Leakage of fluid past the tracheal tube cuff in a benchtop model

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Summary

We have assessed a range of high volume, low pressure (HVLP) cuffed tracheal tubes in a benchtop model, for leakage of fluid from above the cuff to the model trachea below, during various ventilatory modes. Rapid leakage occurred in the model during all modes of ventilation, unless tracheal pressure was greater than the height of fluid in the column above the cuff. This leakage occurred preferentially down longitudinal folds that occur in the HVLP cuff wall. This model suggests that, if a longitudinal fold within the cuff wall is patent, then the possibility exists of subglottic to tracheal leakage. (Br. J. Anaesth. 1997; 78: 557–562).

Key words


A tracheal tube with a high volume, low pressure (HVLP) cuff does not protect the lower airway from contamination by material leaking from the subglottis and above, in all circumstances, even with cuff pressure maintained at presently acceptable levels (25–30 cm H2O). It has been demonstrated in vivo, in patients whose tracheas were intubated with a tube with an HVLP cuff, undergoing general anaesthesia, that leakage occurred in 100% of cases, and this occurred down longitudinal folds within the cuff wall. These folds always occur in an HVLP cuff on inflation within the trachea, as the diameter of the cuff must be greater than that of the trachea for intra-cuff pressure to be equal to tracheal wall pressure. Subclinical leakage of contaminated secretions around the cuff occurs in patients during mechanical ventilation in the intensive care unit (ICU), causing tracheal colonization and increasing the risk of ventilator-associated pneumonia. The objects of this study were to identify the times during standard ventilatory patterns when the dynamic tracheal pressures below the cuff were such that leakage of fluid to the trachea was likely, and to identify aspects of management which are protective against leakage. We have quantified leakage rates in order to demonstrate that macro- (leakage of the order of ml s⁻¹) rather than micro-aspiration (leakage of the order of ml h⁻¹) can occur along a cuff channel.

Materials and methods

LUNGT/ARCHEA MODEL

The patient model (fig. 1) consisted of a model lung (Vent Aid, TTL, Michigan Instruments Inc) with a Bear 3 ventilator driving the second mechanically linked bellows (model lung). This was linked to the distal artificial trachea to simulate a lung capable of spontaneous breaths. The artificial trachea was a silicone tube of 2.2 cm internal diameter (Laerdal Medical silicone extension tubing part No 87100) held vertically. The mean diameter of the adult male trachea is 2.2 cm, and a size 8 tracheal tube is used commonly for adult male patients in intensive care practice. The pressure at the distal tip of the tracheal tube was measured via a central venous catheter used as a manometer and introduced through a connector at the proximal end of the tracheal tube. The manometer was filled with saline and transduced (using a Nihon Kohden Lifescope 8) with a saline–air interface. This dynamic pressure was found to be equal to the pressure recorded on a Bicore pulmonary monitor (CP100) placed within the tracheal segment proximal to the water trap.

EXPERIMENTAL PROCEDURE

The artificial trachea was intubated with a full length (32 cm) size 8 mm internal diameter tube (Portex


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Profile). Intra-cuff pressure was set at 25 cm H₂O manually with a cuff inflator (Monitor control inflator, VBM, Medizintechnik), and was monitored continuously throughout the test. The proximal tracheal tube was connected to a Bird ventilator (6400ST volume ventilator), and continuous positive airway pressure (CPAP) 10 cm H₂O was introduced via the Bird ventilator. When the pressure had stabilized, water coloured with methylene blue was added to the space above the cuff representing the subglottic space and above. The zero point was taken as the top of the cuff in contact with the artificial trachea. A set square and ruler fixed vertically alongside the artificial trachea allowed measurement from the zero point to the lower level of the meniscus. The water was added until a 10 cm head of pressure was achieved. The initial 10 cm H₂O of CPAP was used to hold the fluid in the column above the cuff until the ventilator setting was made, and PEEP/CPAP was adjusted to several different pressures above the cuff until the ventilator setting was made, then a PSV of 7 cm H₂O and trigger sensitivity of 1 cm H₂O were added. HPR during SV, PSV and intermittent positive pressure ventilation (IPPV) modes was recorded 20 min after reduction of CPAP/PEEP to the test level. This time was chosen because the column gradually decreased to an equilibrium level and was static with all ventilatory patterns after 20 min. In the IPPV mode there was no SV, tidal volume was 500 ml, breath rate was 15 bpm, the inspiratory-expiratory ratio was 1:2, and lung compliance was adjusted to create a peak inspiratory pressure at the ventilator of 20 cm H₂O.

