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Postoperative pulmonary edema in young, athletic adults

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ABSTRACT

Pulmonary edema secondary to postextubation laryngospasm is a potentially life-threatening problem, demanding early diagnosis and prompt treatment. We believe that this problem has been grossly underestimated in its incidence, as only seven adults have been reported in the English literature, whereas seven adults have been observed at our institution in only a 24 month period. All were young, healthy, athletic adult males (average weight, 218 pounds) who underwent relatively minor, uncomplicated surgical procedures under general anesthesia.

Five of these patients were collegiate and/or professional athletes and had meticulous medical records detailing their clinical course. Clinical laryngospasm was noted immediately following extubation and anesthesia by mask with subsequent pulmonary edema. The diagnoses were confirmed by clinical examination, arterial blood gas determinations or pulse oximetry, and chest roentgenogram. Four adults required reintubation. Six of the seven adults demonstrated very rapid resolution of the pulmonary edema with prompt diagnosis and institution of a therapeutic regimen including oxygen, diuretics, reintubation, and/or positive pressure ventilation. In one patient, the problem was not immediately recognized, and progressed to florid pulmonary edema requiring emergent intubation 14 hours later in the emergency room, and 3 days of mechanical ventilation.

The etiology of pulmonary edema following upper airway obstruction represents an interplay between several factors: cardiogenic and neurogenic mechanisms, as well as hypoxia contribute. In this group, excessive negative intrathoracic pressure generated by forced inspiration against a closed glottis is the most likely, consistent, and logical explanation.

This study suggests that young, healthy, athletic males may be at increased risk for this complication. We believe that their enhanced ability to generate excessive negative intrathoracic pressures is, at least in part, responsible. A heightened awareness of the problem in this at-risk group should invoke special considerations, including choice of anesthesia, precautions on extubation, prolonged monitoring in the recovery phase if laryngospasm is observed or suspected, and rapid therapeutic intervention.

In 1977, Oswalt et al.[28] first described pulmonary edema following relief of upper airway obstruction. Since then, 44 patients have been reported. The majority of these patients (28) are in the pediatric age group. The exact pathophysiologic mechanism by which pulmonary edema occurs is multifactorial. Hypoxia, neurogenic phenomenon, and hydrostatic forces have all been implicated.[19] Expiration against a partial or complete obstruction produces intrinsic positive and expiratory pressure (PEEP). Some authors suggest loss of PEEP after relief of the obstruction is, in part, responsible for the problem.[32] The most prevalent theory attributes the increased pulmonary interstitial edema to the magnitude of negative inspiratory force or intrapleural pressure generated against the obstruction (modified Mueller maneuver).[19] This produces transpulmonary hydrostatic forces that favor fluid shift from pulmonary vasculature to alveoli.

The causes of reported upper airway obstruction are many and include tumors, interrupted hanging, croup, epiglottitis, enlarged tonsils, soft tissue mass, and laryngospasm.[3,4,6-8,13-17,21,22,24,28,32,33,35,38,40] Croup and epiglottitis are responsible for the vast majority of pediatric cases, the age group in which the problem has been best described.

Pulmonary edema as a consequence of upper airway obstruction in the adult is less common and not well understood. Tami et al.[36] reported three adults; the obstructing agents included aretynoid edema, supraglottic carcinoma,

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and laryngeal polyp. Lorch and Sahn[14] reviewed periintubation laryngospasm leading to pulmonary edema in adolescents and adults. Based on his review, he suggested that certain patients who are predisposed to airway obstruction (those with sleep apnea, a short thick neck, and obese patients) have an increased risk of airway complications upon extubation after general anesthesia.

At our institution between June 1987 and June 1989 we identified eight patients (seven adults and one adolescent) who have demonstrated immediate postextubation (six) or postgeneral anesthetic by mask (two) airway obstruction attributed to laryngospasm and subsequent pulmonary edema.

The purpose of this paper is to review our current understanding of the problem and, based on our own eight cases, proposed a previously undescribed population at risk for postextubation airway obstruction.

METHODS

The hospital records of eight patients known to have demonstrated a postextubation obstructive event and subsequent pulmonary edema were reviewed. The diagnosis of pulmonary edema was made by the radiographic appearance, arterial blood gas determinations or pulse oximetry, physical examination, and pink, frothy sputum production.

