Esophageal Aspiration of Air Through the Drain Tube of the ProSeal™ Laryngeal Mask

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The ProSeal™ laryngeal mask airway (PLMA) has two lumens that emerge from the patient’s mouth (1). The airway tube communicates with the bowl of the PLMA and acts as an extension of the tracheobronchial tree. The drain tube (DT), which exits at the leading tip of the mask, acts as an extension of the esophagus.

We present two patients who experienced partial upper airway obstruction while breathing spontaneously with the PLMA. The first case exhibited behavior characteristic of a variable extrathoracic airway obstruction. The second case displayed features of a fixed obstruction. As a result of the airway obstructions, the patients developed exaggerated negative excursions in pleural pressure. In both cases the esophagus was observed fiberoptically to open during inspiration as a result of the negative pleural pressure, causing aspiration of room air through the DT. Esophageal collapse and belching occurred during expiration.

Case Report #1

A 36-year-old male (170 cm, 97 kg) with a medical history of anxiety, claustrophobia, and recent ankle fracture presented for open reduction internal fixation and adjustment of an existing external fixator. For his previous ankle surgery he had received a subarachnoid block. The patient now requested a general anesthetic.

The patient received midazolam 2 mg IV for premedication and was induced with propofol 300 mg IV and fentanyl 100 µg IV. Mask ventilation with sevoflurane and insertion of a PLMA size 5, using the finger insertion technique (2), took place uneventfully. The cuff was inflated with 30 mL air until the cuff pressure was 60 cm H₂O. Positive pressure ventilation (PPV) with the PLMA was unobstructed, with normal chest movement and also a normal capnograph.

A “soap bubbles” drain tube test was then performed, consisting of placing a nontoxic soap solution (such as used by children to blow bubbles) across the proximal end of the DT (3). If, during PPV, the DT blows a bubble this test indicates the presence of a leak of the mask/esophageal seal. In the present case we administered 40 cm H₂O positive pressure without any bubble formation; an oropharyngeal leak occurred at 40 cm H₂O pressure instead.

The patient was kept ventilating spontaneously with an inspired sevoflurane concentration of 4%. Fentanyl 350 µg IV was titrated while the surgeons began manipulating the ankle, yet the patient remained tachypneic, with tidal volumes of 260 mL. An additional dose of morphine 10 mg IV was given without any change in respiratory rate or tidal volume.

We then began noticing that the respirations via the PLMA were noisy, especially during inspiration. There were no other obvious signs of upper airway obstruction, such as tracheal tug, head bobbing, intercostal retractions, or thoracoabdominal discoordination. Next, belching noises were noted to be regularly emanating from the DT. We repeated the soap bubble test (3) to assess positioning. We now noticed that the soap membrane was powerfully drawn into the DT during each inspiration. The soap bubble DT test revealed negative DT pressure. During belching/expiration, bubbles were blown from the DT tip. Lubricant gel in the DT, as suggested in the instruction manual (2), did not move.

Fiberoptic examination via the airway tube of the PLMA was performed, as shown in Figure 1. The PLMA was well positioned, with the distal tip situated behind the cricoid cartilage. Further fiberoptic examination showed that the cuff occupied both pyriform fossae. The epiglottis was not downfolded. The aryepiglottic folds, valleculae, and lingual tonsillar tissue could all be visualized. The glottic opening, however, seemed narrowed and both vocal cords were drawn inwards during inspiration. This resulted in airway obstruction during inspiration. During exhalation the glottis widened. The arytenoids were motionless.

Next, we examined the DT fiberoptically, as shown in Figure 2. During inspiration the esophageal lumen opened. During expiration the esophageal lumen collapsed. The esophagus enlarged and collapsed in synchrony with the patient’s respirations. Air was alternately being drawn into the esophagus and subsequently being expelled.

Because of our concern about esophageal air, we administered rocuronium 50 mg and finished the case using PPV with peak airway pressures 24 cmH₂O and tidal volumes 600 mL. The patient’s stomach did not feel distended. We passed an orogastric tube (OGT) via the DT once during the case and once before emergence, with scant return at both

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times. Otherwise, we left the DT alone and did not occlude it. The PLMA was removed with the patient awake and there were no postoperative complications.

Case Report #2

A 61-year-old female (155 cm, 66 kg) with a medical history of hypertension, hypercholesterolemia, and anemia presented for hysteroscopy. Her physical examination was normal and a general anesthetic was planned. She was induced with propofol 160 mg and fentanyl 100 µg, ventilated by mask with sevoflurane 4%, and then had a PLMA size 4 placed using the finger-insertion method (2). The cuff inflated was with 20 mL air until the pressure reached 60 cm H2O.