MODES EVALUATED

Trough pressure was defined as the minimum pressure recorded from the manometer pressure–time tracing at the tracheal tube tip during the inspiratory phase. In the spontaneous ventilation mode (SV), trough pressure was set by manipulating the driving ventilator (Bear 3) volume–flow settings, until the pressure trace at the distal tracheal tube was found to mimic that in a patient’s trachea during SV (unpublished observations in human subjects). This was measured with the same technique during settled (–2 cm H₂O trough pressure) and laboured (–7 cm H₂O trough pressure) respiration (fig. 2). A spontaneous rate of 20 bpm was set on the driving ventilator. PEEP of 10 H₂O was then introduced before instilling fluid above the cuff.

For the SV + pressure support (PSV) modes, trough pressure was set as above, then a PSV of 7 cm H₂O and trigger sensitivity of 1 cm H₂O were added. HPR during SV, PSV and intermittent positive pressure ventilation modes was recorded 20 min after reduction of CPAP/PEEP to the test level. This time was chosen because the column gradually decreased to an equilibrium level and was static with all ventilatory patterns after 20 min. In the IPPV mode there was no SV, tidal volume was 500 ml, breath rate was 15 bpm, the inspiratory-expiratory ratio was 1:2, and lung compliance was adjusted to create a peak inspiratory pressure at the ventilator of 20 cm H₂O.

Figure 2  Pressure profile measured by the central venous catheter with the tip at the distal end of the tracheal tube in (A) a patient and (B) the mechanical model.
Leakage past the tracheal tube cuff

Tube along 4 cm of the length of the artificial trachea repeatedly (approximately 15 times) over a 6-h period.

Statistical Analysis
Mean and 95% confidence limits for rate of leakage and HPR were calculated (using a Casio scientific calculator), the confidence interval being SEM × 1.96.

Results
HPR remained the same with cuff pressures of 25 cm H₂O compared with 60 cm H₂O (fig. 5), that is 6.42 (6.38–6.46) cm compared with 6.46 (6.29–6.63) cm at 8 cm H₂O of CPAP and 3.52 (3.33–3.71) cm compared with 3.52 (3.41–3.63) cm at 5 cm H₂O of CPAP. However, the rate of flow past the cuff decreased when cuff pressure was increased from 25 to 60 cm H₂O, for example with a 3-cm H₂O head of pressure above the cuff, the rate of leakage was 0.27 (0.21–0.33) ml s⁻¹ and 0.04 (0.03–0.05) ml s⁻¹, respectively (fig. 3).

CPAP and PEEP protected against leakage. Approximately 1 cm head of pressure was resisted for every 1 cm H₂O that CPAP/PEEP was increased (fig. 6). This finding was true for all HVLP cuffs tested. HPR was significantly greater at each level of CPAP for the Mallinckrodt tube, apart from at zero pressure. With a CPAP of 5 cm H₂O, HPR for the Mallinckrodt tube was 4.66 (4.47–4.85) cm H₂O, significantly higher than HPR for the Portex tube (3.52 (3.33–3.71) cm H₂O). More negative trough pressures during spontaneous ventilation reduced HPR at all CPAP levels. With a CPAP of 5 cm H₂O, a decrease in trough pressure from −2 to −7 cm H₂O reduced HPR from 3.22 (3.05–3.39) cm H₂O to 2.08 (1.98–2.18) cm H₂O (fig. 7). PSV of 7 cm H₂O increased HPR to 5.56 (5.46–5.66) cm H₂O and 3.52 (3.22–3.82) cm H₂O, respectively (fig. 8).

IPPV had a protective effect equivalent to approximately 1.5–2.5 cm H₂O of PEEP/CPAP, for
example at 5 cm H$_2$O of CPAP/PEEP, IPPV increased HPR from 3.52 (3.33–3.71) cm H$_2$O, with no ventilation but constant CPAP, to 4.94 (4.82–5.06) cm H$_2$O (fig. 9).

