Physiological comparison of spontaneous and positive-pressure ventilation in laryngotracheal stenosis

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Background. We compared spontaneous and positive-pressure ventilation in patients undergoing general anaesthesia for the treatment of extrathoracic, intralumenal laryngotracheal stenosis to assess the best method of ventilation in this patient group.

Methods. Records of 30 patients with laryngotracheal stenosis, but not with a tracheostomy, undergoing lumen-restoring surgery were prospectively reviewed. Awake spirometry and flow-volume loops were recorded before the procedure. Patients received i.v. anaesthesia induction, muscle paralysis, and positive-pressure ventilation through a laryngeal mask airway (LMA). Anaesthetized tidal volume (TV) and flow-volume loop measurements were obtained.

Results. We studied 19 males and 11 females [mean age 47 (SD 19) yr], ASA Grade III or IV, with lesions at 31 (10) mm below the vocal cords. Peak inspiratory flow (PIF) and peak expiratory flow (PEF) rates were 2.0 (1.2) litre s⁻¹ and 3.2 (1.7) litre s⁻¹ when awake. Tidal volumes were 657 (193) ml [9.2 (3.6) ml kg⁻¹] and 586 (158) ml [8.3 (3.1) ml kg⁻¹], respectively, when anaesthetized. There was a significant reduction in the PEF/PIF ratio, from a mean of 2.4 (1.3) awake to 1.0 (0.1) when anaesthetized (P<0.0001). A significant correlation was noted between awake PEF and anaesthetized expiratory TV (r=0.57; P<0.001) but not between awake PIF and anaesthetized inspiratory TV.

Discussion. Positive-pressure ventilation through an LMA is an effective method of ventilating patients with laryngotracheal stenosis. Spontaneous ventilation creates negative inspiratory intratracheal pressure that exacerbates an extrathoracic lesion, whereas positive-pressure ventilation generates positive intratracheal pressure that improves ventilation. This helps explain the apparent resolution of airway obstruction after positive-pressure ventilation.

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Laryngotracheal stenosis is a rare but serious condition with an approximate annual incidence of 1 in 200 000 adults.¹ It causes exertional dyspnoea and without prompt treatment can progress to life-threatening airway compromise (Fig. 1).² The most critical aspect of laryngotracheal reconstruction is securing and maintaining a safe airway throughout surgery, particularly at induction of anaesthesia when the patient moves from an upright to supine position and from wakefulness to anaesthesia.³⁴ These changes reduce the patient’s functional residual capacity, and make the upper airways more collapsible and therefore more prone to obstruction and increase the work of breathing.⁵⁶ Laryngotracheal stenosis can also be encountered in the rare scenario of an acute respiratory emergency with a presumptive lower airway aetiology being, in fact, the result of central airway obstruction, identified through the anaesthetist’s inability to advance a tracheal tube to secure the airway.

A clear understanding of ventilatory changes associated with induction of anaesthesia and a safe approach to management that is based on sound physiological principles is essential in ensuring patient safety. To address this,
we undertook a study to compare spontaneous and positive-pressure ventilation in patients with laryngotraheal stenosis to determine the optimal method of ventilation in this patient group.

**Methods**

Surgical and anaesthetic records of 30 adult patients with laryngotraheal stenosis, but without a tracheostomy, undergoing endoscopic airway reconstruction in a national referral airway unit over a 9 month period in 2006 were prospectively studied. Information about patient demography and lesion characteristics at the time of surgery was recorded. Formal ethical approval for this observational study was sought but was not deemed necessary, given that all of the measurements reported in the present study form part of our standard clinical practice and no additional tests were performed.

All patients underwent spirometry and flow-volume loop examinations in the anaesthetic room before general anaesthesia. None of the patients received premedication. Measurements were done according to the American Thoracic Society/European Respiratory Society guidelines,\(^1\) using a MicroLoop portable spirometer.

