Title: WHY DOES PULMONARY VENOUS ADMIXTURE INCREASE WITH CARDIAC OUTPUT?

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Introduction: Changes in cardiac output (Q_t) and venous admixture (VA) are directly related (1). For healthy lungs the relationship may be approximated by (VA New/VA Old) = 0.16 + 0.84 (Q_t New/ Q_t Old). The purpose of this communication is to suggest the physiological basis for this phenomenon and to explore some of its implications.

Venous admixture and hypoxic pulmonary vasoconstriction (HPV): In pulmonary pathophysiology, Q_s/Q_t% results from blood perfusing atelectatic or poorly ventilated lungs and the severity of the gas exchange impairment is dependent on HPV. When regional oxygen tensions vary, HPV results in precapillary constriction so that blood flow is diverted to regions of greatest oxygen tension and arterial hypoxemia is reduced. The stimulus (2) for HPV is hypoxia (P_{SO2}) and both alveolar (P_{AO2}) and mixed venous ($P_{\bar{v}O2}$) oxygen tension contribute so that EQ1:

$$P_{SO2} = P_{\bar{v}O2}^{0.375} \times P_{AO2}^{0.626}$$

In dogs, and probably in man also, the sigmoid stimulus/response curve can be expressed as EQ2:

$$R\%MAX = [100(P_{SO2})^{-6.32}/((1 \times 10^{-11}) + P_{SO2}^{-6.32})]$$

where R%MAX is the response, i.e., pressure or flow change, expressed as a percentage of the maximum response.

Pressure/flow relations for normoxic and hypoxic lung: Earlier work has shown that the magnitude of type of response to HPV varies not only with P_{SO2} but also with the amount of lung affected (3). The HPV response for global lung hypoxia is measured only as a perfusion pressure change; for a very small hypoxic segment only a reduction of blood flow to that region will be observed while for other segment sizes a mixture of both responses will be observed. The basis for this effect is the pressure flow curve and these curves have been derived and extended for a range of cardiac outputs. For ease of comparison, perfusion pressure during normoxia (PP_N) and hypoxia (PP_H) and flow to the normoxic (Q_N) or hypoxic (Q_H) lung segments are expressed as relative values where normal perfusion pressure and flow are 1.0. The normoxic flow/pressure curve is given by EQ3:

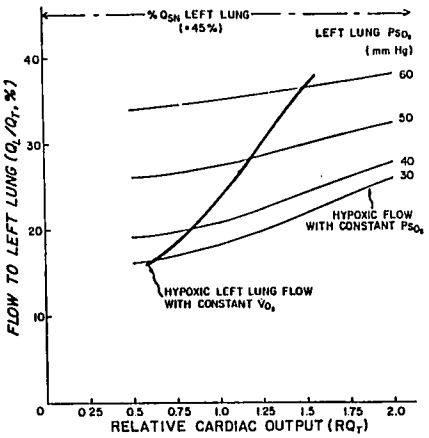
$$Q_N = 0.6\ (PP_N) + 0.4\ (PP_N)^2$$

and the hypoxic flow/pressure curve during maximal hypoxic stimulation is EQ4:

$$Q_H = 0.22\ (PP_H) - 0.14\ (PP_H)^2 + 0.15\ (PP_H)^3$$

when total flow is greater than 1.6 the lines are drawn as parallel. From these four equations, the blood flows to the normoxic and hypoxic lung regions can be derived for any combination of segment size, cardiac output, and alveolar and mixed venous hypoxic stimulus.

These relationships are shown in the figure assuming that the entire left lung is subjected to hypoxia while the right lung receives 100% oxygen and the Q_t is varied from 0.5 to 2.0 times normal. If the hypoxic stimulus is maintained constant when cardiac output is varied then the change of percent flow to the left lung (venous admixture%) is shown by the narrow lines. The venous admixture increases as cardiac output increases but the increase is greatest when the stimulus is greatest (smallest P_{SO2}). The thicker line shows what occurs if oxygen consumption remains constant so that $P_{\bar{v}O2}$, and hence P_{SO2}, increases with Q_t. In these circumstances, changes in percent left lung flow are exaggerated by the reduced stimulation of HPV.



Conclusion: The direct relationship between venous admixture and cardiac output is based on two principal determinants of HPV. The first is the difference in the curvature of the pressure/flow curves for hypoxic and normoxic lung and the second is the importance of $P_{\bar{v}O2}$ as a part of the stimulus for HPV. These relationships appear to underly much that has appeared contradictory for pulmonary abnormalities associated with anesthesia and other pathophysiologic states accompanied by changes in cardiac output.

References:

1) Cheney FW. Anesthesiology 52:496-503, 1980.
2) Marshall C, Marshall BE: J. Appl. Physiol. 55:711-716, 1983.
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