Negative-Pressure Pulmonary Edema After Routine Septorhinoplasty

Discussion of Pathophysiology, Treatment, and Prevention

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Objective: To provide rhinologic surgeons with an understanding of acute negative-pressure pulmonary edema (NPPE) and its treatment.

Design: Case report and literature review of all published adult cases of NPPE. Patient factors, anesthetic variables, and outcomes are assessed.

Results: A total of 146 cases in 45 case reports and series were compiled. There was approximately a 2:1 male-female patient ratio. The average age of the patients was 33 years. Fifty percent of patients had surgery on the upper aerodigestive tract, and 8% had intranasal surgery. No patients received laryngotracheal anesthesia, and 5 of the 146 received intravenous lidocaine prior to extubation. One patient had NPPE following laryngeal mask airway treatment, and 2 patients experienced this complication after conversion from monitored anesthesia care to general endotracheal anesthesia; 33.5% of patients were treated with continuous positive airway pressure alone, while 66.5% required intubation and mechanical ventilation. The average time to resolution was 11.75 hours. Three patients died.

Conclusions: It is known that surgical procedures involving the upper aerodigestive tract have a higher risk of NPPE than other procedures. Rapid diagnosis and treatment is necessary to achieve early resolution and avoid significant patient morbidity. A thorough understanding is integral to the practice of nasal and paranasal sinus surgery, especially with the rising use of outpatient and office-based surgical suites. Therefore, we present a review of pathophysiologic mechanisms, possible risk factors, treatment options, and potential steps that can be taken to minimize this potentially devastating complication of general anesthesia.

Arch Facial Plast Surg. 2006;8:8-15

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NEGATIVE-PRESSURE PULmonary edema (NPPE) caused by acute upper airway obstruction was first clinically recognized by Lee and Downes[1] in 1983. While fewer than 150 adult cases have been cited in the world literature, independent studies demonstrate a 0.05% to 1% incidence of laryngospasm and an 11% rate of NPPE in those patients requiring active intervention for acute upper airway obstruction.[2-4] These numbers suggest that this complication occurs with greater frequency than it would appear from case reports alone.

Further investigation over the 2 decades since Lee and Downes[1] published their study has yielded a greater understanding of the pathogenesis and treatment of NPPE. Two subtypes exist, differentiated by their triggering events: type 1 results from acute upper airway obstruction, while type 2 occurs after relief of a chronic obstructive process. Both ultimately result in a patient generating a sudden high negative intrathoracic pressure, which causes fluid exudate to enter the lungs. For the purposes of this report, we will focus on type 1 NPPE because it is more relevant than type 2 to physicians performing rhinologic surgery.

Although strangulation, laryngeal trauma, epiglottitis, croup, hematoma, foreign-body aspiration, hiccups, and goiter have been reported to precipitate type 1 NPPE, laryngospasm remains the most common cause, accounting for approximately 50% in reported series.[1,5,6] It is also well recognized that surgical procedures involving the upper aerodigestive tract are more likely than other procedures to cause laryngospasm, which creates higher risk for patients undergoing upper gastrointestinal tract endoscopy and general otolaryngologic, nasal, and paranasal sinus

surgery. Therefore, some anesthesiologists recommend spraying topical anesthesia on the larynx just prior to intubation in these high-risk groups, while others prefer administering intravenous (IV) lidocaine prior to extubation. The practice of topical anesthetic application to the larynx is otherwise known as laryngotracheal anesthesia (LTA).

Negative-pressure pulmonary edema has not been reported following monitored anesthesia care (MAC) alone. It is believed that awake patients are less likely to maintain a closed glottis after a laryngospastic event and that a heightened laryngeal reflex response may be present when general anesthesia is used. However, several reports exist of patients receiving MAC who required laryngeal mask airway (LMA) or endotracheal intubation for airway protection and then subsequently developed NPPE.[7,8] Therefore, all patients undergoing surgical manipulation of the upper airway are potentially at risk for this complication.

Herein, we include an illustrative case report and a brief literature review to highlight several factors that place the patient undergoing septorhinoplasty at particular risk for NPPE. The increasing number of cosmetic procedures being performed in private office–based surgical suites makes it essential that both otolaryngologic and plastic surgeons be aware of predictive factors, signs and symptoms, and treatment options for NPPE because prompt recognition and treatment is integral to resolution.