Anaesthesia was induced with a bolus of propofol 3 mg kg\(^{-1}\) and fentanyl 1–1.5 \(\mu\)g kg\(^{-1}\) and atracurium 0.5 mg kg\(^{-1}\) for muscle relaxation. Intermittent positive-pressure ventilation was established using a classic laryngeal mask airway (LMA\(^{†}\)) (Intavent, Orthofix, UK), and patients received 100% oxygen during induction of anaesthesia. In the operating theatre, the LMA was replaced with a Dedo-Pilling laryngoscope, which was positioned to visualize the laryngotraheal complex. Ventilation was re-established using high-frequency supraglottic jet ventilation delivered at 100 Hz to allow shared-airway surgery to proceed. Total i.v. anaesthesia was maintained using infusions of propofol and alfentanil. At the end of the operation, the laryngoscope was removed and the LMA was again used to secure a supraglottic airway. Neuromuscular block was antagonized with neostigmine 2.5 mg and glycopyrolate 0.5 mg at the end of the procedure. Train-of-four neuromuscular monitoring was used to ensure adequate reversal of neuromuscular block, and the lungs were ventilated until spontaneous breathing was established.

Measurements were obtained at the beginning of each procedure after muscle paralysis and establishment of supraglottic positive-pressure ventilation before any surgical manipulation had occurred. In all cases the driving inspiratory pressure was 20 cm H\(_2\)O, the ventilation frequency was 10 min\(^{-1}\) and the inspiratory:expiratory (I:E) ratio was 1:1. These ventilatory settings were chosen to minimize auto-PEEP. After steady-state conditions were reached, anaeasthetized flow-volume loops and inspiratory and expiratory tidal volumes (TVs) were recorded using a Datex Ohmeda AS\(_3\) anaesthesia monitor.

**Data analysis**

Awake spirometry and flow-volume loop variables were calculated using Spida 5.0 software. To assess the degree of airway obstruction within individual patients between awake and anaesthetized conditions, the ratio of peak expiratory (PEF) to peak inspiratory flow (PIF) rates was calculated\(^1\) and compared using Student’s \(t\)-test. Correlations between different variables were assessed with Pearson’s method. Backwards multiple regression analysis was used to correlate independent predictors of the different awake spirometry variables. Spirometry variables were then stratified based on independent predictors. Data were analysed and illustrated using SPSS 12.0 for Windows (SPSS Inc., Chicago, IL, USA), and \(P<0.05\) was considered statistically significant.

**Results**

Over the study period, 30 adult patients with laryngotraheal stenosis undergoing endoscopic airway reconstruction were studied. Of the 30 adult patients, 19 were males and 11 were females and the mean age was 47 (19 yr (sd)) (range 17–82) of ASA Grade III or IV. The commonest aetiologies were post-intubation tracheal stenosis, which occurred in 23 (77%) patients, and idiopathic subglottic stenosis (23%). Airway lesions were, on average, located 31 (10) mm below the vocal cords (range 5–50). Twelve

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lesions (40%) caused lumenal obstruction of between 0 and 50% (Myer–Cotton Grade I), while nine lesions encroached on 51–70% of the airway lumen (Myer–Cotton Grade II). A further nine lesions led to lumenal obstruction between 71 and 99% (Myer–Cotton Grade III). Table 1 provides further information about awake spirometry findings.

The mean PEF/PIF ratio measured from awake flow-volume loops was 2.4 (1.3), decreasing to 1.0 (0.1) during anaesthesia (P<0.0001; Student’s t-test) (Fig. 2).

The mean inspiratory and expiratory TVs were 657 (193) (range 267–1153) [9.2 (3.6) ml kg\(^{-1}\), range 3.6–16.7] and 586 (158) (range 219–930) [8.3 (3.1) ml kg\(^{-1}\), range 3.0–14.2], respectively. In all cases, oxygen saturation remained >95% throughout induction of anaesthesia. There was a significant correlation between awake PEF and anaesthetized expiratory TV, but not between awake PIF rate and anaesthetized inspiratory TV (Fig. 3).

### Discussion

This study found that in patients undergoing general anaesthesia for extrathoracic intralumenal laryngotracheal stenosis, i.v. induction of anaesthesia, muscle paralysis, and positive-pressure ventilation through an LMA is an effective method of ventilating patients during anaesthesia, regardless of the severity of the stenosis. The degree of upper airway obstruction, observed and quantified using flow-volume loops, was significantly lessened when anaesthetized flow-volume loops were recorded under conditions of muscle relaxation and positive-pressure ventilation, in comparison with awake spontaneous ventilation. We also observed a significant correlation between anaesthetized expiratory TV and awake PEF, but not between anaesthetized inspiratory TV and awake PIF.

