

group. The group with the lowest CO had the highest PVR despite a normal (PADP-PAo) gradient. In this situation assessing impedance based on PVR calculations overemphasizes the importance of CO.

In contrast to calculated PVR the (PADP-PAo) gradient may better reflect drug-produced (*e.g.*, nitrous oxide) pulmonary vasoconstriction since this gradient is less dependent on CO and PAo in several disease states.^{3,4} The use of this gradient in measuring the response of nitrous oxide on PVR (impedance) has not been studied. The data by Drs. Schulte-Sasse, Hess, and Tarnow can not be used since PADP values were not reported. However, use of this pressure gradient in assessing pulmonary vascular responses to nitrous oxide inhalation was tested (using Student's *t* test for paired data) using the data reported by this author in a previous study.⁵ Compared with awake measurements the inhalation of 50% nitrous oxide in oxygen in patients with mitral stenosis and pulmonary hypertension resulted in a significant ($P < 0.05$) increase (5 mmHg) in the (PADP-PAo) gradient. Furthermore, changes in the (PADP-PAo) gradient were not related to CO ($r = 0.19$) or PAo ($r = 0.3$) which suggests that in contrast to calculated PVR, this gradient is less likely to be influenced by passive factors such as flow and left heart filling pressure. Although a significant ($P < 0.05$) direct correlation ($r = 0.66$) existed between changes in calculated PVR and the (PADP-PAo) gradient following inhalation of nitrous oxide, calculated PVR overestimated and underestimated resistance to flow at the end of cardiac diastole as determined by the (PADP-PAo) gradient in some patients (fig. 1).

In conclusion, I feel that changes in resistance in the pulmonary circulation resulting from pharmacologic agents such as nitrous oxide are best expressed by the (PADP-PAo) gradient and not calculated PVR since this pressure gradient is less dependent on passive factors which influence the calculation of PVR.

JOHN C. HILGENBERG, M.D.
Assistant Professor
Department of Anesthesia
Indiana University School of Medicine
Indianapolis, Indiana 46223

NITROUS OXIDE INHALATION

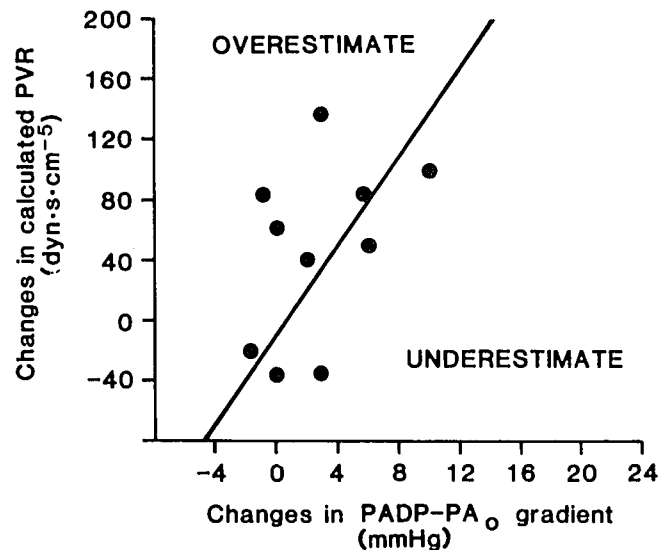


FIG. 1. Comparison between changes in calculated PVR and PADP-PAo gradient following nitrous oxide inhalation (50%) in 11 patients with mitral stenosis and pulmonary hypertension. Solid line is line of best fit. Data modified from Hilgenberg JC *et al.*⁵

REFERENCES

1. Schulte-Sasse U, Hess W, Tarnow J: Pulmonary vascular responses to nitrous oxide in patients with normal and high pulmonary vascular resistance. *ANESTHESIOLOGY* 57:9-13, 1982
2. Harvey RM, Enson Y: Pulmonary vascular resistance. *Adv Intern Med* 15:73-93, 1969
3. Enson Y, Schmidt DH, Ferrer MI: The effect of acutely induced hypervolemia on resistance to pulmonary blood flow and pulmonary artery compliance in patients with chronic obstructive lung disease. *Am J Med* 57:395-401, 1974
4. Sibbald WJ, Paterson NAM, Holliday RL, Anderson RA, Lobb Tr, Duff JH: Pulmonary hypertension in sepsis. *Chest* 73:583-591, 1978
5. Hilgenberg JC, McCammon RL, Stoelting RK: Pulmonary and systemic vascular responses to nitrous oxide in patients with mitral stenosis and pulmonary hypertension. *Anesth Analg (Cleve)* 59:323-326, 1980

(Accepted for publication November 1, 1982.)