REPORT OF A CASE

A 28-year-old healthy black man underwent an uncomplicated septorhinoplasty procedure. Following administration of 250 mg of sodium pentothal, he was easily intubated with a 7.5-mm oral endotracheal tube. Anesthesia was maintained using enflurane and rocuronium bromide followed by fentanyl for analgesia. At the onset of the procedure, prior to nasal injection, a throat pack was placed in standard fashion.

The patient underwent an uneventful septorhinoplasty lasting less than 2 hours. Prior to extubation, he was given IV lidocaine and extubated easily without bucking or coughing. After starting 40% oxygen by face mask, the patient showed blood oxygen saturation at 100% and was brought to the recovery room. On arrival (approximately 8 minutes after extubation), he became agitated, dyspneic, and stridorous. Pink frothy secretions were noted. Respiratory distress ensued, and pulse-oximetry showed a 68% blood oxygen saturation. An oral airway was placed, and the patient was ventilated with a bag-valve mask but continued to be hypoxic. After paralysis was induced with succinylcholine, he was reintubated easily, and mechanical ventilation was begun. Rales were present bilaterally, and chest radiography showed diffuse bilateral pulmonary edema.

The patient was transferred to the surgical intensive care unit and continued receiving mechanical ventilation with a fraction of inspired oxygen of 50%. Arterial blood gas values at that time demonstrated respiratory acidosis. A Foley catheter was placed, and 20 mg of IV furosemide was given. Approximately 12 hours later, the pulmonary edema had completely resolved, both radiographically and clinically. The patient was slowly weaned off the ventilator and extubated 18 hours after the initial event. Breathing room air, he showed normal arterial blood gas values. He was then transferred to the general otolaryngology care floor, observed overnight, and discharged home early on postoperative day 2 without adverse sequelae.

METHODS

A MEDLINE search was performed from the present day back to 1970, anteceding the first report by Lee and Downes,[1] to collect all publications of case reports and studies related to NPPE. Pediatric patients were excluded because they do not accurately represent the population of individuals seeking rhinoplasty and likely have different pathophysiologic mechanisms than adults. Cases of NPPE from nonsurgical causes (eg, attempted hanging, goiter, or epiglottitis) and those cases secondary to endotracheal tube biting were also excluded. The articles were then evaluated, and references contained in those documents that were not found on the initial search were compiled. After exhaustive searching, 47 publications discussing adult incidents were included in this review, 43 directly related to perioperative events and 4 nonoperative cases (**Table**).

These articles were assessed for the mention of LTA, IV lidocaine use, conversion from MAC to general anesthesia, or the use of LMA. Case reports that did not specify their use were counted as negative. The total number and type of cases were noted, as were the number of otolaryngologic or head and neck procedures. These were then further subdivided into nasal cases. The number of men and women was tabulated. Adults were defined as those 15 years or older, and their ages were compiled and averaged.

Postoperative interventions and the time to resolution, when reported, were noted. The number of patients successfully treated with either supplemental oxygen or continuous positive airway pressure was recorded. The number of deaths due to NPPE was compiled.

RESULTS

A total of 146 cases of adult NPPE were compiled from 43 case reports or series (Table). There were no prospective studies found in this review. No patients had been treated with LTA prior to intubation and 5 (3%) were treated with IV lidocaine immediately before extubation. One patient (0.6%) experienced NPPE after receiving general anesthesia through an LMA. No cases of NPPE following MAC alone were identified, but 2 cases (1.3%) who required conversion to general anesthesia were found. Seventy-four cases (50.6%) involved upper aerodigestive tract or deep neck structures, and 12 (8.2%) involved only the nose (septoplasty and/or rhinoplasty). The male-female ratio was 71:31, with several reports not giving sex-specific data.