Specific attention was given to past medical history, allergies, problems with previous or subsequent surgery with general anesthesia, height, weight, the obstructive event, perioperative fluids, ECG, chest roentgenographic findings, time to the resolution of the pulmonary edema, as well as therapeutic interventions. Cases of postextubation laryngospasm and subsequent pulmonary edema have been described in other patients after upper airway procedures.[3, 4, 17, 22]

Patients who demonstrated either cardiomegaly on chest roentgenogram and/or ECG abnormalities underwent echocardiography to rule out cardiac etiology for the pulmonary edema. No anatomical or physiological cardiac abnormality was found. The data are summarized in Table 1.

While it is important to recognize and describe this problem in all eight of the patients in our series, we will focus on the seven adult patients. Their average weight was 218 pounds. Five of these were young, strong, healthy athletic males and all of whom played competitive intercollegiate and/or professional football. The remaining two adults were recreational athletes.

PATIENT EXAMPLES

Patient 4

A 24-year-old male (6 feet 3 inches, 225 pounds), former collegiate athlete, presented with a traumatic left Achilles rupture and was scheduled for operative repair. His medical history was unremarkable, and he had no drug allergies. Preoperative medications included midazolam and morphine. After routine induction using 400 mg of thiamylal sodium and 50 mg of fentanyl, the patient was intubated on the first pass without difficulty. Anesthesia was maintained with enflurane at 2% to 2.5%, as well as nitrous oxide at 30%. He was paralyzed with vecuronium. The procedure lasted approximately 45 minutes with a minimal blood loss and 1600 cc of crystalloid fluid replacement. The patient's intraoperative course was unremarkable with stable vital signs and oxygenation, and he was given neostigmine and glycopyrolate to reverse his paralysis approximately 45 to 50 minutes after surgery began.

Immediately upon extubation, he demonstrated strider and coughing, symptoms consistent with laryngospasm. His oxygenation on pulse oximetry decreased to 55%, and he was promptly reintubated in the operating room. There was no antecedent emesis. A large amount of pink, frothy sputum was suctioned from his endotracheal tube and chest roentgenogram revealed bilateral pulmonary infiltrates consistent with pulmonary edema (Fig. 1). He was taken to the recovery room and was noted to be "sleepy but thrashing." He was placed on a mechanical ventilator with a PEEP of 5 cm H_2O under constant pulse oximetry. His skin color was dusky, with saturation on the monitor of 70% on 100% FiO_2. He was repeatedly suctioned for bloody secretions in the endotracheal tube. Twenty-five minutes after the reintubation, the patient was maintained with 100% FiO_2 with a saturation of 85%, a pO_2 of 57, a pCO_2 of 53, and a pH of 7.25. He was given 5 mg of midazolam to relax him because of his agitation plus 10 mg of furosemide at that time. Another chest roentgenogram 40 minutes after the reintubation showed the same pulmonary edema signs. One hour and 20 minutes after his reintubation, while on the ventilator with a PEEP of 5 cm H_2O and FiO_2 of 100%, his arterial blood gases revealed a saturation of 99%, a pO_2 of 250, a pCO_2 of 51, pH of 7.29, and a bicarbonate of 24. Proper ventilator adjustments were made, and his FiO_2 was decreased to 70%. He continued to "buck" the ventilator and was suctioned many times for bloody secretions. By 2 hours after surgery, under constant pulse oximetry monitoring, his FiO_2 was weaned to 50% with a 95% saturation. One hour after this he again received 10 mg of intravenous furosemide. By 4 hours after reintubation, the patient was disconnected from the mechanical ventilator but remained intubated with an FiO_2 of 50%. His oxygen saturation by pulse oximetry was 98% at that time.

