2.4 mmHg in normal newborn infants, 3.5 mmHg in preterm infants recovering from respiratory distress syndrome (RDS) and 9 mmHg in preterm infants with bronchopulmonary dysplasia.[2] The correlation between $PaCO_2$ and $P_{ET}CO_2$ was good in infants with healthy lungs,[2] though the correlation was not good in infants who are critically ill.[3,4] McCann *et al.*, however, used successfully (a-ET) PCO_2 difference, where $P_{ET}CO_2$ was sampled from nasal cavity, as a trend monitor to evaluate the improvement of pulmonary functions following therapy in critically ill infants with RDS.[3]

Second, Campbell *et al.* confirmed that negative (a-ET) PCO_2 gradients occurred in children and may not be due to experimental errors. The incidence of negative (a-ET) PCO_2 gradients is 12% in healthy adults,[5] whereas the incidence may be higher (50%) in pregnant patients,[6,7] and children.[1,8] Negative (a-ET) PCO_2 values have also been observed during exercise.[9] Although late emptying of well-perfused alveoli with higher CO_2 tensions contribute to the occurrence of negative (a-ET) PCO_2 gradients, there are other important factors which may result in the negative (a-ET) PCO_2 values in infants and children. These include reduced functional residual capacity (FRC), increased CO_2 production and better overall ventilation/perfusion ($\dot{V}/\dot{Q}$) matching. These factors also result in negative (a-ET) PCO_2 values in pregnant patients.[6,7] Fluctuations in $PACO_2$ (alveolar CO_2) during the respiratory cycle determine the absolute values of $P_{ET}CO_2$, with the $P_{ET}CO_2$ being close to peak $PACO_2$ and the $PaCO_2$ being close to mean $PACO_2$. Fluctuations in $PACO_2$ become exaggerated with increased CO_2 production to reduce FRC. This results in an increase of $PACO_2$ to the level of $PvCO_2$ (mixed venous CO_2), as a large amount of CO_2 is evolved into a lung which becomes smaller as expiration continues. When dead space and $\dot{V}/\dot{Q}$ mismatch are minimized, as in infants and children with normal lungs and circulation, the changes of $P_{ET}CO_2$ exceeding $PaCO_2$ are increased.[6-12] The presence of lung areas with increased time constants (i.e., alveoli with higher $PACO_2$ emptying last) may further increase the chances of negative (a-ET) PCO_2 gradient.

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Co-axial placement of endobronchial blocker

To the Editor:

An 8-14 Fogarty occlusion catheter (Baxter Healthcare Corporation, Santa Ana, CA) has been used to achieve endobronchial blockade for one lung anesthesia,[1] and the technique has been modified to place the Fogarty catheter coaxially using a fibreoptic bronchoscope and two bronchoscopic airway connectors placed in series.[2] However, positioning under direct fibreoptic vision can be difficult. We report a modification of these techniques to place the 8-14 Fogarty catheter coaxially as an endobronchial blocker by selectively intubating a mainstem bronchus, advancing a blocker through the endotracheal tube, and then withdrawing the tube into the trachea.

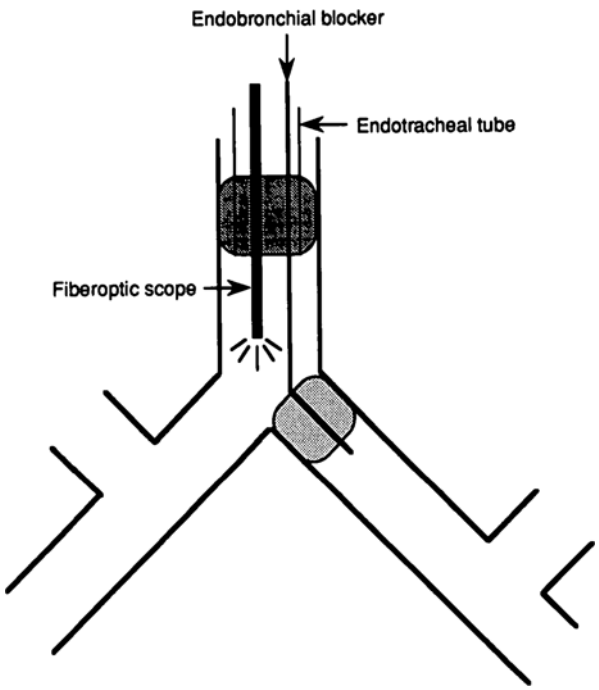

FIGURE The endotracheal tube has been retracted into the trachea and the fibreoptic bronchoscope used to adjust its final position.

A bronchoscope with an O.D. of ≤4.0 mm and an 8 or 8.5 mm endotracheal tube should be used. Two Bodi double swivel airway connectors (Sonotek Medical Inc., Hingham, MA) connected in series are attached to the endotracheal tube to allow passage of the Fogarty catheter and bronchoscope.

The bronchoscope is advanced into either the right or left mainstem bronchus. An endotracheal tube is then advanced over the bronchoscope and positioned into the mainstem bronchus. The bronchoscope is removed, and an 8-14 Fogarty catheter is advanced through the endotracheal tube into the bronchus via the proximal port of the airway connector. The endotracheal tube is withdrawn to mid-trachea while holding the blocker to maintain its position. The endotracheal tube is securely taped in place, and the bronchoscope re-introduced to allow final positioning of the endobronchial blocker (Figure). The Fogarty catheter is inflated under direct vision, and should fill the bronchus without herniating into the trachea. The volume of air required should be noted to avoid over-inflation of the balloon at a later time.

This method of endobronchial blockade has proved timely and effective. The technique is useful in patients requiring one-lung ventilation for oesophageal resection, thorascopy, pulmonary resection, thoracic aortic aneurysm repair, and thoracic spine surgery. However, the blocker should be used with caution in surgical procedures involving the hilum when the risk of displacement from manipulation or surgical rupture of the balloon is high. The blocker position should be verified by auscultation and bronchoscopy following changes in patient position, and muscular paralysis should be maintained to avoid displacement secondary to patient movement.

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