These observations can be explained by the physiological principles that govern airflow through the upper airways during inspiration and expiration and the impact of an extrathoracic intraluminal airway stenosis on ventilatory dynamics. During spontaneous inspiration, there is negative extrathoracic intratracheal pressure that causes in-drawing of the mobile tracheal segments, further narrowing the lumen, and limiting inspiratory airflow. In contrast, during expiration, there is positive intratracheal pressure, which improves airway dimensions and airflow (Fig. 4). Thus, under conditions of spontaneous ventilation, laryngotracheal stenosis selectively impairs inspiratory airflow as detected on flow-volume loop examination. With positive-pressure ventilation, however, there is positive intratracheal pressure during both phases of ventilation, delivered during inspiration by the anaesthetic machine, and during expiration by the elastic recoil of the lungs (Fig. 4). With positive-pressure ventilation, therefore, the mechanics of both inspiration and expiration are favourable in respect of their impact on the stenosis.

These findings have implications for managing patients with laryngotracheal stenosis undergoing general anaesthesia. The optimal method for inducing anaesthesia in these patients is controversial. The traditional view is that spontaneous respiration is maintained and anaesthesia is induced with a volatile inhalation anaesthetic given by facemask. This is based on the assumption that this is safer than i.v. induction and muscle paralysis, because if apnoea occurs the patient will stop inhaling the anaesthetic and will awaken to regain airway control and spontaneous ventilation. In our opinion, this approach is unsafe in patients with intralumenal laryngotracheal stenosis, and

### Table 1 Spirometry findings. All variables were expressed (±) and with [range]. Myer–Cotton stenosis grading system: Grade I, 0–50% lumen stenosis; Grade II, 51–70% lumen stenosis; Grade III, 71–99% lumen stenosis; Grade IV, no lumen; FEV\(_1\), forced expiratory volume in 1 s; FVC, forced vital capacity; MEF\(_{50}\), maximal expiratory flow at mid vital capacity. Variables found to be independently associated with FEV\(_1\), FVC, and MEF\(_{50}\) on backward multiple regression were Myer–Cotton grade of the stenosis and patient age which was dichotomized at 45; PEF, peak expiratory flow. The only variable found on backward multiple regression to be independently associated with PEF was Myer–Cotton grade of the stenosis; MIF\(_{50}\), maximal inspiratory flow at mid vital capacity; PIF, peak inspiratory flow. No variables were found to be independently associated with MIF\(_{50}\) and PIF.

