Cardiac vs Pulmonary Amyloidosis

To the Editor:

The recent study by Celli and associates1 on pulmonary involvement by deposits of amyloid in conjunction with systemic amyloidosis provides important information. Systemic amyloidosis is an unusual disease, and thus few patients are available for study. Observations on the actual clinical impairment by amyloid pulmonary involvement detected at necropsy have been few. As Celli et al1 appropriately noted, a distinction between derangements of pulmonary and cardiac physiologic functions by amyloidosis may be clinically difficult. In addition to the laboratory studies recommended by Celli et al1 for evaluation of these two possibilities, I would add the echocardiogram. If cardiac infiltration by amyloid is sufficient to cause clinical symptoms, the echocardiogram reveals symmetric thickening of the interventricular septum, left ventricular free wall, and usually the right ventricular free wall, with associated hypokinesia of these walls.2 Although such findings will not rule out associated pulmonary disease, they will provide evidence of clinically important cardiac involvement. Conversely, the absence of such an echocardiographic pattern may not exclude early cardiac infiltration by amyloid but does make its clinical significance doubtful.

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REFERENCES

Position and Positive End-Expiratory Pressure in Lobar Atelectasis

To the Editor:

In the report by Fowler et al1 entitled "Positive End-Expiratory Pressure in the Management of Lobar Atelectasis," information regarding the posture of the patient during treatment with positive end-expiratory pressure (PEEP) is needed to properly assess the value of this mode of therapy. The application of PEEP could potentially worsen the existing hypoxemia associated with localized atelectasis by producing a shift of blood flow from the normal lung to the less compliant lung.2 In order to prevent this possibility, it seems best to ventilate the patient with the atelectatic side up, so that blood can gravitate to the normal lung.

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To the Editor:

In response to the inquiry of Sadnouri and Heurich, all patients in our study (Chest 74:497-500, 1978) were treated in the recumbent position, with periodic rotation from side to side. Specifically, no attention was paid to maintaining the atelectatic lung in the upright position. Although the possibility of positive end-expiratory pressure (PEEP) increasing hypoxemia in the manner suggested is physiologically sound, from a practical standpoint, no patient demonstrated a fall in arterial oxygen pressure (PaO2) after initiation of therapy.