Approximately 20 minutes after discontinuance of the ventilator, while fully awake, and demonstrative adequate ventilatory parameters, the patient was given lidocaine spray per endotracheal tube, extubated, and placed on 100% oxygen by face mask. The extubation was uncomplicated. Auscultation revealed coarse bilateral breathing with good air exchange. His initial arterial blood gases on 100% oxygen by face mask revealed a saturation of 99%, a pO_2 of 240, a pCO_2 of 51, pH of 7.34, and bicarbonate of 28. He was eventually weaned to 35% O_2 per mask by 5½ hours after reintubation. His arterial blood gases at that time revealed a saturation of 95%, a pO_2 of 77, a pCO_2 of 40, and normal

TABLE 1
Summary of patient data

Patient	Age	Sex	Ht./Wt.	DX./procedure	(cc) Fluids	Obstructive event	Treatment	Clinical course/resolution
1	32	M	6'1"/270	Lateral internal sphincterotomy for hemorroids	160	Dyspnea and coughing following anesthesia by mask	Treatment delayed 14 hours; emergently intubated furosemide, steroids	Intubated for 3 days, hospitalized for 8 days
2	18	M	5'9"/196	Knee arthroscopy lateral meniscus tear, ACL	1600	Cough and stridor on extubation	Succinylcholine, + pressure ventilation, furosemide, oxygen	Weaned to 4 liters oxygen (98% sat.) 6 hours after laryngospasm, hospitalized for 48 hours
3	24	M	6'/220	Knee arthroscopy medial collateral ligament tear	1800	Stridor, cough, poor inspiration on extubation	Oxygen, naloxone (lethargic in paru), nebulized bronchodilator	Oxygen requirement decreased from 70% to room air in 6 hours, hospitalized 18 hours
4	24	M	6'3"/225	Achilles tendon repair	1600	Immediate laryngospasm after extubation	Re-intubated, furosemide, oxygen	Oxygen requirement weaned to 2 liters at 6½ hours, extubated at 6 hours, hospitalized <36 hours
5	22	M	6'2"/262	Hardware removal S/P shoulder reconstruction	600	Stridor, immediate upper airway obstruction on extubation	Re-intubated, furosemide, oxygen	Oxygen weaned from 100% to 30% in 4 hours, extubated 10 hours after incident, hospitalized 22 hours
6	20	M	6'/170	Knee arthroscopy ACL tear	2100	Immediate stridor on extubation	Re-intubated, oxygen, furosemide	Weaned to 39% oxygen at 9 hours, extubated at 11 hours, hospitalized 22 hours
7	25	M	/185	Debridement of burn, hand	1700	Short bout of laryngospasm after reversal of anesthesia. (MASK) saturation fell to 60%	Succinylcholine, + pressure oxygen, racaemic epinephrine, observation	Weaned to 2 liters oxygen by 22 hours (details of oxygenation unavailable), hospitalized 3 dyas (skin graft)
8	13	M	/125	Septorhinoplasty for fracture	1500	Laryngospasm upon extubation	Succinylcholine, +pressure oxygen, observation, furosemide	Weaned to room air by 19 hours (detail of weaning unavailable), hospitalized 21 hours

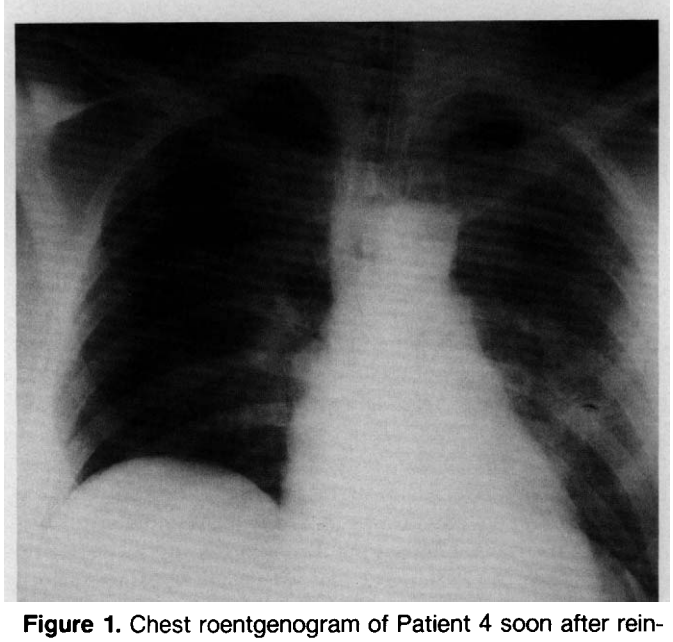

Figure 1. Chest roentgenogram of Patient 4 soon after reintubation demonstrating bilateral pulmonary infiltrates consistent with pulmonary edema.

pH and bicarbonate. Another roentgenogram was taken at this time and revealed resolving pulmonary edema. By 8 hours after surgery, the patient was still on 35% oxygen by face mask, his pO_2 had improved to a saturation of 98% with a pO_2 of 110, and chest roentgenogram revealed continued resolution of the pulmonary edema.

