Correspondence

Surgery for mediastinal masses

To the Editor:

The excellent review of mediastinal masses by Dr. Pullerits and Dr. Holzman¹ raises several points.

Perioperative airway obstruction has been reported in 22 patients with mediastinal masses undergoing general anaesthesia.^{2,3} However, in only one case could death in the perioperative period be related to upper airway obstruction,² though the details of that particular case are insufficient for a firm conclusion to be drawn. Airway obstruction, of course, should always be considered life-threatening, yet, with the possible exception of this one case, the airway obstruction was treated successfully. Control of the airway was generally achieved by means of a "long" (i.e., uncut) tracheal tube or a rigid ventilating bronchoscope. In these 22 patients, the airway became obstructed with the initiation of positive-pressure ventilation (after paralysis) in ten and during spontaneous breathing in the other 12. In nine of the 22 cases the obstruction occurred late, most often after extubation. Since six of the case reports showed that the obstruction was primarily expiratory (i.e., dynamic airway obstruction), tracheomalacia may have been a factor. The situation is different when tumour involves intrathoracic cardiovascular structures. Five such cases have been reported, death occurring in three of them. Autopsy revealed tumour involvement of a main pulmonary artery in all, the pericardium in two and the pulmonary veins in one.

Therefore, perioperative airway obstruction is by no means necessarily fatal in patients with mediastinal masses. It can be treated safely and efficaciously if recognized and if, as pointed out by Dr. Pullerits and Dr. Holzman, the anaesthetist is prepared. However, in contrast, because of the high mortality associated with the administration of an anaesthetic to a patient with tumour involvement of intrathoracic vascular structures, we must learn to identify these patients better preoperatively. Perhaps by using CAT scanning, NMR imaging, and echocardiography (2-D and Doppler) in some combination, we can accomplish this important task. Furthermore, if cardiovascular collapse does occur in the perioperative period, and the chest is not open, thoracotomy must be considered "immediately."

Finally, less than half the incidents of airway obstruction occurred during mechanical ventilation. Although definite benefits accrue from maintenance of spontaneous

ventilation,⁴ it seems that it does not diminish the risk of airway obstruction in this population. It does, however, emphasize the need to know whether any airway obstruction that exists is fixed or variable.³

Scott A. Lang MD FRCPC
David A.E. Shephard MB FRCPC
Department of Anaesthesia
University Hospital
Saskatoon, Saskatchewan
S7N 0X0

REFERENCES

- 1 Pullerits J, Holzman R. Anaesthesia for patients with mediastinal masses. *Can J Anaesth* 1989; 36: 681-8.
- 2 Meeker WR, Richardson JD, West WO, Parker JC. Critical evaluation of laparotomy and splenectomy in Hodgkin's disease. *Arch Surg* 1972; 105: 222-9.
- 3 Prakash UBS, Abel MD, Hubmayr RD. Mediastinal mass and tracheal obstruction during general anesthesia. *Mayo Clin Proc* 1988; 63: 1004-11.
- 4 Sibert KS, Biondi JW, Hirsch NP. Spontaneous respiration during thoracotomy in a patient with a mediastinal mass. *Anesth Analg* 1987; 66: 904-7.

Precordial Doppler diagnosis of haemodynamically compromising air embolism during Caesarean section

To the Editor:

I would like to congratulate Drs. Fong, Gadalla and Gumbel for their timely and interesting case report published in the March 1990 issue of the Journal.¹

Air embolism undoubtedly occurs in patients undergoing Caesarean section,² though the incidence is not documented in the literature. Its true incidence in the obstetrical population cannot be determined if we insist on attributing every event that sets off a precordial Doppler to intravascular air. These "Doppler" events should be called "embolic events"³ without further definition, unless additional evidence exists that it is air (i.e., an increase in end-tidal N₂ concentration, aspiration of air