Different Conclusions from Similar Data

The Half Empty Glass

Is the glass half full or half empty? This question applies to the report in this issue (see page 75). Farman and Speir describe a retrospective analysis of patients from a teaching institution, similar to one previously reported by Khan et al.1 These two articles come to different conclusions. Farman and Speir state that the roentgenographic manifestations were typical and that atypical roentgenographic presentations of pulmonary tuberculosis are still uncommon. Khan et al, on the other hand, emphasize the "unusual" presentation of their patients.

Comparison of two retrospective analyses is fraught with danger. Different populations with the same disease may manifest their illness in different manners. Criteria for inclusion into the study may vary. The bias of the authors may cause the interpretation of the results to vary. Nonetheless, to resolve the conflict of the half full versus the half empty glass, an attempt to resolve these differences can be made. Farman and Speir report pleural effusion alone in three patients in their series (5.9 percent) of the 51 with pulmonary tuberculosis. Khan et al noted five of 88 with this finding (5.7 percent), a nearly identical prevalence. The frequency of lymphadenopathy was similar also: 5.9 percent in the present report compared to 6.8 percent in the previous report. Thus, the occurrence of probable primary pulmonary tuberculosis is also comparable for these two groups with nine instances (17.6 percent) of pleural effusion and hilar adenopathy reported by Farman and Speir, and 12 instances (13.5 percent) by Khan et al. Again, when the prevalence of lower lung field tuberculosis is analyzed, the data appear similar to Farman and Speir's report of three of 48 patients (6.25 percent) with pulmonary tuberculosis and parenchymal involvement and six of 88 patients (6.28 percent) in the Khan et al series. Despite differences in time and place of the two studies, these are remarkable likenesses. Pulmonary nodules occurred in eight patients (15.7 percent) reported by Farman and Speir and also in eight of Khan's for prevalence of 9.1 percent. Applying the criteria for "unusual" pulmonary tuberculosis to the present series, one finds that 33 percent can be classified in this category. This is comparable to the 34 percent of Kahn et al.

While both studies substantiate that pulmonary tuberculosis presents with upper lung field cavity infiltrates in the majority of cases, the data in both also make the important point that this curable cause of pulmonary disease must be considered when the chest roentgenogram is not typical, especially when the clinical course is not characteristic of a nontuberculous pneumonia or when the infiltrate fails to resolve in the expected fashion. Absence of upper lobe involvement should not dissuade one from considering tuberculosis as a possible diagnosis when the etiology of pulmonary infiltration is not readily apparent. Multiple examinations need to be carried out. When the etiology continues to remain a mystery, tissue must be obtained for histologic examination as well as culture. That the pattern of presentation of tuberculosis may be changing is further substantiated by Farman and Speir in their finding of extrapulmonary tuberculosis comprising 32 percent of their total of 75 patients. We must remain vigilant to the possibility of this disease which can be so devastating when undiagnosed and thus untreated.

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REFERENCE


Sleep Apnea, Hypoxemia, and Cardiac Arrhythmias

To even the casual observer, there is some relationship between hypoxemia and cardiac dysfunction. However, the specific parameters of this relationship appear to be anything but simple and pellucid. Patients with obstructive sleep apnea (OSA) exhibit profound hypoxemia during apneic episodes, as well as a wide variety of ventricular and supraventricular arrhythmias. In studying polysomnographic records of patients with OSA, one is soon
struck with the complexity of the relationship between the frequency, type and duration of apnea, the degree of arterial desaturation, and the type and incidence of cardiac dysrhythmias.

Recent investigations have demonstrated quite different relationships between arterial desaturation associated with apnea, and concomitant ventricular and supraventricular arrhythmias. Guilleminault et al. found no consistent relationship between the degree of hypoxemia and ventricular arrhythmias, while Zwillich et al. demonstrated a strong positive correlation between the level of arterial oxygen desaturation during an apneic event and the degree of bradycardia. It is axiomatic that longer apneic episodes will produce a greater degree of oxygen desaturation, and according to the study by Zwillich et al., a predictable decline in heart rate. In fact, they state that virtually every apnea is associated with some degree of bradycardia. The mechanism for this appears to be enhanced vagal tone, probably due to a decrease in reflex respiratory inhibition of vagal tone. Ventricular ectopy is not so manifestly predictable.

In their study of hypoxemia and ventricular arrhythmias in OSA, Shepard and colleagues have provided excellent data which substantially enhance our understanding of this complex relationship. In their study, as well as the extensive study by Guilleminault et al., no correlation was noted between episodes of ventricular dysfunction and arterial oxygen saturation. The data of Shepard et al. suggest that there is a threshold of oxygen saturation below which the risk of encountering ventricular ectopy is markedly increased. They noted a twofold increase in PVC frequency in a subgroup of 16 patients whose arterial oxygen desaturation reached 60 percent or less. This threshold effect explains quite nicely why the correlation is low when the full range of arterial oxygen saturation is evaluated. In effect, there is minimal correlation until the oxygen saturation goes below 60 percent.

Should we become too encouraged that an important and useful parameter has been unmasked by these findings? The authors themselves point out that the increase in ventricular ectopy in patients with oxygen saturations below 60 percent was due primarily to seven patients who had a marked increase in PVCs. Two of the other patients showed no increase in ventricular ectopic activity with comparable oxygen desaturation. However, over half of the 31 patients studied exhibited complex ventricular arrhythmias. It is evident from this result that complex arrhythmias are present with oxygen saturation above 60 percent. Thus, oxygen saturation does not necessarily afford protection from malignant ventricular ectopy. It seems clear that hypoxemia alone does not predict ventricular dysfunction. Obviously, there are other factors which can contribute to the development of an irritative ventricular focus. Among these would be the balance of the sympathetic and parasympathetic input to the heart, myocardial effects of repeated episodes of hypoxemia and local alterations in myocardial blood supply. Sympathetic/parasympathetic balance is also undoubtedly disturbed by catecholamine discharge associated with repetitive obstructive apnea. Clearly, these are complex interactions which remain poorly understood in this clinical entity.

There are useful treatment implications in the data presented in the study by Shepard et al. Although a relatively high degree of oxygen saturation does not afford protection from ventricular ectopy, it seems clear from these data that saturation levels below 60 percent do increase the probability of a ventricular arrhythmia. Thus, patients obtaining apnea-associated oxygen saturation levels in the range of 60 percent or below should be considered doubly at risk since they are more likely to experience an increase in ventricular arrhythmias, and the previously cited work of Zwillich et al. suggests that this level of oxygen desaturation would also be associated with profound bradycardia. Such individuals should be singled out for more immediate aggressive treatment intervention.

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REFERENCES
1 Guilleminault C, Connolly J, Winkle RA. Cardiac arrhythmia and conduction disturbances during sleep in 400 patients with sleep apnea syndrome. Am J Cardiol 1983; 52:490-94

Lung Reexpansion—For Better or Worse?

In the current issue of Chest (see page 70) a second potential hazard of rapidly evacuating persistent pneumothorax is described—"reexpansion hypotension." It has long been appreciated that sudden evacuation of pneumothorax may cause edema of the underlying lung (reexpansion edema, REE). Protein concentration in edema fluid is high, and pulmonary vascular pressure normal or low suggesting pulmonary vascular injury as a cause of REE. Recently, a more acute form of REE has been reported with lung reexpansion after only four hours of atelectasis. We