He was transferred from the recovery room to the nursing floor 8½ hours after surgery, and was placed on 2 liters of oxygen by nasal cannula. Oxygenation at that time revealed a saturation of 98% with a pO_2 of 110 and a pCO_2 of 45. His pulmonary examination was remarkable for bibasilar rales, although there was significant interval improvement. There was significant diuresis of nearly 2 liters of urine between 1 to 9 hours after reintubation. The oxygen was discontinued the following morning, and the patient experienced no dyspnea, chest pain, or anxiety. Pulmonary examination revealed his lungs to be clear to ausculation. He was discharged from the hospital the morning after surgery procedure with no recurrent signs of cyanosis, strider, symptoms of dyspnea, or productive cough. At 7 months after surgery, there was no recurrence of symptoms. He has resumed regular aerobic exercise without difficulty.

Patient 5

A 22-year-old male (6 feet 2 inches, 262 pounds), an offensive lineman on a collegiate football team, underwent surgery for

removal of a painful staple. He had no significant past medical history. Eight months earlier, he had undergone an entirely uncomplicated right shoulder reconstruction for anterior instability.

In the supine position, after a standard anesthetic induction of 400 mg of thiamylal sodium, 250 mg of fentanyl, and 140 mg of succinylcholine, the patient was intubated on the first pass without any difficulty. The anesthetic was maintained with enflurane (initially at 5%) and nitrous oxide without difficulty. The patient was paralyzed with vecuronium. Pulse oximetry was maintained with oxygen saturation above 95%. The staple was removed and the case was uncomplicated. The patient received 600 cc of crystalloid fluid with a minimal blood loss. Forty-five minutes after induction, his paralysis was reversed with neostigmine and inhalation agents were reduced for "wake-up." After the patient had been suctioned and given 100 mg of intravenous lidocaine, he was felt to be "responsive." He was extubated at that time and immediately began vigorous coughing and strider without antecedent emesis. He had obvious airway difficulty and demonstrated laryngospasm at clinical presentation.

The patient had immediate desaturation by pulse oximetry to 50% and was urgently reintubated on the first pass without problems. He remained very agitated and required paralysis with vecuronium after the intubation. Upon arrival in the recovery room, a copious amount of pink, frothy sputum was suctioned from his endotracheal tube. The patient's immediate postreintubation examination revealed rales throughout all lung fields, with upper airway rhonchi present. Monitoring by pulse oximetry continued, and his FiO_2 was gradually decreased to 50% by 30 minutes after reintubation. Arterial blood gases on 50% FiO_2 revealed a saturation of 95%, a pO_2 of 83, a pCO_2 of 42, pH of 7.34, and a bicarbonate of 23. His FiO_2 was increased to 100% and he was given 20 mg of furosemide. A chest roentgenogram revealed diffuse pulmonary edema (Fig. 2). Under constant pulse oximetry monitoring, the patient's FiO_2 was slowly decreased.

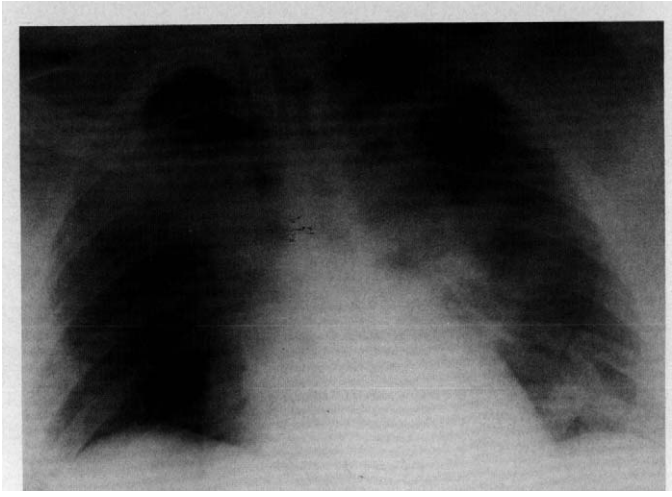

Figure 2. Chest roentgenogram of Patient 5 within 1 hour of his obstructive event, demonstrating diffuse pulmonary edema.

