

Atelectasis and Shunting

ATELECTASIS strictly speaking means imperfect expansion, and applied to the lung it connotes airspace collapse. Complete airlessness is not necessarily implied. In practice atelectasis has accounted for the clinical picture, often seen two or three days following operation, characterized by fever, tachycardia and homogeneous densities in the chest roentgenogram; sometimes dyspnea and coughing are also present. Bronchial obstruction is considered a leading cause, although W. Pasteur in 1910¹ had already proposed "arrest of diaphragmatic movement" as the primary event.

Hamilton and his co-workers—in a paper published in this issue of *ANESTHESIOLOGY*—suggest that atelectasis may be present without roentgen-ray evidence. They view this type of atelectasis as widespread ("miliary"), rather than localized and well defined. Accordingly this lesion has *no* specific roentgen-ray appearance, except that which may be caused by a reduction in lung volume, *i.e.*, elevation of the diaphragms and increased density of vascular and bronchial markings. Their observation supports the view that a homogeneous density on the roentgenogram is in most cases caused by fluid retention in that area.

If chest roentgenogram is of little use in the diagnosis of this important type of atelectasis, how then can we make the diagnosis? A change in lung volume could occur with atelectasis; it does not necessarily happen, however, and the measurement is difficult to make.² Atelectasis is a cause of reduced pulmonary compliance,³ but so are many other factors, including a change in surface tension.

In recent years *increased physiologic shunting* has been observed during and following anesthesia,^{4, 5, 6} and atelectasis has been implicated as a cause. The physiologic shunt refers to that part of the cardiac output which has not participated in alveolar-capillary gas exchange. The shunt blood is returned to the left heart unoxygenated and constitutes what is also called "venous admixture" to arterial blood.

Atelectasis can indeed cause shunting, but does not invariably do so; it all depends on whether the collapsed airspaces continue to be perfused. Furthermore, there *are* other causes of shunting, and in order to avoid the impression that shunting is equivalent with atelectasis, the causes of shunting must be reviewed.

The bronchial, pleural and Thebesian veins contribute to the anatomical shunt, which varies relatively little and accounts for only about 2 per cent of the cardiac output.⁷ In addition, we recognize three intrapulmonary causes of shunting: diffusing block, uneven distribution of ventilation in relation to perfusion, and atelectasis.

The term "diffusing block" (or "diffusing gradient") embraces the factors opposing diffusion of an oxygen molecule from alveolus to erythrocyte in the pulmonary capillary. The most important of these factors are a reduced partial pressure gradient and an increased distance (increased thickness of the membrane). As a cause of shunting a diffusing block has little significance, certainly when alveolar oxygen tension exceeds 100 mm. of mercury⁸; alveolar proteinosis may be one of the rare examples of a "true" diffusing block.

A diffusing block should not be confused with diffusing capacity which is a measurement also dependant on the *area* available for diffusion. Diffusing capacity is not a specific measurement of diffusing block; on the contrary, the most frequent causes of a decreased diffusing capacity are atelectasis and uneven distribution. In these conditions the area available for diffusion is decreased.

Discounting a diffusion block we are left with uneven distribution and atelectasis as frequent causes of increased shunting. Hamilton and his co-workers suggest that these two disturbances be considered as one. On the premise that it is obviously impossible to ascertain if an alveolus is half collapsed or completely collapsed, they conclude that a distinction between atelectasis and uneven distribution is impractical. Unfortunately, both

premise and conclusion are questionable. The distinction between uneven distribution and atelectasis is made on the basis of whether an airspace is hypoventilated or not ventilated at all, *not* on the basis of the degree of collapse.

A differentiation between atelectasis and uneven distribution is certainly possible. If some alveoli are hypoventilated, with room air, the partial pressure of oxygen in them will fall; the deficit in total pressure is made up in small part by carbon dioxide, but mostly by nitrogen. In this situation the capillary blood in the hypoventilated area is poorly oxygenated and a shunt established. When breathing 100 per cent oxygen no "third" gas such as nitrogen is present; under these circumstances hypoventilation of some alveoli will cause the oxygen tension to fall *only* so far as the carbon dioxide tension increases. The shunt caused by uneven distribution is therefore negligible on 100 per cent oxygen; it is most significant at alveolar oxygen tensions of 70 to 90 mm. of mercury.⁹

By contrast, if some alveoli are atelectatic, and not ventilated at all, the degree of oxygenation is immaterial to the degree of shunting. Specifically, a shunt caused by atelectasis will be present also when breathing 100 per cent oxygen, and this is its distinguishing feature.

A further reason for keeping uneven distribution and atelectasis separate is the interesting possibility that etiology and therapy of the two conditions may at times be mutually exclusive, or at least antagonistic. Thus, a pattern of shallow breathing will almost invariably lead to atelectasis, which may be reversed by hyperinflation with deep breaths.⁶ Atelectasis may be prevented by a pattern of large tidal volumes,¹⁰ but it is possible that such a pattern, while preventing atelectasis, could promote uneven distribution, particularly in patients with pre-existing lung disease.

The 27 patients studied by Hamilton and his co-workers were all breathing room air postoperatively. Under these conditions we cannot be certain whether the unexpectedly low arterial oxygen tensions were caused by atelectasis, or by uneven distribution of ventilation in relation to perfusion. If improvement follows deep-breathing then atelectasis of short

duration is a likely cause; after longer periods of collapse re-expansion requires progressively higher airway pressures.² In the group studied, the mean arterial oxygen tension was only 77 mm. of mercury before the operation, 70 mm. of mercury afterwards. Clearly, the arterial blood of the average patient is less well oxygenated than previously suspected, both before and following operation.

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References

1. Pasteur, W.: Active lobar collapse of the lung after abdominal operation, *Lancet* 2: 1080, 1910.
2. Laver, M. B., Morgan, J., Bendixen, H. H., and Radford, E. P., Jr.: Lung volume, compliance and arterial oxygen tensions during controlled ventilation, *J. Appl. Physiol.* 19: 725, 1964.
3. Mead, J., and Collier, C.: Relation of volume history of lungs to respiratory mechanics in anesthetized dogs, *J. Appl. Physiol.* 14: 669, 1959.
4. Gordh, T., Linderholm, H., and Norlander, O.: Pulmonary function in relation to anaesthesia and surgery evaluated by analysis of oxygen tension of arterial blood, *Acta Anaesth. Scand.* 2: 15, 1958.
5. Nunn, J. F., and Payne, J. P.: Hypoxaemia after general anaesthesia, *Lancet* 2: 631, 1962.
6. Bendixen, H. H., Hedley-White, J., and Laver, M. B.: Impaired oxygenation in surgical patients during general anesthesia with controlled ventilation. A concept of atelectasis, *New Engl. J. Med.* 269: 991, 1963.
7. Aviado, D. M., deBurgh Daly, M., Lee, C. Y., and Schmidt, C. F.: The contribution of the bronchial circulation to the venous admixture in pulmonary venous blood, *J. Appl. Physiol.* 155: 602, 1961.
8. Riley, R. L., and Courmand, A.: Analysis of factors affecting partial pressures of O₂ and CO₂ in gas and blood of lungs; Theory, *J. Appl. Physiol.* 4: 77, 1951.
9. Fahri, L. E., and Rahn, H.: A theoretical analysis of the alveolo-arterial O₂ difference with special reference to the distribution effect, *J. Appl. Physiol.* 7: 699, 1955.
10. Hedley-Whyte, J., Laver, M. B., and Bendixen, H. H.: Effect of changes in tidal ventilation on physiologic shunting, *Amer. J. Physiol.* 206: 891, 1964.