quality and quantity of anaesthesia care. The goal is to recruit volunteers to serve, one at a time, as teachers (not as providers) of anaesthesia at an already established overseas anaesthesia teaching program and to recruit enough volunteers so as to provide continuous teaching assistance, 12 months of the year for four years, all in the same department.

The overseas program with which OTP will be associated for four years starting in 1991 is the anaesthesia program at the University of Zambia's teaching Hospital in Lusaka. A similar OTP affiliation with another overseas anaesthesia program will be initiated elsewhere starting in 1992 and continuing for three years.

Enquiries about the program may be addressed to the undersigned.

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REFERENCE

- 1 Greene NM. An A.S.A. Overseas Teaching Program. *Anesthesiology* 1990; 72: 1-2.

Pulmonary oedema associated with airway obstruction

To the Editor:

Dr. Lang *et al.* have described the pathogenesis of pulmonary oedema associated with airway obstruction as multifactorial and ascribe a dominant role to the negative intrapleural pressure developed during obstruction.¹ This is debatable.

The Pi-max is the maximum inspiratory pressure measured at the mouth after complete expiration to residual volume and sustained for a one second period.² In a healthy male of less than 50 yr it is 127 cm of water or nearly 10 mmHg³ and this will be the intrapleural pressure achieved, at least initially, following obstruction of the trachea.

Unpressurized aircraft take passengers up to 12,000 feet where the barometric pressures and hence the airway pressures during most of the respiratory cycle would be in the range of 483 mmHg.⁴ These airway pressures are much lower than a healthy subject can achieve during Mueller's manoeuvre during obstruction. Despite this, passengers in such aircraft do not develop pulmonary oedema. This is because at high altitudes the subject hyperventilates to compensate for the low oxygen content

of air and hence tries to maintain a near normal alveolar oxygen content.⁴ Obviously hyperventilation cannot maintain the airway pressure above the altitude barometric pressures (483 mm). Hence it can be surmised that if pulmonary oedema does not develop it is due to the alveolar oxygen content being maintained above a particular threshold and alveolar pressures even at that value do not precipitate oedema. Hypoxia could have been the cause of pulmonary oedema in cases with airway obstruction described in the article.

Thus, probably, hypoxia plays a major role in the loss of capillary integrity and pulmonary edema in airway obstruction rather than the negative intrapleural pressures.

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- 3 Braun NMT, Arora NS, Rochester DF. Respiratory muscle and pulmonary function in polymyositis and other proximal myopathies. *Thorax* 1983; 38: 616-23.
- 4 *Respiratory aspects at high altitude*. In: Nunn JF (Ed.). *Applied Respiratory Physiology*, 3rd ed., London: Butterworth, 1987; 310-20.

REPLY

I have read Dr. Mahashur's critique and am puzzled by his reference to unpressurized aircraft. He suggests that much larger negative pressures can be developed in the lung parenchyma because the subject is at high altitude. However, he is overlooking the fact that all pressures are referenced to atmospheric pressure and it is not the absolute pressure but the transmural pressure which determines airway patency and fluid shifts across vessels.

We concluded that negative pressure pulmonary oedema was a real entity and the development of markedly negative intrapleural pressures was a prerequisite for its development. However, it cannot be concluded from the literature that markedly negative intrapleural pressures cause pulmonary oedema solely by creating negative pulmonary interstitial pressures. Negative interstitial pressures would help facilitate the development of pulmonary oedema simply by "sucking fluids" into the interstitial space as predicted by the Starling