At 2 hours after reintubation on 50% FiO_2, the patient's arterial blood gases revealed a saturation of 99%, a pO_2 of 220, a pCO_2 of 29.5, and a pH of 7.53. Ventilator adjustments were made, and he was maintained on a PEEP of 5 cm of H_2O. His breathing sounded much more clear by 2½ hours after surgery. At 3 hours after surgery, the patient was weaned to 30% FiO_2, with a saturation of 99% and a pO_2 of 273. He remained intubated throughout the evening with no further diuretic administration. His agitation resolved with increased pO_2.

By the next morning, he continued to demonstrate saturations of over 95% by pulse oximetry on 30% FiO_2. His endotracheal tube was suctioned, and he was given 100 mg of intravenous lidocaine. He was extubated while alert approximately 14 hours after his initial reintubation, and had arterial blood gases on 4 liters of oxygen by nasal cannula of 99% saturation, a pO_2 of 153, a pCO_2 of 37, with a pH of 7.33 and a bicarbonate of 22. His oxygen was subsequently discontinued. Eight hours after this, the patient remained asymptomatic with clear breath sounds. A chest roentgenogram at that time was clear. He had no complaints of dyspnea, chest pain, nor any symptoms of coughing or strider. He was discharged that afternoon. At the 6 month followup he had experienced no recurrence of his respiratory problems.

RESULTS

The seven adult patients in this series share many common factors (Table 1). Five were young, strong males in excellent physical condition (average weight, 236 pounds) and were collegiate and/or professional athletes. The remaining two patients were recreational athletes in very good condition. All were Class I anesthesia risk. All surgical procedures done on these patients were of relatively short duration. While no vocal cord visualization occurred in any of the seven, all had coughing, retractions, and/or strider immediately following extubation, or correlating with the end of anesthesia, which was deemed to be consistent with laryngospasm. In all cases, this respiratory event was associated with an immediate fall in oxygen saturation measured by pulse oximetry, which is monitored continuously throughout anesthesia at our institution. Four patients required reintubation. Six of the seven demonstrated very rapid resolution of the pulmonary edema after prompt diagnosis and institution of a therapeutic regimen including oxygen, diuretics (four patients), and in Patients 2 and 4 through 7, reintubation and/or positive pressure ventilation after paralysis. While the amount of fluids varied, these figures are not consistent with fluid overload in the presence of normal cardiovascular function after overnight fasting.

In five of the six patients in whom detailed hourly oxygenation data is available (Patients 1 through 6), a significant decrease of oxygen requirement was noted within 6 to

9 hours of oxygen supplement. Withdrawal of oxygen entirely was seen within 27 hours in these five cases.

Patient 1 is somewhat unique in that his signs and symptoms were not recognized immediately after surgery. He was discharged from the recovery room 2 hours after his procedure with persistent cough and dyspnea. He returned to the emergency room 14 hours after his surgery with increased dyspnea, cough producing a frothy sputum, and pO_2 of 28. A chest roentgenogram showed obvious florid pulmonary edema. This patient required ventilatory support for 3 days.

None of the seven patients have experienced a recurrence of this phenomenon at 3 to 24 months of followup. Patient 2 has subsequently undergone general endotracheal anesthesia for reconstruction without incident, and Patient 1 has undergone carpal tunnel release with intravenous regional anesthesia and sedation without problems.