Neither open nor closed tracheal suctioning protected against generation of large negative tracheal pressures (less than – 40 cm H$_2$O) or leakage of fluid in this model. Movement of the tube within the trachea caused minimal leakage. Loss of cuff pressure (to 15 cm H$_2$O) caused rapid leakage independent of CPAP. On deflation of the cuff, however, it was possible to expel some of the fluid out of the model, particularly at higher CPAP/PEEP levels (e.g. 15 cm H$_2$O). It was possible to ventilate the model for 24 h with no leakage (for example with an SV rate of 15 bpm, trough pressure ≤ 2 cm H$_2$O, PSV 7 cm H$_2$O and PEEP 5 cm H$_2$O, and 4 cm of fluid above the cuff). After this period of no leakage, the tracheal tube was disconnected as is our practice for tracheal suctioning. At disconnection rapid leakage of fluid to the trachea occurred; this was increased with suctioning. Application of KY jelly to the cuff before intubation protected against leakage during all ventilatory modes studied. Application of high airway pressures during bagging, or movement of the tube over time, allowed a jelly filled channel to open, and leakage to occur. When a channel had cleared of jelly the leakage persisted over time.

Discussion

This model of an intubated trachea did not attempt to simulate the surface of contact between the tracheal mucosa and the cuff, the static or dynamic properties of the tracheal and extra-tracheal tissues during ventilation, the properties of different consistencies of secretions or the effect of mucus on the cuff–tracheal interface. Fluids of different viscosities were not assessed and it is possible that rate of leakage would be reduced and HPR increased with viscous secretions. Within the confines of a benchtop model, this study suggests aspects of ventilatory management which are protective against leakage and aspects which make leakage more likely.

Leakage occurs down longitudinal folds within the cuff walls, and these folds formed in all HVLP cuffs tested. Testing different tubes showed that HVLP cuffs of a range of specifications all behaved similarly regarding leakage, the Mallinckrodt tube having a marginally but significantly higher HPR when CPAP/PEEP levels were greater than zero. This protective effect of the Mallinckrodt tube might be beneficial when the subglottic space is suctioned mechanically to reduce secretions above the cuff. Indeed, the rate of ventilator-associated pneumonia can be reduced with the use of mechanical subglottic secretion drainage and this cuff (Mallinckrodt Hi-Lo EVAC tube).

Several factors increasing the possibility of leakage were identified. Tracheal suctioning through a 14CH catheter caused tracheal pressures to decrease to less than – 40 cm H$_2$O and caused rapid leakage. Increasing cuff pressure from 25 to 60 cm H$_2$O decreased the rate of flow past the cuff, but did not increase HPR, so the column of fluid decreased to...
the same level ultimately, although it took longer to do so. Clinically, this exerts unacceptable pressures on the tracheal wall,2 but it might be acceptable to increase cuff pressure for brief periods during times when the risk of leakage is greatest. A decrease in cuff pressure to 15 cm H₂O caused rapid leakage; this can occur clinically with a leak from the cuff or pilot balloon, a change in tracheal compliance or a change in position or angle of the cuff within the trachea. The high variability of the data for flows at a cuff pressure of 15 cm H₂O may have been caused by errors in measuring short time intervals, with high flows, using a stopwatch.

Aspects protective against leakage are those that increase the pressure head of fluid resisted above the cuff. CPAP and PEEP were protective (approximately 1 cm head of pressure was resisted for every 1 cm H₂O of CPAP/PEEP for all cuffs), as was expected. This held for all HVLP cuffs tested, the Mallinckrodt cuff performing modestly better for a given CPAP level. If fluid builds up to above the level of CPAP/PEEP, then leakage occurs on a millilitre-for-millilitre basis as fluid is formed, provided the obligatory fold in the cuff is patent and not occluded with KY jelly or viscous secretions. Application of KY jelly to the cuff before intubation was protective against leakage until an air leak developed. This air leak was small and not detectable on the inspiratory–expiratory tidal volume monitors. This suggests that the temporary plugging of the cuff channels by application of jelly before intubation could be beneficial in the short term.

More negative trough pressures (−7 cm H₂O) during spontaneous ventilation, representing laboured respiration (for example during testing for response to painful stimuli in an obtunded patient), reduced HPR at all CPAP levels and were therefore more likely to produce leakage. PSV protected against leakage during spontaneous respiration.