The number of hours intubated after NPPE ranged from 1 to 336, averaging 11.75 hours overall. Forty-nine

Table. List of Studies Reviewed*

Source	Patients (Age, y)	LTA	IV Lidocaine	LMA	ENT–H&N	Nasal Surgery	Procedure(s) or Cause of Abnormality	Men	Women	Mean Intubation Time, h	CPAP or Oxygen	Death	Notes
Oswalt et al,[9] 1977	3 (62, 26, and 23)	0	0	0	1	0	Larynx cancer, strangulation, hanging	2	1	48	1	NR	1 CPAP
Rivera et al,[10] 1979	1 (40)	0	0	NR	1	0	Tracheotomy for airway obstruction: epiglottitis	1	0	72	0	NR	NR
Jackson et al,[11] 1980	1 (33)	0	0	0	0	0	Tubal ligation	1	0	0	1	NR	Failed intubation trigger, 1 CPAP management
Cass,[12] 1981	1 (15)	0	0	NR	1	1	"Nasal"	1	0	16	0	NR	NR
Dash et al,[13] 1983	1 (30)	0	0	0	1	0	Neck trauma, tracheotomy, neck exploration	1	0	8	NR	NR	NR
McGonagle and Kennedy,[14] 1984	2 (20 and 25)	0	1	0	2	1	"Nasal," submandibular gland excision	2	0	21	0	NR	NR
Weissman et al,[15] 1984	1 (46)	0	0	0	0	0	Hip arthroscopy	1	0	24	0	NR	NR
Melnick,[16] 1984	2 (21 and 48)	0	0	0	2	1	Laryngoscopy, septorhinoplasty	2	0	12	1	NR	1 CPAP only
Jenkins,[17] 1984	1 (60)	0	0	0	1	0	Direct laryngoscopy (larynx carcinoma)	0	1	24	0	NR	NR
Kamal and Agha,[18] 1984	1 (15)	0	0	NR	1	0	NR	0	1	NR	1	NR	NR
Glasser and Siler,[7] 1985	1 (30)	0	0	0	0	0	Childbirth	0	1	36	0	NR	MAC first
Lorch and Sahn,[19] 1986	1 (48)	0	0	0	1	1	Panendoscopy	1	0	NR	1	NR	NR
Frank and Schreiber,[20] 1986	1 (38)	0	0	0	1	0	Esophageal dilation	0	1	24	0	NR	NR
Tami et al,[21] 1986	3 (45, 57, and 69)	0	0	0	2	0	Rheumatoid arthritis, supraglottic cancer, laryngeal polyp	0	3	54	0	NR	NR
Randour et al,[22] 1986	1 (79)	0	0	0	1	0	Direct laryngoscopy	1	0	48	0	NR	Swan-Ganz: normal at 1 h
Price and Hecker,[23] 1987	1 (27)	0	0	0	1	0	Cervical node biopsy	0	1	24	0	NR	NR
Lagler and Russi,[24] 1987	1 (35)	0	0	0	0	0	Intubation for respiratory distress from goiter	0	1	24	0	NR	NR
Willms and Shure,[25] 1988	1 (28)	0	0	0	0	0	Repair of ankle fracture	1	0	0	0	NR	NR
Andersen et al,[8] 1988	1 (21)	0	0	NR	0	0	Dilation and evacuation	0	1	6	0	NR	MAC first
Miro et al,[26] 1989	1 (71)	0	0	0	0	0	Laser excision of lower tracheal lesion	0	1	24	0	NR	NR
Holmes et al,[27] 1991	7 (18, 20, 22, 24, 24, 25, and 32)	0	1	0	1	1	Sphincterotomy, anterior cruciate ligament repair ×2, medial cruciate ligament repair, Achilles tendon repair, shoulder hardware removal, hand debridement, septorhinoplasty	7	0	24.7	4	0	NR
Soldano et al,[28] 1993	3 (19, 23, and 24)	0	0	0	1	0	Appendectomy, hernia, submental node excision	3	0	27.6	0	NR	NR
Cascade et al,[29] 1993	8 (19, 19, 23, 25, 25, 44, 45, and 55)	0	1	0	2	1	Osteotomy, tendon repair, hernia ×2, spine surgery, ovarian cyst, septorhinoplasty	6	2	0	6	NR	NR
Halow and Ford,[30] 1993	1 (34)	0	0	0	0	0	Appendectomy	1	0	1	0	NR	NR

(*continued*)

Table. List of Studies Reviewed* (cont)

