Changes in Intrapulmonary Shunting with Alterations in Pulmonary Vascular Resistance

To the Editor: — In a recent article, Suter et al. attributed the changes in intrapulmonary shunt fraction Qs/Qt induced by ventilation with oxygen in part to variations in pulmonary vascular resistance (PVR).¹

We wondered whether changes in PVR might explain variations in Qs/Qt observed during the treatment of acute respiratory distress syndrome in adults (ARDS). To answer this question, we compared values obtained for Qs/Qt (measured during breathing of oxygen) with values for PVR obtained simultaneously in 13 cases of ARDS. The shunting in these patients prior to study ranged from 30 to 40 per cent as a result of viral pneumonia (4); drug overdose (2); fresh-water drowning (1); multiple trauma (1); fat emboli (1); other causes (4). Pulmonary vascular pressures and cardiac outputs were measured using a thermistor 7-F Swan-Ganz catheter. In all, 44 measurements were obtained during the first 24 hours of treatment, which included volume expansion, dopamine and isoproterenol infusions, and/or membrane lung oxygenation via venoarterial bypass. A close inverse relationship was evidenced between Qs/Qt and PVR (n = 33; r = −0.74; P < 0.001) (fig. 1). When we considered separately the relationships between Qs/Qt and cardiac index, pulmonary wedge and arterial pressures, it appeared that Qs/Qt varied linearly with cardiac index (n = 15; r = 0.73; P < 0.001), and with arterial pressure only when the PVR value was low (n = 15; r = 0.66; P < 0.01). The relationship with the wedge pressure is more complex. Although it does exist in some individuals, it was not found during the following conditions: when the pulmonary wedge pressure was greater than 15 torr (maximal vascular recruitment, as suggested by Suter²); when drugs that modified the relationship of cardiac index and pulmonary wedge pressure (isoproterenol) were used; when venoarterial bypass was used.² Rapid changes in Qs/Qt during varying PVR, their reversibility, and the absence of concomitant chest roentgenographic changes all argue against an anatomic modification, e.g., augmentation in pulmonary extravascular fluid.³ The effect is more likely to be due to a blood flow redistribution or recruitment phenomenon.⁴

In conclusion, when assessing treatment of ARDS, Qs/Qt is an indication not only of the anatomic status of the lung, but also of the extent of recruitment of pulmonary capillaries. For instance, a decreased Qs/Qt, which might be attributed to a healing process subsequent to treatment, may in fact be due solely to shifts of blood flow out of the damaged areas when PVR increases.

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References


(Accepted for publication May 3, 1977.)