Easier Fiberoptic Intubations

To the Editor—Fiberoptic intubation is a useful adjunct to direct laryngoscopy, particularly in patients with difficult airways. However, intubation attempts may fail because of an inability to pass the endotracheal tube (ET) over the fiberscope into the trachea. Failure occurs more frequently during oral intubation and is related to the tube’s becoming caught on the epiglottis, arypepiglottic folds, or corniculate cartilages. Repositioning the tube is helpful but does not always improve the chances of success. Complete failure to pass the tube occurs at a rate of 1.2%. However, failure can be expected to be much more likely in inexperienced hands.

We have devised an economical and simple method to alleviate the obstruction and facilitate otherwise difficult or impossible intubations.

The reason for difficulty in passing the ET is related to the difference in diameters between the ET and the fiberscope. The larger the tube, the greater the likelihood of difficulty in passing the ET. Minimizing this difference will facilitate intubation. One solution is to thread a smaller ET into the ET being used.

For example, at this institution the Olympus LF-1 Intubating Fiberscope and Mallinckrodt Lo-Pro Tracheal tubes are routinely used. The fiberscope allows an ET as small as 5.0 mm internal diameter to be threaded over it. When a 7.0- or 8.0-mm tube is cut to the recommended oral length of 23 or 25 cm, respectively, it can readily be threaded over a lubricated uncuffed pediatric ET. A 7.0-mm tube fits over a 5.0-mm tube, and an 8.0-mm tube fits over a 5.5-mm tube. The tip of the pediatric tube protrudes beyond the end of the larger tube and fills the space that would otherwise exist between the larger tube and the fiberscope (fig. 1). This minimizes the difference in diameters and allows the tube to be easily threaded into the trachea. The adaptors of both tubes are held together and advanced simultaneously (fig. 2). Once in place, the fiberscope and smaller tube are removed. Lubrication of the outside of the smaller tube with a polyethylene glycol lubricant (Americaine® or Xylocaine ointment) is important to ensure its easy removal.

A series of 30 patients with anticipated difficult direct laryngoscopy were selected for fiberoptic intubation under general anesthesia. Seventeen-millimeter ETs were used in the women (n = 12) and 8.0-mm ETs were used in the men (n = 18). With each patient serving as his or her own control, intubation was initially attempted with the ET alone. Our experience confirms that of others that in a large proportion of patients (n = 27, 89.9%), significant resistance to passage of the tube occurs. In these patients the tube was withdrawn, and, with the fiberscope left in position, the ET tube and pediatric tube were passed together. With the second attempt, the tube passed smoothly in all cases. No cases were encountered that required additional manipulation of the tube or airway.

We currently use this technique routinely during fiberoptic intubation, particularly when the patient is at increased risk of hemoglobin oxygen desaturation during a prolonged intubation attempt. The technique provides us the confidence to know that once the fiberscope is in the trachea, the tube can be advanced smoothly and quickly into position in virtually all cases. In addition, using the pediatric tube reduces the risk of inadvertently passing the fiberscope through the Murphy eye of the ET and complicating the intubation.

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Increased Pulmonary Artery Pressure Related to Sternal Retraction

To the Editor—The pulmonary circulation is a highly distensible, low-pressure, and low-resistance system that can accept substantial increases in blood flow with only small changes in pulmonary artery pressure.1 Pulmonary vascular resistance may increase in response to a variety of stimuli, including hypoxia, hypothermia, hypercarbia, and acidosis.2

A 50-year-old woman was admitted to the hospital because of fatigue, angina, and dyspnea on exertion. Cardiac catheterization revealed a heavily calcified bicuspid 0.3-cm² aortic valve, a mean left ventricular–aortic systolic pressure gradient of 78 mmHg, and a left ventricular ejection fraction of 78%. The patient was scheduled for aortic valve replacement.