Source	Patients (Age, y)	LTA	IV Lidocaine	LMA	ENT–H&N Surgery	Nasal Surgery	Procedure(s) or Cause of Abnormality	Men	Women	Mean Intubation Time, h	CPAP or Oxygen	Death	Notes
Maroof et al,[31] 1994	12 (NR)	0	0	0	9	4	Septoplasty ×2, pilonidal cyst, eyelid cyst, hernia ×2, septorhinoplasty, tympanoplasty, tongue lesion excision, oro-antral fistula repair, keratotomy	NR	NR	NR	NR	NR	NR
Hsu et al,[32] 1995	3 (26, 27, and 48)	0	1	0	2	0	Microlaryngoscopy, appendectomy, thyroidectomy	1	2	10	2	1	Mortality from gastrointestinal tract bleeding and leukemia complications
Guffin et al,[5] 1995	9 (16, 19, 21, 31, 41, 55, 63, 63, and 73)	0	0	0	7	1	Tonsillectomy ×2, ankle fracture, septoplasty, hernia, direct laryngoscopy ×3, tympanoplasty	7	2	0	NR	NR	No intubation time given
Koch et al,[33] 1996	1 (42)	0	1	0	0	0	Spinal fusion	1	0	2	0	NR	Bronchoscopy done; pinpoint hemorrhages seen
Ingrams et al,[34] 1997	1 (NR)	NR	0	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR
Goldenberg et al,[4] 1997	6 (22, 23, 28, 32, 32, and 34)	0	0	0	6	0	No data	5	1	1	5	1	Abnormal echocardio-gram in 50%
Scarbrough et al,[35] 1997	3 (21, 28, and 40)	0	0	0	3	0	Mandible fracture repair, palatal expansion, temperomandibular joint arthroscopy	1	2	3	2	NR	NR
Deepika et al,[3] 1997	30 (NR)	0	0	0	20	0	No data	NR	NR	NR	NR	NR	Incidence, 0.094%
Schwartz et al,[36] 1999	1 (46)	0	0	0	0	0	Cataract	1	0	48	0	NR	NR
Lathan et al,[37] 1999	7 (31, 38, 39, 39, 48, 55, and 66)	0	0	0	1	0	Dilation and evacuation, breast reconstruction, breast biopsy, anterior cruciate ligament repair, hysterectomy, middle ear exploration, incision and drainage of leg abscess	3	4	0	7	0	NR
Langan and Michaels,[38] 1999	1 (25)	0	0	0	0	0	Shoulder arthroscopy	1	0	5	NR	0	4-h Delay preintubation, bit on tube in OR
Sullivan,[39] 1999	1 (21)	0	0	1	0	0	Femoral nail removal	1	0	0	1	NR	NR
McConkey,[40] 2000	6 (15, 20, 21, 26, 27, and 36)	0	0	0	1	0	Anoscopy, herniorrhaphy, appendectomy, tibia repair, dental incision and drainage, skin excision	6	0	1	5	NR	5 CPAP, 1 oxygen; incidence, 0.05%
Patton and Baker,[41] 2000	14 (mean, 28)	0	0	0	1	0	All orthopedic, 1 anterior cervical approach	11	3	0	11	0	NR
Dolinski et al,[42] 2000	1 (33)	0	0	0	1	1	Septoplasty	0	1	144	0	NR	Incidence, 0.1%
Gupta et al,[43] 2001	1 (70)	0	0	0	1	0	Bronchoscopy	0	1	0	0	1	NR
Broccard et al,[44] 2001	1 (21)	0	0	0	0	0	Spinal nerve graft	1	0	0	1	NR	Bronchial lavage done; acute hemorrhage seen

(*continued*)

Table. List of Studies Reviewed* (cont)

Source	Patients (Age, y)	LTA	IV Lidocaine	LMA	ENT–H&N	Nasal Surgery	Procedure(s) or Cause of Abnormality	Men	Women	Mean Intubation Time, h	CPAP or Oxygen	Death	Notes
Sharma,[45] 2002	1 (60)	0	0	NR	1	0	Thyroidectomy	0	1	72	0	NR	NR
Lloyd and Kamisetty,[46] 2003	1 (22)	0	0	0	1	0	Mandible repair	1	0	336	0	NR	NR
Total	**146** (mean, 33.2)	**0**	**5**	**1**	**74**	**12**	NA	**71**	**31**	**11.75**	**49**	**3**	NR
							Nonsurgical Examples						
Eid et al,[47] 1997	1 (NR)	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	ETT obstruction
Batra et al,[48] 1984	1 (19)	0	0	0	1	0	Hanging	1	0	72	NR	NR	Attempted suicide
Leatherman and Schwartz,[49] 1983	1 (70)	0	NR	NR	0	0	Paratracheal mass with examination on admission	0	1	NA	NR	NR	Respiratory distress on admission
Stradling and Bolton,[50] 1982	1 (35)	0	0	0	1	0	Obstructing goiter	0	1	NR	NR	NR	NA, present on admission