DISCUSSION

While pulmonary edema secondary to upper airway obstruction is becoming a more easily recognized problem, the mechanisms by which it occurs are not well understood. The etiology is likely to be multifactorial. Hypoxia may be, at least in part, responsible. Decreased partial pressure of oxygen triggers massive sympathetic discharge, constricting peripheral arterial beds, and causing increased venous return to the thorax.[19,38] In addition, hypoxia is known to cause pulmonary vascular constriction.[25] This constriction coupled with already expanded pulmonary venous volumes causes increased pulmonary capillary pressure and favors hydrostatic fluid shift into the pulmonary interstitium. In reexpansion of pneumothoraces, prolonged hypoxia may cause pulmonary capillary damage and has been suggested as a cause of pulmonary edema.[20] This mechanism may play a role in postextubation pulmonary edema. Moss et al.[25] demonstrated pulmonary edema in several different species with isolated cerebral perfusion of hypoxic blood. He proposed that the pulmonary response was neurologically mediated. This is not unlike the changes seen with high altitude pulmonary edema.[11,37]

In Patient 1, we postulate that delay in diagnosis and the resultant sustained hypoxia may have contributed to the increased membrane permeability and/or membrane damage. This would account for his protracted recovery (when compared to others in this study) and underscores the need for prompt recognition and treatment. This patient had elevated serum CPK to 10,000 units per liter and his urine sediment contained tubular casts. This enzyme elevation and apparent rhabdomyolysis can be explained on the basis of prolonged hypoxia, although he is currently undergoing evaluation to rule out primary myopathy. Interestingly, this patient played professional football and experienced a similar constellation of symptoms when playing on a cold day in Denver's Mile High Stadium. In retrospect, he appears to have suffered from high altitude pulmonary edema, a phenomenon not well understood, but believed to be partially caused by persistent hypoxia.[11,37] This raises some question about the simplicity of pure hydrostatic etiology of postlaryngospasm pulmonary edema.

Altered cardiac function with changes in intrapleural pressure are also well documented. Using a dog model, Robotham et al.[29] found an increase in left ventricular filling pressure in animals during Mueller maneuver. They concluded that this occurred as a result of increased sympathetic discharge, rather than pooling of blood in the lungs. Buda et al.[2] found that large intrathoracic pressure changes influenced cardiac performance. These changes included left ventricular load, decreased ejection fraction, and a rise of 18% to 25% of end systolic volume with negative inspiratory force of −60 cm H_2O. These changes serve to increase pulmonary venous capillary pressure favoring transudation. The contribution of cardiac factors to pulmonary edema in this clinical setting is unclear but appears, in part at least to play a role.

While cardiac and neurologic mechanisms likely contribute to the development of pulmonary edema, pure hydrostatic transudation is the most prevalent explanation. Intrapleural pressures vary from −3 to −9 mm Hg (−4 to −12 cm H_2O) with normal respiration.[30] Maximum negative alveolar pressures that can be expected by adult men range from −100 to −140 cm of water.[5,39] Maximum intrapleural pressures of −38 (−51 cm H_2O) and −50 mm Hg (−67 cm H_2O) have been shown in asthmatics and adults with upper airway obstruction, respectively.[26,34] In 1950, Haddy et al.[10] measured pulmonary vascular pressures in dogs with occluded airways and assessed pulmonary edema at autopsy. They found that pulmonary edema was produced by airway obstruction during inspiration or expiration. Both occlusions caused increased pulmonary venous pressure that correlated with the amount of pulmonary edema. Using a rabbit lung preparation, Smith-Erichsen and Bo[3] found an 8-fold increase in pulmonary edema when intrapleural pressure against the closed glottis was decreased 5 cm H_2O. This theory, at least in part, explains unilateral pulmonary edema along with rapid reexpansion of pneumothoraces.[12]

Since Ostwalt et al.[28] described the problem in 1977, approximately 41 cases of pulmonary edema secondary to upper airway obstruction have been reported in the literature and only 15 were adults (Table 2). The largest single series in any age group to date is that of Sofer et al.,[32] who described six children with pulmonary edema after relief of upper airway obstruction by intubation. These cases were gathered over a 2 year period. In adults, the three cases of Oswalt et al. remain the largest series of reported cases from a single institution. Our eight cases over a 24 month period suggest that this phenomenon is much more common than previously recognized, and is in agreement with Tami et al.[36] A heightened awareness of the problem should benefit our ability to treat these patients.

Croup, epiglottitis, or laryngospasm account for over 85% of obstructive events in the pediatric age group. Children have an increased chest wall compliance that allows greater transmission of respiratory muscle force to negative intrathoracic force. This probably explains the prevalence of this problem in youngsters.

