Calculation of Resistance to Pulmonary Perfusion

In their interesting study (1) the authors report that nebulized iloprost was more potent than inhaled nitric oxide (NO) as a pulmonary vasodilator in patients with primary pulmonary hypertension.

They conclude that iloprost caused a more intense decrease in pulmonary vascular resistance (PVR) and a higher increase in cardiac output (CO) than inhaled NO. However, from a hemodynamic point of view, several findings reported in their study remain to be explained. In Table 1 data for the changes in systemic vascular resistance (SVR) and PVR in response to NO and iloprost are listed. Not only PVR, but also SVR, significantly decreased after iloprost.

As presented in the methods section of their paper, the calculation of PVR and SVR was done according to the following formulas that are widely used (2) to calculate the resistance to pulmonary and systemic perfusion:

\[
PVR = \frac{(PAP_{\text{mean}} - PCWP) \times 80}{CO}
\]

\[
SVR = \frac{(SAP_{\text{mean}} - RAP) \times 80}{CO}
\]

As can be seen easily, any increase in cardiac output will lead to a calculatory decrease of PVR and SVR, merely as a result of the way these parameters are calculated (CO in the denominator). Interestingly, the authors report a significant increase in CO after iloprost nebulization (Fig. 1B, Table 1).

Any increase in CO that may result from systemic arterial vasodilation will result in a decrease of calculated values for PVR and SVR without any change of the tone of the pulmonary vasculature itself. Consistent with this interpretation, the authors clearly state that both systemic and pulmonary arterial mean pressures decreased significantly after iloprost compared with NO (Table 1). It would be interesting to see to what extent the ratios of pulmonary/systemic blood pressure and the ratios of calculated PVR/SVR changed after NO or iloprost. Only by reporting data for the ratios of Pp/Ps and PVR/SVR specific changes in pulmonary hemodynamics after exposition to a given drug can be ascertained to be a real effect rather than representing merely a calculatory phenomenon.

Matthias Gorenflo, MD
Department of Pediatric Cardiology
University Children’s Hospital
INF 153
D-69120 Heidelberg, Germany

REFERENCES

REPLY
We appreciate Dr. Gorenflo’s interest in our manuscript. We do not, however, share his concerns regarding the interpretation of our results. In our study in patients with primary pulmonary hypertension, inhaled nitric oxide caused a fall in the pulmonary artery pressure by a mean of 7% from baseline, while the systemic arterial pressure remained stable, clearly indicating selective pulmonary vasodilation. Aerosolized iloprost resulted in a fall of the pulmonary artery pressure by a mean of 13%, whereas the systemic artery pressure declined by 3%. The mean decline in pulmonary vascular resistance with aerosolized iloprost was 33%, and the mean decline in systemic vascular resistance was 21%, also suggesting real pulmonary vasodilation. As we stated in our manuscript, aerosolized iloprost exerted preferential, but not selective, pulmonary vasodilatory effects in patients with pulmonary hypertension.

Whenever an increase in cardiac output is accompanied by stable or decreased pulmonary artery pressures, there must be some degree of pulmonary vasodilation (or recruitment of pulmonary vessels). Back in time, when we used to test oral calcium channel blockers in almost every patient with primary pulmonary hypertension (1), we observed several patients in whom the systemic arterial pressure decreased, the cardiac output increased and the pulmonary artery pressure also increased. In these patients, the pulmonary vascular resistance remained constant. This is what happens when there is systemic vasodilation accompanied by reflexory increase in cardiac output but lack of pulmonary vasodilation.

Marius M. Hoeper, MD
Department of Pulmonary Medicine
Hannover Medical School
30623 Hannover, Germany

REFERENCE