<table>
<thead>
<tr>
<th>Spirometry variables</th>
<th>Age≤45 yr</th>
<th>Age&gt;45 yr</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEV(_1) (litre)</td>
<td>Grade I stenosis</td>
<td>3.28 (0.98) [1.81–4.22]</td>
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<tr>
<td></td>
<td>Grade II stenosis</td>
<td>2.46 (0.14) [2.29–2.55]</td>
</tr>
<tr>
<td></td>
<td>Grade III stenosis</td>
<td>2.03 (0.78) [0.67–2.59]</td>
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<tr>
<td></td>
<td>Grade IV stenosis</td>
<td>4.50 (0.74) [3.50–5.43]</td>
</tr>
<tr>
<td></td>
<td>Grade I stenosis</td>
<td>3.52 (0.57) [2.94–4.07]</td>
</tr>
<tr>
<td></td>
<td>Grade II stenosis</td>
<td>2.31 (1.01) [1.95–4.67]</td>
</tr>
<tr>
<td></td>
<td>Grade III stenosis</td>
<td>4.61 (1.45) [1.58–9.52]</td>
</tr>
<tr>
<td></td>
<td>Grade IV stenosis</td>
<td>2.26 (0.93) [2.02–2.63]</td>
</tr>
<tr>
<td></td>
<td>Grade I stenosis</td>
<td>2.19 (1.06) [0.16–2.88]</td>
</tr>
<tr>
<td>FVC (litre)</td>
<td>All ages</td>
<td>3.80 (1.03) [2.64–5.43]</td>
</tr>
<tr>
<td>PEF (litre s(^{-1}))</td>
<td>Grade I stenosis</td>
<td>3.09 (0.69) [1.69–4.07]</td>
</tr>
<tr>
<td></td>
<td>Grade II stenosis</td>
<td>3.80 (1.03) [2.64–5.43]</td>
</tr>
<tr>
<td></td>
<td>Grade III stenosis</td>
<td>2.83 (1.07) [1.58–4.67]</td>
</tr>
<tr>
<td></td>
<td>All ages</td>
<td>1.9 (1.2) [0.7–5.6]</td>
</tr>
<tr>
<td>PIF (litre s(^{-1}))</td>
<td>All ages</td>
<td>2.0 (1.2) [0.7–5.7]</td>
</tr>
</tbody>
</table>
that muscle paralysis and supraglottic positive-pressure ventilation using an LMA is more effective. The use of an LMA is already recognized as being integral to managing the difficult airway. The Difficult Airway Society guidelines indicate that an LMA should be used as a rescue device after unanticipated failure to intubate the trachea and after failure of mask ventilation.17 Thus, the use of the LMA immediately after induction of anaesthesia in patients with laryngotracheal stenosis who can be considered to be anticipated impossible intubations is both logical and consistent with published guidelines. We have found that lesion consistency (soft fibro-inflammatory lesions or mature fibrotic scars) does not appear to have a bearing on the improvement in ventilation after transition from spontaneous to positive-pressure ventilation.

However, large pedunculated and mobile supraglottic lesions can obstruct the laryngeal inlet. Assessment of size, mobility, and site of lesions is therefore essential before this method is used. In our practice, this involves visualization of the supraglottis and the laryngotracheal complex with flexible naso-endoscopy before induction of anaesthesia in all patients.

In the presence of an extrathoracic airway stenosis, spontaneous ventilation is associated with significant ventilatory impairment, even in awake, upright patients, as a consequence of the effect of the stenosis on ventilatory dynamics (Fig. 4). Spontaneous ventilation during induction of anaesthesia makes use of this same ‘disadvantaged’ ventilatory dynamics, except for an increased collapsibility of the upper airways,5 6 added work of respiration,3 7 and reduced functional residual capacity, making spontaneous ventilation during induction of anaesthesia less reliable and more dangerous.18 In contrast, the use of muscle relaxants followed by positive-pressure ventilation through an LMA lessens the detrimental impact of the stenosis on ventilatory dynamics, achieving adequate ventilation to maintain safe levels of oxygenation. We have used this method extensively in this group of patients and have not encountered major problems related to induction of general anaesthesia.

In conclusion, we have demonstrated that in the presence of upper airway obstruction as a result of an
intraluminal, extrathoracic, laryngotracheal stenosis, placement of a laryngeal mask with muscle paralysis and positive-pressure ventilation is associated with improved ventilatory dynamics compared with spontaneous ventilation. We propose that in patients with known laryngotracheal stenosis undergoing shared-airway reconstructive surgery, or in patients who present as an emergency in whom an extrathoracic laryngotracheal stenosis is unexpectedly diagnosed, LMA with positive-pressure ventilation offers the most physiological method of achieving and maintaining a safe airway during induction of anaesthesia.

Fig 4 Schematic representation of spontaneous (A) and ventilator-driven positive-pressure ventilation (B) in the presence of extrathoracic laryngotracheal stenosis. $R$, resistance; $C$, capacitance; $RM$, respiratory musculature; $VE$, ventilator; $R_{(stenosis)}$, a variable resistor which has a higher resistance (darker colour) with spontaneous ventilation than with positive-pressure ventilation; $R_{(airway)}$, resistance to airflow through the remainder of the tracheobronchial tree; $R_{(lung)}$, resistance of lung tissue which is caused predominantly by resistance to stretching during inspiration. This resistance also stores the potential energy in the pulmonary capacitor $C_{(lung)}$ which is then released during, and in part drives expiration. $R_{(thorax)}$ denotes chest wall’s resistance to movement, and the potential energy stored $C_{(thorax)}$ during inspiration is also a driver of expiration.

References
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