Lack of Correlation between Rales and Arterial Oxygen Saturation in Patients with Pulmonary Congestion and Edema

By Albert Vitale, M.D., Paul R. Dumke, M.D., and J. H. Comroe, Jr., M.D.

It is generally believed that pulmonary edema impairs the diffusion of oxygen from alveoli to pulmonary capillary blood and produces anoxemia. However, some patients who apparently have widespread pulmonary edema, as judged by the extent of rales heard over the lung fields, still have normal arterial oxygen saturation. Explanations are advanced for the lack of correlation between extent of rales and arterial oxygen saturation.

IT IS generally agreed that pulmonary congestion, per se, does not lead to arterial anoxemia. However, it is widely believed that pulmonary edema is associated with an impairment in the diffusion of oxygen from the alveolar gas into the pulmonary capillaries, and as a consequence leads to anoxemia. During the past five years, we have measured arterial oxygen saturation in 40 patients with congestive heart failure and pulmonary edema. We have been impressed by the poor correlation between the arterial oxygen saturation and severity of the pulmonary edema as judged by the extent of rales heard over the lungs. We wish to describe these studies briefly and suggest several possible explanations for the lack of correlation.

Since there is at present no accepted objective and quantitative method for measuring pulmonary capillary congestion or pulmonary edema in man, the patients were selected on the basis of their physical findings and medical record. All had heart disease, dyspnea at rest or on exertion and rales easily audible at least over the lung bases. In so far as was possible on the basis of clinical judgment, patients with pulmonary disease were excluded. In more than half of the cases, chest x-ray films were made within five hours of the study, and all patients with radiologic evidence of pleural effusion or pulmonary disease were excluded. No pulmonary function studies were performed; since many of the patients included in this study were in the fifth to eighth decades, it is likely that pulmonary function was slightly impaired in some instances.1

The study consisted simply of a careful auscultation of the chest for rales, followed by the withdrawal of arterial blood for measurement of oxygen saturation2 while the patient was breathing room air. Rales were graded as follows: Bilateral basal = 1; bilateral, over lower one half of chest = 2; bilateral, over three fourths of chest = 3; and bilateral, throughout all lung fields = 4. All patients with grade 4 rales had acute pulmonary edema.

Results and Discussion

Figure 1 shows the data obtained. It is not surprising that some of the patients with only grade 1 or grade 2 rales had low arterial oxygen saturations because (a) some of these patients may have had pulmonary disease in addition to congestive heart failure, and (b) some may have had interstitial pulmonary edema which could impair the diffusion of oxygen without leading to the production of rales. However, it is surprising that 7 of the 12 patients with acute pulmonary edema and with bubbling rales

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From the Departments of Physiology and Pharmacology, and Anesthesiology, Graduate School of Medicine, University of Pennsylvania, Philadelphia, Pa.

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audible over all lung fields had arterial oxygen saturation greater than 93 per cent.\(^*\)

Two explanations may be offered: (1) A slight to moderate impairment of diffusion or a reduction in the diffusing capacity of the lungs does not necessarily lead to a reduction in arterial oxygen saturation in the resting patient. Figure 2 is a diagrammatic representation of a possible explanation for this. Ordinarily, with a normal alveolar-capillary membrane, mixed venous blood comes into almost complete equilibrium with the alveolar oxygen tension about one fourth the way along the course of the pulmonary capillary (curve 1) so that full oxygenation is achieved by a low mean oxygen gradient between the alveolar gas and capillary blood. With progressive impairment of diffusion (curves 2, 3, and 4), full oxygenation is delayed until, in curve 4, it is just barely achieved at the very end of the pulmonary capillary. With more severe impairment of diffusion, even the end-pulmonary capillary blood will not become fully saturated (curve 5) and anoxemia occurs even with the patient at rest in bed. One of the important reasons for making the more complex measurement of the diffusing capacity of the lungs is to detect those cases which have impaired diffusion but still have normal arterial oxygen saturation.

(2) Pulmonary edema is not necessarily a diffuse process impairing diffusion in all alveoli. It is quite possible that a major problem in pulmonary edema is that of airway obstruction produced by distribution through the airways of edema fluid formed in one or two regions of the lung. If this be true, blood flow through the obstructed or poorly ventilated areas must be rerouted in some cases to the nonobstructed, well-ventilated areas of the lungs so that edema seems to originate in many areas of the lungs simultaneously without the occurrence of severe anoxemia.

**SUMMARY**

Arterial oxygen saturation was measured in 40 patients with heart disease, all of whom had rales of varying extent. There was little correlation between the arterial oxygen saturation and the diffuseness of rales. In seven patients with acute and apparently widespread pulmonary edema, arterial oxygen saturation was greater than 93 per cent. Several explanations are suggested.

**SUMARIO ESPAÑOL**

La saturación arterial de oxígeno se determinó en 40 pacientes con enfermedad cardiaca, todos los cuales tenían estertores en variable extensión. Hubo poca correlación entre la
saturación arterial de oxígeno y la difusión de los estertores. En siete pacientes con edema pulmonar aguda y aparentemente difusa, la saturación arterial de oxígeno fue mayor de 93 por ciento. Algunas explicaciones se sugieren.

REFERENCES

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