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To the Editor:—The importance of the factors responsible for distribution of pulmonary blood flow in the determination of arterial oxygenation have long been recognized, and their relevance in the management of patients with respiratory failure is now becoming clear. It is important to recognize that in both normal[1] and abnormal[2,3] lung, pulmonary vascular resistance and cardiac index vary inversely. A positive correlation between cardiac index and Q_s/Q_t has also been reported.[4,5] These correlations do not reveal a cause-and-effect relationship, but they do suggest the difficulty of elucidating problems involving these variables.

In our previous article, to which Lemaire *et al.* refer,[5] Q_s/Q_t increased and PVR decreased with oxygen administration. The statistical test applied suggested that this might be anticipated in 99.5 per cent of similar patients.

The data provided in the letter from Lemaire *et al.* are difficult to interpret. In the presentation of data such as these, there is some question as to whether the probability of changes within the individual can be inferred from population sample data, *i.e.*, given the fact that, in a given population (as in figure 1 in Lemaire *et al.*'s letter), those with the greatest Q_s/Q_t's also had the least pulmonary vascular resistance, does this necessarily indicate that changes in both variables (induced by increased inspired oxygen concentration) would then be in the opposite direction? Although we believe this is frequently the case, the data in figure 1 neither confirm or deny this, nor do they confirm a cause-and-effect relationship between PVR and Q_s/Q_t. That is, a situation in which dilatation of vessels in nonventilated parts of the lung results in an increase in Q_s/Q_t. Lemaire *et al.*'s data suggest that, in patients such as those studied, those with the largest shunt fractions would have the least pulmonary vascular resistance while breathing oxygen. However, since 44 measurements were made in the course of therapy in 13 patients, and we have no further description of the distribution of these measurements, it is not clear that the data are not heavily weighted by one or two patients in whom a large number of measurements were made.

Zapol *et al.*[3] have shown that in acute respiratory failure (*i.e.*, a situation associated with a large Q_s/Q_t), pulmonary vascular resistance may be unusually great. At first sight, this may be in conflict with the data in figure 1, but it is possible that two different patient populations are involved. In one (Zapol's), the pulmonary vasculature is grossly compromised and a severely limited vascular bed in the few remaining ventilated areas may be overdistended. All recruitable vessels in nonventilated areas may also be overdistended and pulmonary arterial pressure is grossly increased. This vascular overdistention possibility is supported by the failure of partial right-heart bypass to decrease pulmonary arterial hypertension. In Lemaire's less seriously ill (?) patients, the pulmonary vessels in the nonventilated portions of lung may not have been fully distended. Under such circumstances, recruitment of these vessels by any mechanism would decrease PVR and increase Q_s/Q_t, giving the correlation presented.

Despite our questioning the relevance of Lemaire *et al.*'s pulmonary vascular resistance/(Q_s/Q_t) correlations, in the context of our previously published study, we are in accord with their closing paragraph.

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