Ventilation through the PLMA was slightly abnormal with a prolonged expiratory phase. This prolonged expiration was felt clinically with the circle system bag and was also demonstrated by the capnograph with a delayed increase in end-tidal CO2. The mask had an oropharyngeal leak at 24 cm H2O; this was less than expected. The soap-bubble test (3) did not reveal any DT leak with PPV. The leak at 24 cm H2O was oropharyngeal rather than at the mask/esophageal seal.

The patient was kept ventilating spontaneously with an inhaled sevoflurane concentration of 3%. Fifteen minutes later, soft stridorous noises were heard and slight tracheal tug was noted. A soap-bubble test was performed that demonstrated negative DT pressures during inspiration, with the soap membrane being sucked down the tube. During expiration, the same test showed bubbles emanating from the DT tip.

Figure 3 shows the fiberoptic view through the airway tube and the PLMA position. The mask is fully inserted, with the tip located behind the cricoid cartilage. The PLMA cuffs are malpositioned. They are rotated medially inwards, partially covering the bowl of the mask. The epiglottis is downfolded, compromising the airway further. It does not appear as if flow can go around the downfolded epiglottis, and the only aperture is between the epiglottic tip and arytenoids. When the cuff was deflated, it collapsed over the arytenoids and caused near-complete airway obstruction. When the cuff was inflated beyond 60 cm H2O, the airway was not changed substantially from that shown in Figure 3.

As in Case 1, the esophagus opens during inspiration and collapses during expiration. The circuit bag was then removed from the anesthetic circle system. By occluding the arm connected to the bag of the circle system it was determined that the patient’s maximum inspiratory effort was negative 12–14 cm H2O. The bag was then reattached. The stomach did not feel distended. The DT was not occluded, and an OGT was not passed. The patient was maintained spontaneously ventilating for another 20 min and then had an uneventful emergence from anesthesia. There were no postoperative complications.

Discussion

We have presented two cases in which patients experienced partial upper airway obstruction while spontaneously ventilating with the PLMA. In both cases, the increased respiratory effort exerted to overcome
the airway obstruction resulted in esophageal aspiration of room air through the DT. We discuss the mechanism for esophageal aspiration of air (one form of aerophagia), belching, and the potential risks of esophageal air. Equally important, we analyze the two forms of airway obstruction that these patients encountered.

Esophageal pressure is normally equated with pleural pressures; in fact, an esophageal probe is often used to measure pleural pressure (4). Both esophageal and pleural pressures are usually negative during inspiration. During spontaneous inspiration with an obstructed airway, the extent of negative pressure becomes exaggerated. Cases 1 and 2 demonstrate how this easily results in aspiration of room air through the DT. We discuss the mechanism for esophageal aspiration of air (one form of aerophagia), belching, and the potential risks of esophageal air. Equally important, we analyze the two forms of airway obstruction that these patients encountered.

Esophageal pressure is normally equated with pleural pressures; in fact, an esophageal probe is often used to measure pleural pressure (4). Both esophageal and pleural pressures are usually negative during inspiration. During spontaneous inspiration with an obstructed airway, the extent of negative pressure becomes exaggerated. Cases 1 and 2 demonstrate how this easily results in aspiration of room air through the DT. In Case 2 the inspiratory pressure was only negative 12–14 cm H₂O. Neither patient had dramatic evidence of upper airway obstruction (marked intercostal retractions, tracheal tug, rocking thoracoabdominal motions, or oxygen desaturation); the only sign was inspiratory stridor. The ease with which esophageal aspiration of air occurred in Cases 1 and 2 suggests that the DT can stent open and bypass the upper esophageal sphincter.

Once the esophageal lumen opens, it is no longer a potential space. Its pressure equalizes with atmospheric despite pleural pressure continuing to decrease with completion of inspiration. At the end of inspiration esophageal pressure remains atmospheric and esophageal-wall and surface tension exactly balance the maximum negative pleural pressure. When the expiratory cycle begins and the pleural pressure begins to ascend, esophageal pressure increases above atmospheric. Esophageal pressure increase can be dramatic if exhalation is active and rapid and if egress of esophageal air is obstructed–esophageal pressure equals pleural pressure plus pressure contributed by esophageal-wall and surface tension. If exhalation is passive and slow and egress of esophageal air is unobstructed, esophageal pressure will increase only slightly above atmospheric pressure. Egress of esophageal air through the drain tube is heard as belching or eructation (5).