Abbreviations: CPAP, continuous positive airway pressure; ENT–H&N, otlaryngologic–head and neck surgery; ETT, endotracheal tube; IV, intravenous; LMA, laryngeal mask airway; LTA, laryngotracheal anesthesia; MAC, monitored anesthesia care; NA, not applicable; NR, not reported; OR, operating room.

*Unless otherwise indicated, data are given as the number of relevant cases or procedures.

(33.5%) of the 146 patients were successfully treated without intubation. Three mortalities (2%) occurred.

COMMENT

PATHOPHYSIOLOGIC CHARACTERISTICS

Negative-pressure pulmonary edema secondary to postextubation laryngospasm begins with glottic closure from an unknown stimulus and ends with accumulation of transudate fluid within the interstitial and alveolar spaces of the lungs. The series of physiologic steps that bridges these 2 events can be understood in terms of the causes of laryngospasm and the resulting alterations in transmembrane Starling forces that occur in the pulmonary microvasculature and alveoli. By understanding this process, it is possible to identify those patients at risk for NPPE and to take steps to minimize its occurrence.

The pathophysiologic characteristics of types 1 and 2 NPPE have been defined.[5] Type 1 involves acute glottic closure as a precipitating event. After laryngospasm, the patient continues to inspire against a closed glottis, performing a modified Mueller maneuver. This generates a high negative intrathoracic pressure increasing venous return to the right heart and pulmonary arteries. This volume expansion causes high arteriole and capillary fluid pressures that favor transudation into the alveolar space. It has been hypothesized that athletic men are capable of generating higher intrathoracic pressures and maintaining apnea for longer periods of time than other patients. They are, therefore, at increased risk of NPPE.[51] The greater than than 2:1 ratio of men to women in our review supports this hypothesis.

Further imbalance of the Starling forces results from hypoxic vasoconstriction of both pulmonary and systemic arterioles, raising systemic blood pressure and increasing the afterload on the left and right ventricles. Catecholamine release simultaneously occurs and further increases total peripheral resistance. The result is essentially the trapping of increased fluid volume within the thoracic cavity with unbalanced transmembrane pressures in the alveoli.

On expiration against a closed system, a Valsalva maneuver generates high intraluminal airway pressures. This serves as an "auto-PEEP" (positive end–expiratory pressure) mechanism, where positive intraluminal pressure prevents fluid from leaving the capillaries and entering the alveoli. However, when the laryngospasm is relieved naturally or by reintubation, a rapid drop in airway pressures occurs with prompt transudation of fluid into the alveoli and severe acute pulmonary edema. Many authors also believe that temporary damage to capillary membranes and supporting cells within the pulmonary interstitium results from these rapid fluid shifts.

Type 2 NPPE results from chronic extrathoracic airway obstruction with a high "set-point" for intrathoracic and airway pressure balance. Relief of the obstruction causes a series of events almost identical to type 1. Patients at risk for type 2 would include those with laryngeal cancer who undergo surgery other than total laryngectomy, individuals with obstructive sleep apnea who are undergoing correction, and patients with an obstructing goiter undergoing thyroidectomy.

LARYNGOSPASM AND UPPER AIRWAY OBSTRUCTION

Postoperative laryngospasm was reported in approximately 9 of every 1000 adult patients undergoing general anesthesia in the 1980s.[4] Other estimates have ranged from 0.05% to 0.1%.[2-4] Data related to outpatient anes-

thesia in Massachusetts compiled from the year 2000 revealed laryngospasm occurring in 1 of 345 patients undergoing general anesthesia (n=80 323) and 1 of 834 for sedation and local anesthesia combined (n=57 575). These numbers are comparable to, although slightly lower than, those in older published series and demonstrate that changes in anesthesia or instrumentation have not significantly decreased the incidence of laryngospasm over the last 30 years.