In the operating room, an oximetric pulmonary artery catheter, placed through the left internal jugular vein, revealed a mixed-venous hemoglobin oxygen saturation (SVO₂) of 78%, a cardiac index of 2.2 l·min⁻¹·m⁻² and a pulmonary artery pressure of 36/18 mmHg. Anesthesia was induced with intravenous fentanyl 50 μg·kg⁻¹ and midazolam 5 mg. Atraumatic tracheal intubation with a 7.5-mm cuffed tube followed the administration of 10 mg vecuronium bromide. Heparin 20,000 U was administered and cardiopulmonary bypass initiated. The aortic valve was replaced with a 19-mm valve (St. Jude Medical, Inc., St. Paul, MN), with a cross-clamp time of 97 min.

The patient was separated from cardiopulmonary bypass after 2 h while dobutamine 5 μg·kg⁻¹·min⁻¹ was administered. Her blood pressure was 80/40 mmHg and paced heart rate 90 beats per min. Pulmonary artery pressure immediately after bypass was 44/15 mmHg, cardiac index 2.5 l·min⁻¹·m⁻², and SVO₂ 83%. Heparin was antagonized with 200 mg protamine given slowly over 10 min. The activated clotting time was 135 s. Twenty minutes after bypass was terminated, the pulmonary artery pressure gradually increased to equal or exceed the systemic arterial pressure of 80/40 mmHg. SVO₂ was 82–85% (table 1), central venous pressure 10–12 mmHg, pH 7.34, PaO₂ 497 mmHg, and PaCO₂ 43 mmHg at the time of the increase in pulmonary artery pressure. Visualization revealed normal cardiac contraction, no evidence of right ventricular distension, and no pulmonary torsion.3

Transducers were checked and recalibrated, and a needle was inserted in the left pulmonary artery to confirm pulmonary artery pressure measurement of 80/60 mmHg. The chest roentgenogram confirmed the pulmonary catheter to be in the right pulmonary artery. The sternal retractor was loosened and removed, and the pulmonary artery pressure immediately decreased to 44/18 mmHg. The patient recovered without further difficulty and was discharged from the cardiac intensive care unit 2 days later.

Direct measurement of pulmonary artery pressure confirmed pulmonary hypertension and precluded measurement artifact. The possibility of a pulmonary hypertensive reaction to protamine was considered but ruled out because enough time had elapsed since the protamine had been administered. Fortunately, right ventricular failure did not occur, as has been reported following allergic reaction to protamine.4,5 In addition, SVO₂ and cardiac index remained within acceptable ranges (table 1), which we were unable to explain.

We determined that the elevation of pulmonary artery pressure was caused by the sternal retractor, which may have been opened unusually wide while attempting to gain hemostasis. Stretching and compression of the left atrium or of the pulmonary veins may have compromised pulmonary venous return and caused elevation of the pulmonary artery pressure. In addition, a reaction to protamine was unlikely because this drug was given slowly, and the elevation of pressures occurred later. Hence, when other possible causes of unexplained elevation of pulmonary artery pressures—such as hypoxia, hypothermia, hypercarbia, acidosis, light anesthesia, and drug reaction—have been eliminated, then loosening of the sternal retractor should be considered.

Table 1. Results of Hemodynamic Measurements

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<tr>
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<th>Before Bypass</th>
<th>After Bypass</th>
<th>No Retraction</th>
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<tbody>
<tr>
<td></td>
<td>Initial</td>
<td>20 min</td>
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<tr>
<td>Pulmonary artery pressure (mmHg)</td>
<td>34/16</td>
<td>44/15</td>
<td>90/40</td>
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<tr>
<td>Systemic blood pressure (mmHg)</td>
<td>100/60</td>
<td>80/50</td>
<td>80/40</td>
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<tr>
<td>Cardiac index (l·min⁻¹·m⁻²)</td>
<td>2.5</td>
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<td>SVO₂ (%)</td>
<td>85</td>
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<td>Heart rate (beats)</td>
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<tr>
<td>PETCO₂ (mmHg)</td>
<td>30</td>
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