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 (17) 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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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
have observed that protein concentration in edema fluid is also high with this more acute form of REE.

The cause of altered pulmonary vascular permeability is unclear. Theoretically, vascular injury could be caused by atelectasis itself, by mechanical stress to normal vessels during reexpansion, or by oxygen-derived free radicals generated by restoration of perfusion and ventilation to previously hypoxic areas of lung. Pulmonary blood flow is reduced by atelectasis, and we have observed that blood flow is restored towards normal by reexpansion, consistent with the hypothesis that re-perfusion may contribute to edema formation.

What causes vascular injury? Hypoxia is often cited as a cause of altered pulmonary vascular permeability, but breathing an hypoxic gas mixture does not reliably cause pulmonary edema. However, with atelectasis, hypoxia of the lung may be more severe than occurs breathing hypoxic gas mixtures because oxygen delivery to the lung is reduced by absent ventilation, hypoperfusion, and a low CVO₂. Thus, areas of hypoxia may exist that cause vascular injury directly or that promote free radical formation and lung injury with restoration of ventilation and perfusion. We have observed that an increased FIO₂ (40 percent [0.4] O₂) that prevents systemic hypoxemia during pneumothorax, prevents edema when lungs are reexpanded. This lends support to the hypothesis that hypoxia in some way contributes to the mechanism of injury.

In favor of a mechanical stress being the initiating event are observations that less vigorous attempts to reexpand lungs are less likely to cause edema. However, slow rates of reexpansion may favor slow rates of re-perfusion and could thereby lessen the effects of a re-perfusion type of injury.

Reduction of interstitial pressure during lung re-expansion is also a theoretic cause of edema. If one thinks of interstitial fluid as a liquid that transmits changes in pleural pressure to alveolar walls, sudden reexpansion of stiff lungs could reduce interstitial pressure and promote transudation of fluid into the interstitium. This implies a hydrostatic basis of edema formation which is inconsistent with the observations of high protein concentrations in edema fluid. However, such a mechanism might enhance the rate of edema formation in the presence of pre-existing vascular injury.

Now, a new side effect of lung reexpansion has come to light, namely: its potential to cause adverse hemodynamic effects. Pre-existing volume depletion, rapid translocation of fluid into the thorax, and myocardial depression all appear to contribute to circulatory failure. The mechanism of myocardial depression is unknown. The presence of a circulating myocardial depressant factor is one possible cause, perhaps analogous to what has been observed after occlusion and reperfusion of splanchnic vessels. The injured lung would be an obvious candidate for the release of such a factor. Clarification of this issue will require further investigation.

At present, it would seem prudent to adopt a cautious attitude in treating patients with long-standing pneumothorax (or pleural effusion). Although not rigorously tested, current evidence suggests that the volume of pneumothorax be reduced gradually. Although a majority of patients may tolerate lung re-expansion without adverse consequences, the patients described in this issue of Chest attest to the fact that traditional therapy of pneumothorax is not uniformly benign.

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