, Nucho CKN. Pulmonary alveolar microlithiasis: report on 5 cases with special reference to roentgen manifestations. AJR 1968; 103:508-18
3 Cole WB. Pulmonary alveolar microlithiasis. J Fac Radiol 1959; 10:54-56
These data stress the need for direct comparative studies of anatomic-functional correlations in UAO. The difficulty, however, is that these studies should be done during quiet breathing as well as during forced maneuvers.

**REFERENCES**


To the Editor:

We appreciate the comments of Drs Melissant and Demedts and agree with them that physiologic (spirometric) data indicating obstruction to airflow should predict the extent of anatomic narrowing. Ideally, imaging techniques should identify regions of upper airway narrowing that would correspond approximately to the orifice diameters used by Miller and Hyatt to produce similar decrements in the spirometric variables reflecting airflow. However, in order to obtain optimal functional-anatomic correlations, imaging must be performed under identical conditions of pressure, lung volume, and upper airway muscle activity. Our CT data, along with those of Kuna et al, have documented changes in upper airway dimensions with pressure, while Brown et al have shown that upper airway size varies directly with changes in lung volume. Recent work with fast CT scanning has additionally revealed significant variation in upper airway dimensions during quiet tidal breathing, which would account for the intrabreath variations in airways resistance that have long been observed. The importance of upper airway muscle tone to the maintenance of airway patency is widely acknowledged, especially during sleep. With these caveats in mind, we concur that future studies of the upper airways should combine physiologic assessment of function with imaging-based evaluation of upper airway anatomy.

**REFERENCES**