Of those patients requiring active intervention for acute airway obstruction, 11% progress to NPPE.[4] Although relatively rare, the sequelae of acute NPPE can be devastating: current studies quote a mortality rate ranging from approximately 10% to 40%.[51] Our data demonstrated a much lower rate of 2% in reported cases. While early intervention may play a key role in preventing a poor outcome, a certain proportion of those affected will not improve, despite receiving optimal therapy. In light of this, prevention may be the most important factor to consider when examining NPPE as a clinical entity.

Some potential causes of laryngospasm have been evaluated in animal models. Sasaki and Suzuki[52] and Ikari and Sasaki[53]demonstrated the glottic closure reflex due to superior laryngeal nerve (SLN) stimulation in an animal model. They found that repetitive stimulation resulted in laryngospasm that could outlast the initial stimulus by a variable period of time. Because SLN stimulation is considered to be integral in triggering this reflex, it is possible that the use of LTA could decrease the incidence of postextubation laryngospasm. Several studies in the anesthesiology literature have demonstrated a decrease in laryngospasm using topical LTA or IV lidocaine during intubation and extubation, respectively. The use of LMA has also been evaluated and found to decrease cough during the postoperative period.[54-57]

Additional studies on the English bulldog by Kirchner[58] demonstrated that a more muscular pharynx will produce higher than normal airway pressures and supraglottic collapse during inspiration. Bulldogs are known to have a high risk of pulmonary edema, presumably owing to their overall body habitus and pharyngeal anatomy. A correlation between a short muscular or redundant pharynx and NPPE may be surmised. This potentially places patients with short thick necks, long palates, and obstructive sleep apnea at an increased risk of postextubation supraglottic collapse and NPPE.

RHINOPLASTY-SPECIFIC NPPE

During rhinoplasty performed with the patient under general anesthesia, many events are capable of producing SLN stimulation. Intubation, the collection of blood, the placement and removal of throat packing, and suctioning prior to extubation all could stimulate the hypopharynx and larynx. The population seeking rhinoplasty is often characterized by younger and potentially more muscular individuals, with a recent increase in the number of male patients. The potential risk factors for NPPE are age younger than 40 years, muscular body type, male sex, short thick neck, sensitive gag reflex, underlying cardiac anomalies, Malampotty 3 (defined by the inability to visualize the uvula or oropharynx with full mouth opening) found on physical examination, history of obstructive sleep apnea, and no LTA or IV lidocaine during anesthesia.

TREATMENT

Most authors agree on some basic interventions, but there is no defined treatment strategy for NPPE. Since the problem results from a combination of alveolar pressure differentials and transudate leaking through damaged intracellular connections, providing the patient with positive pressure ventilation is critical. Some authors report treatment with supplemental oxygen (Table). Although tempting, we do not recommend this intervention because it does not address the pathophysiologic abnormalities of NPPE and may delay implementing more definitive therapies.

Treatment options include continuous positive-pressure ventilation using a face mask, LMA, or endotracheal intubation with ventilatory support. In the immediate postoperative period for a patient who underwent rhinoplasty, we do not recommend mask ventilation because the pressure on the nasal dorsum required for a seal may cause undue cosmetic sequelae. Prompt transfer to a critical care facility or intensive care unit should also be done.

Although NPPE does not result from fluid overload, most authors recommend treatment with a foley catheter and gentle diuresis using low-dose furosemide. Regarding interstitial damage, steroids may be beneficial, although their effectiveness has not been proven by evidence-based studies. Because both diuresis and short-term steroid therapy have minimal adverse effects, we recommend treatment with 20 mg of IV furosemide every 6 to 12 hours as well as 6 to 8 mg of dexamethasone every 6 hours until the NPPE resolves.

A chest radiograph should be obtained at the onset of symptoms and every 12 to 24 hours thereafter until complete resolution of radiographic findings. While most cases of NPPE completely resolve in less than 24 hours, some will take several days to weeks. Our analysis showed an average time until extubation of 11.75 hours but with a wide overall range.

PREVENTIVE MEASURES

There is no intervention proven to prevent NPPE and no prospective studies to evaluate treatment-based outcome measures. However, based on the currently accepted pathophysiologic mechanism, one can reasonably assert that laryngospasm caused by laryngeal irritation precedes NPPE. Therefore, maneuvers to prevent laryngeal stimulation or postoperative stridor from partial laryngospasm would be expected to reduce the likelihood of its occurrence.