The protective effect of IPPV was equivalent to only a few centimetres of PEEP. HPR in the trough pressure of −2 cm H₂O + PSV group was higher than that of the IPPV group, which was higher than the no ventilation group for a given CPAP/PEEP. The reason for this might be because of a shorter expiratory phase in the −2 cm H₂O trough pressure + PSV group than in the IPPV group, or the nature of the cyclical cuff compression with each ventilatory waveform.

The time (20 min) allowed for equilibrium in the ventilated groups was necessary because of the nature of the leak in the period before equilibrium was reached. A column of fluid would form in a fold, and a drop would drip into the distal model trachea initially with every ventilator cycle, then as the level approached equilibrium a drip would occur with increasingly less frequent cycles. Between each of these cycles the column within the fold would be expelled proximally with each increase of airway pressure, and form again when the distal tracheal pressure decreased. No further drops fell after a 20-min period.

Movement of the cuff within the trachea caused minimal leakage, but loss of cuff pressure (to 15 cm H₂O) caused rapid leakage independent of CPAP. It was possible, however, to expel some of the fluid out of the model, particularly at higher PEEP levels (e.g. 15 cm H₂O); this represents the clinical practice of those who apply PEEP or a large tidal volume on deflating the cuff at extubation.

In patients undergoing ventilation in the ICU with tracheal colonization, 85% were found to have identical microorganisms on both sides of the cuff.6 It is likely that many cases of ventilator-associated pneumonia are a result of this route of contamination.7 In ICU patients, up to 150 ml per day can be removed from the subglottic space by intermittent aspiration using a Hi-Lo EVAC tube (Mallinckrodt),8 and in our experience with a patient prone, over 50 ml per hour at times could be collected dripping from the nose and mouth (unpublished observations). In supine or semi-recumbent patients, secretions do not overflow from the mouth, but pool in the oropharynx. When a pool has collected, further secretion production is presumably either inhibited, reabsorbed through oropharyngeal mucosa or drains into either the stomach or past the tracheal cuff. In this model, if fluid is added continually above the cuff, it continues to drain to the model trachea at the same rate, maintaining a constant pool above the cuff equal to the head of pressure resisted for this ventilatory pattern.

If this model represents the clinical situation then there are readily identifiable times when a patient with an intubated trachea aspires subglottic fluid to the trachea, and manoeuvres that protect against this. Manoeuvres to reduce pressure differentials across the cuff, such as large tracheal tube diameters, better demand valves to avoid decreases in airway pressure and CPAP/PEEP would increase protection against leakage of subglottic fluid. This pool, however, would be available for leakage at any time during routine care when this protection was lost.

In summary, in an intubated model trachea, when a patent longitudinal fold exists in the HVLP cuff, there are identifiable times when gross leakage past the cuff occurs. This happens when the pressure difference across the cuff is favourable for flow to the trachea. Such clinical situations would be: (1) tracheal suctioning with 14CH catheters, either open or closed; (2) loss of cuff pressure; (3) loss of PEEP/CPAP such as disconnection from the ventilator; (4) continuing secretion production when the maximal protective effect of the particular ventilatory pattern has been reached; and (5) negative tracheal inspiratory pressures.

The possibility of leakage in this model is reduced when the pressure difference across the cuff favours flow from the trachea or the channels are blocked. This would occur with: (1) Trendelenburg position or subglottic aspiration before disconnection, extubation or suctioning the trachea; (2) maintaining CPAP/PEEP in the trachea, at all times; (3) use of PSV/IPPV and not allowing SV through a tube without support; (4) KY jelly lubrication on intubation; and (5) reduction in the subglottic and pharyngeal pool.

A new design of cuff in which folds are not present and wall pressure is maintained at acceptable levels would offer advantages. If such a cuff could be
maintained at a tracheal wall pressure of 30 cm H₂O reliably, then the barrier to fluid flow past the cuff would be at least equal to a HPR of 30 cm H₂O, and would withstand at least negative 30 cm H₂O tracheal pressures. This would prevent leakage under most clinical circumstances. There is a need for the development of a tracheal tube cuff which protects against leakage, while exerting acceptable pressures on the tracheal wall.

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References