If a patient has normal anatomy, an intact lower esophageal sphincter, and a normal abdominal pressure exceeding pleural pressure, it is likely that all of the esophageal air will be belched (5). The risk of esophageal aspiration of air is therefore slight. However, if inspiration and expiration are forceful, as might occur during emergence, air could potentially be diverted to the stomach. It is conceivable that esophageal pressure could overcome both lower esophageal sphincter tone and transdiaphragmatic pressure. Active swallowing of esophageal air could also cause gastric insufflation.

Cases 1 and 2 also dramatize two important etiologies of airway obstruction that we have encountered previously. For the purpose of discussion, it is helpful to classify the obstructions according to the nomenclature for upper airway obstructions (4). In Case 1 the principal airflow limitation occurred only during spontaneous inspiration. This is characteristic of variable extrathoracic airway obstructions (e.g., bilateral vocal cord paralysis, trying to breathe through a Penrose drain). With variable extrathoracic airway obstructions exhalation is not problematic; neither is PPV by mask (such as the PLMA). Variable extrathoracic airway obstructions are surrounded on their extraluminal side by atmospheric pressure. During spontaneous inspiration, when the tracheal pressure is negative, the obstruction undergoes dynamic inspiratory collapse (4). When the airway pressure is positive (exhalation, PPV) the obstructing region actually widens.

We believe that Case 1 resembles a patient with bilateral vocal cord paralysis. Despite the recommendation to deepen the anesthetic to abolish stridor (6), this strategy failed. During fiberoptic examination we observed normal positioning of the mask. The arytenoids appeared slightly tilted inwards. The vocal cords did not appropriately abduct during inspiration. The glottis appeared narrowed, especially near the anterior commissure. The result was a posterior glottic chink that was inadequate to maintain critical airway patency to resist Bernoulli forces. Figure 1 illustrates how the true vocal cords collapse passively with inspiration as a result of negative tracheal pressure and Bernoulli effect. No motion of the arytenoids is observed and we do not believe that there is a paradoxical motor component closing the glottis. To manage this situation we converted to PPV.

The features of Case 2 resemble a fixed airway obstruction. This patient encountered difficulty during both inspiration and expiration. The delayed expiration was noted soon after the PLMA was placed; the anesthesia bag filled slowly and the capnograph
demonstrated delayed increase. Fiberoptic examination of the cause of obstruction showed malposition of the PLMA cuff. In fact, this case demonstrates an important point that distinguishes the PLMA from the classic LMA. The PLMA was satisfactorily inserted to the depth of the upper esophageal sphincter yet caused partial airway obstruction as a result of medial infolding of the cuff over the bowl. By contrast, when a classic LMA is fully inserted to the depth of the upper esophageal sphincter (and it is not inserted completely into the esophagus, or grossly rotated), it is rare to have a compromised airway as a result of the cuff (6). We believe that several PLMA insertions may occasionally be necessary (despite adequate insertion depth) before an optimal mask/cuff position can be achieved.

A deeper bowl and a more compliant cuff distinguish the PLMA from the Classic LMA (1). This design increases the risk of cuff infolding, as demonstrated in Figure 3. We consider this to be an abnormal position. In a normally positioned PLMA, the cuff does not meet in the midline; rather it lies alongside the epiglottis on both sides. Fiberoptically it is usually possible to view the entire epiglottis, the valleculae, and lingual tonsillar tissue. Finally, the tip of the PLMA is observed to pass behind the cricoid cartilage and the DT passes underneath the arytenoids, as well.

In Figure 3a, the PLMA cuffs are rotated inwards and meet midline in the wide, proximal portion of the bowl. Closer to the glottis (Fig. 3b) the separation between the cuffs widens. The epiglottis is down-folded, further compromising the airway. From the photographs it appears impossible for flow to go around the sides of the downfolded epiglottis, as likely occurs with the mask of a classic LMA. The only apparent airway is located between the downfolded epiglottis and the arytenoids. We consider cuff infolding to be a significant risk factor for airway obstruction with the PLMA. The epiglottis may play a role in stenting the cuff apart, but this is unknown.

In summary, we have discussed two cases of esophageal aspiration of air through the DT and have described the mechanism of partial upper airway obstruction in both of these cases. It is likely that most aspirated air will be belched out the DT. However, with significant obstruction and active expiratory efforts the risks from aerophagia could become substantial. Further investigation of the risks of spontaneous ventilation with the PLMA and of aerophagia in anesthetized patients is necessary.

References