Since stimulation of the larynx is an inciting event in this process, limiting this is critical in prevention. The placement of a throat pack prior to injections as well as careful oropharyngeal suctioning prior to pack removal helps to prevent blood and other material from contacting and irritating the larynx. However, even with extreme care, small amounts of material can pool near the posterior glottis prior to extubation.

The anesthesiologist plays an integral part in this process. Smooth intubation, limiting aggressive suctioning, and the use of either IV lidocaine or topical LTA all play a role. As shown by Sasaki and Suzuki,[32] a single stimulus to the SLN will not produce laryngospasm, but repetitive events will. This laryngeal response can persist for a variable period of time. It is therefore likely that multiple stimuli at either intubation or extubation cause laryngospasm and its possible sequelae.

The findings of our literature review support these proposals. The greater than 50% prevalence of procedures involving the upper aerodigestive tract or deep cervical structures lends support to the concept that tactile stimuli cause irritation and NPPE. In addition, the percentage of nasal surgery in the study group is higher than what would be expected in a random sampling of cases.

Specific conclusions about anesthetic or operative techniques cannot be drawn because not all of the case reports had enough detail for consistent data. A regression analysis would be required to highlight specific interventions or medications that were risk factors for NPPE in this population. However, it is highly suggestive that no patients received LTA and only 5 of the 146 patients were treated with IV lidocaine. National data on the use of these agents cannot be obtained, but one can assume that the routine use of both LTA or IV lidocaine by anesthesiologists is higher than that seen in the present cohort. It cannot be stated that the use of the agents would have prevented NPPE, but the incidence might have been reduced.

Studies have demonstrated that LTA and IV lidocaine are equally effective in preventing laryngospasm, both in animal models and after tonsillectomy in humans.[44] Placement of LTA prior to intubation prevents triggering mechanisms at the onset of the case. Since most simple rhinoplasty procedures take approximately 2 hours, agents lasting less than this will not have an effect during extubation. For this reason, a mixture of 1% lidocaine and 2% tetracaine (2 mL each) is recommended. This provides both rapid onset and a duration greater than 2 hours. Intravenous lidocaine does not prevent stimulation at the time of intubation and can cause a depressed mental status in the early postextubation period. Provided that there is no contraindication, we recommend the use of LTA rather than IV lidocaine prior to intubation.

Contraindications to LTA may include the need for a rapid sequence induction of anesthesia in a patient at risk for regurgitation and pulmonary aspiration of gastric contents. These would include patients with symptoms of gastroesophageal reflux disease, hiatal hernia, or delayed gastric emptying secondary to systemic disease or obstruction. In addition, the use of IV lidocaine prior to emergence from anesthesia could be questioned in this same group of patients at risk for aspiration at the conclusion of the procedure.

Additional measures that may reduce laryngospasm include extubation in a light (stage 1) plane of anesthesia. It is generally accepted that there are increased rates of laryngospasm when extubation occurs during stage 2. Although bucking and coughing is a concern with light-plane extubation, the routine use of LTA may help to prevent this. Maintaining the patient with IV anesthesia (remifentanyl and propofol supplemented with 70% nitrous oxide) can also be used in conjunction with LTA. This combination of agents may allow a lighter anesthetic plane, quicker emergence at the end of the procedure, and tolerance of the endotracheal tube in a stage-1 plane of anesthesia. In total, this regimen may help to prevent a stormy emergence from anesthesia.

In summary, identifying patients at risk and coordinating with the anesthesiologist to address these concerns will help to avoid many complications, including NPPE. Physician awareness and education will help to facilitate early recognition and intervention. This may help to avoid the need for reintubation and/or shorten the time to resolution, thus improving safety for patients who undergo outpatient rhinoplasty procedures.

Accepted for Publication: March 1, 2005.
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Announcement

Calendar of Events: A New Web Feature

On the new Calendar of Events site, available at http://pubs.ama-assn.org/cgi/calendarcontent and linked off the home page of the *Archives of Facial Plastic Surgery*, individuals can now submit meetings to be listed. Just go to http://pubs.ama-assn.org/cgi/cal-submit/ (also linked off the Calendar of Events home page). The meetings are reviewed internally for suitability prior to posting. This feature also includes a search function that allows searching by journal as well as by date and/or location. Meetings that have already taken place are removed automatically.