TABLE 2
Adults with upper airway obstruction, pulmonary edema

Ref.	Age/Sex	Procedure/Event
Jackson[14]	33/F	Unable to intubate, UAO after attempted intubation
Melnick[22]	48/M	Bronchoscopy for UAO, laryngospasm after self-extubation
	21/M	Septorhinoplasty, postextubation laryngospasm
Weissman[40]	46/M	s/p total hip arthroplasty, stridor, after extubation
Lorch[19]	48/M	Nasopharyngeal extubation followed by apnea
Tami[36]	45/F	Airway compromise secondary to aretynoid edema; pulmonary edema after extubation
	57/F	Supraglottic carcinoma—pulmonary edema after tracheostomy
	69/F	Laryngeal polyp—pulmonary edema after relief of obstruction
Ostwalt[28]	62/M	Supraglottic carcinoma
	26/F	Strangulation
	23/M	Interrupted hanging
Stradling[35]	35/F	Goiter-causing obstruction
Frank[7]	38/F	Esophygeal dilation, postextubation
McGonagle[31]	20/M	Nasal surgery, laryngospasm and pulmonary edema after extubation
	25/M	Submandibular gland excision, laryngospasm after extubation and subsequent pulmonary edema

In 1986, Lorch and Sahn[19] reviewed the seven reported cases of periintubational laryngospasm in the literature at that time, and added one case from their own practice. They postulated that patients with sleep apnea symptoms, sleep apnea body habitus, or obesity are at increased risk for airway complications upon extubation. We would agree with this. However, we feel our cases highlight a second group of adults susceptible to this complication after extubation. All adult patients in our series were young, healthy, athletic individuals. Although not proven, we believe that these body types are capable of generating very large negative intrathoracic pressures, and therefore, large hydrostatic pressure gradients with subsequent pulmonary edema formation. We currently are attempting to quantify negative inspiratory force generated in individuals with this body types in a separate investigation.

Olsson and Hallen[27] reviewed (by computer) 136,929 patients who underwent anesthesia. They noted an overall incidence of laryngospasm alone of 8.7/1000 patients receiving general anesthesia. Additionally, they found a diurnal variation with more laryngospasm occurring later in the day. They postulated anesthetist fatigue as a possible contributor. Indeed, five of eight patients in our series had procedures later in the day.

The clinical implications of identifying a group at risk for a complication are significant. In our own situation, individuals scheduled for lower extremity surgery are strongly encouraged to undergo regional versus general anesthesia. In addition, if the patient does undergo general endotracheal anesthesia by choice or because of the region operated upon, we encourage the anesthesia personnel to make sure that the patient is fully awake or deeply anesthetized prior to extubation. We believe this will minimize the possibility of laryngospasm,[1,23] and possibly secondary pulmonary edema. Other measures, of course, help to minimize this risk, including adequate pharyngeal suctioning,[23] intravenous lidocaine[9] (although this is controversial[18]), and possibly scheduling these cases early in the day. There can be a delay in diagnosis as demonstrated by Patient 1. Should symptoms of laryngospasm occur without pulmonary edema in an individual in this "at risk" group, we feel they should be monitored carefully for an extended time in the recovery room. Orthopaedic staff and residents are now well aware of the risk potential involved in these situations and closely monitor anesthetic decision-making and postoperative recovery.

Just as the literature to date grossly underestimates the incidence of this complication, its treatment has not been well outlined. Certainly, relief from the obstructive event is the first step. Paralysis, followed by assisted ventilation with a mask or endotracheal tube is often necessary. In those patients with persistent desaturation, intubation with mechanical ventilation including a positive end expiratory pressure of 5 to 10 cm H_2O is recommended. Supplemental oxygen is also a critical part of treatment to prevent persistent hypoxia and the related problems. In milder cases, oxygen may be the only therapy needed. The clinical role of diuretics is less controversial. Tami et al.[36] point out that diuresis in the reviewed cases has been successful, but does not necessarily recommend this. Five of eight adult patients received furosemide, among other treatment modalities, and improved. In the series of Lorch and Sahn, four of seven postlaryngospasm patients received diuretic therapy as well. One could argue that, by systemic diuresis, a more favorable hydrostatic balance is established in all capillary beds, including pulmonary vasculature. The indications for diuresis remain unclear, however. Since hypoxia is the chief problem associated with this complication, adequate oxygen saturation remains the primary goal of treatment.

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