In reply:—Dr. Hilgenberg's arguments are based on the data of Harvey and Enson¹ which imply that a high calculated PVR overestimates the impedance to flow in the presence of a small (PADP-PAo) gradient and a low

cardiac output. The Harvey and Enson paper is interesting but does not really find solutions to the problem. Our comment on their final paragraph is that their major premise is mutually exclusive in that they state:

"Resistance to flow in the pulmonary circulation is best identified, at the present time, by the diastolic pressure gradient." However, resistance in any hydraulic system is no more than the ratio of pressure to flow, and yet Harvey and Enson are advocating measuring a pressure gradient at a time of negligible blood flow through the system.

Because of the distensibility of the pulmonary vascular system and the sustained pulsatility throughout the system, it appears necessary to use pulsatile pressure/flow relationships to define the effects of drugs and other perturbations (such as hypoxia and lung inflation), since PVR has been shown to underestimate the true impedance to right ventricular ejection.²⁻⁴ The concept of pulmonary vascular impedance which describes the ratio of oscillatory pressure to oscillatory flow is a logical expression of the characteristics of the pulmonary vascular bed. The development of electromagnetic flowmeters and pressure transducers of high fidelity has enabled physiologists to measure instantaneous blood flow and pressure simultaneously at many sites in the circulation. Such measurements allow computations of true vascular impedance and have been extensively applied to the pulmonary circulation.^{2,5,6}

Even though flow measurements on a beat-to-beat basis are difficult to obtain in the clinical situation, systematic studies of the pulmonary circulation in terms of impedance are needed to aid our understanding of the effect of physiologic and pharmacologic interferences with pulmonary blood flow.

Anesthesiology
58:486, 1983

Advantages of Standing Bellows Ventilators and Low-flow Techniques

To the Editor:—One advantage of low-flow techniques has not received much attention. Small to moderate size leaks are detected earlier than with high-flow systems. This is especially true when standing bellows ventilators are used, rather than hanging bellows ventilators.

In our area, disconnections are an everyday occurrence. Problems have occurred with the fresh gas supply, entrainment of air through vaporizers, and with oxygen analyzers. Some of these accidents have been fatal. Many would have been detected earlier if a standing bellows ventilator had been used.

A large disconnect or cessation of fresh gas flow becomes obvious very quickly by the collapse of the bellows. This often happens before the disconnect alarm sounds. In addition, when used with lower flows, the standing bellows becomes a sensitive leak detector. For example, with a respiratory rate of 10/min and a fresh gas flow of 2 l/min (common values in our hospital), any leak of 200 ml per breath or more will become

apparent. In fact, any time the volume of gas lost from the system is greater than the fresh gas flow, the bellows will collapse. When placed at eye level, this type of bellows becomes a valuable monitor of respiratory rate, volume, and most importantly, the physical integrity of the system.

I believe that the standing bellows ventilator is an inherently safer design than the hanging bellows. It is an important advance in anesthetic technology. Its use, especially with moderate gas flows, should be encouraged.

DOUGLAS H. GRAHAM M.D., F.R.C.P.(C)
*Department of Anaesthesia
St. Paul's Hospital
1081 Burrard Street
Vancouver, B.C.
Canada*

(Accepted for publication November 2, 1982.)

UWE SCHULTE-SASSE, M.D.

Instructor in Anesthesiology

WOLFGANG HESS, M.D.

Assistant Professor of Anesthesiology

JOERG TARNOW, M.D.

Associate Professor of Anesthesiology

Department of Anesthesiology

Charlottenburg Clinic

Free University of Berlin

Spandauer Damm 130

1000 Berlin 19

Germany

REFERENCES

1. Harvey RM, Enson Y: Pulmonary vascular resistance. *Adv Intern Med* 15:73-93, 1969
2. Bergel DH, Milnor WR: Pulmonary vascular impedance in the dog. *Circ Res* 16:401-415, 1965
3. Reuben SR, Swadlow JP, Gersh BJ, Lee G de J: Impedance and transmission properties of the pulmonary arterial system. *Cardiovasc Res* 5:1-9, 1971
4. Foëx P: Pulmonary haemodynamics, The circulation in Anaesthesia. Edited by Prys-Roberts C. Oxford, Blackwell, 1980, pp 253-263
5. Bergel DH, Caro CG, McDonald DA: The input impedance of the pulmonary vascular bed. *J Physiol* 154:18P-19P, 1960
6. Caro CG, McDonald DA: The relation of pulsatile pressure and flow in the pulmonary vascular bed. *J Physiol* 157:426-453, 1961

(Accepted for publication November 1, 1982.)