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IDENTIFYING RISK FACTORS FOR BRONCHOSPASMS IN THE PEDIATRIC PATIENT

By

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Bachelor of Science in Nursing, University of North Dakota, 2012

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Date______________________
Title: Identifying Risk Factors for Bronchospasms in the Pediatric Patient

Background: A 7 month old male patient undergoing hypospadias repair experienced a severe bronchospasm upon emergence from general anesthesia. The patient was treated with albuterol and epinephrine along with chest compressions. Pediatric patients can have several risk factors that place them at a higher risk for perioperative adverse respiratory events. Due to their limited oxygen reserve and increased oxygen demand, the pediatric population does not tolerate these adverse respiratory events. Identifying risk factors for perioperative bronchospasms along with being adequately prepared can help prevent life-threatening complications.

Purpose: To identify risk factors for perioperative bronchospasms and evaluate treatment options

Process: A systematic search was conducted utilizing CINAHL and PubMed for research articles that were published in the last 8 years that pertained to identifying risk factors for bronchospasms in the pediatric patient along with treatment options.

Results: Several risk factors exist that place pediatric patients at risk for perioperative adverse respiratory events. These risk factors include but are not limited to the following: recent upper respiratory tract infection, asthma or a family history of asthma, exposure to second-hand smoke, prematurity, and low birth weight. Patients with multiple risk factors could potentially benefit from a pretreatment of albuterol. Bronchospasm treatment varies depending on the severity of the spasm and can include inhaled albuterol, volatile agents, epinephrine, or steroids.

Implications: Informed decision-making should be utilized on a case-by-case basis in regards to identifying and treating bronchospasms in the pediatric population.

Keywords: pediatrics, bronchospasm, smoking, asthma, upper respiratory tract infection, albuterol, epinephrine
Background

“Three quarters of all critical incidents and a third of all perioperative cardiac arrests in pediatric anesthesia are caused by adverse respiratory events (von Ungern-Sternberg et al., 2015, pg. 440).” It is not uncommon for children to present for surgery with signs and symptoms of an upper respiratory tract infection, such as a cough, runny nose, or congestion. Anesthesia practitioners understand that children who have an active upper respiratory tract infection are at a greater risk for perioperative complications, such as a laryngospasm and bronchospasm.

Unfortunately, there are children who present for surgery and appear perfectly healthy and asymptomatic but still possess significant risk factors for respiratory complications. It is these ‘hidden’ or not-so-well-known risk factors that anesthesia practitioners need to be equally aware of and screen for preoperatively in order to anticipate the possible complications that could arise.

Considered an anesthetic emergency, bronchospasms can lead to life-threatening respiratory and cardiac sequelae especially in the pediatric patient because they possess a limited oxygen reserve and consume oxygen at an increased rate as compared to adult patients. Risk factors for developing a perioperative bronchospasm include organic heart disease, higher ASA classification, recent upper respiratory tract infection, prematurity, and low birth weight (Regli & von Ungern-Sternberg, 2014). According to Stratelak (1990), children age 0 to 9 years had a greater frequency of bronchospasm with the highest incidence occurring in children 0 to 3 months old. Becke (2012) claimed that the incidence of perioperative adverse respiratory events is increased in all children less than six years old, but particularly in those younger than one year old. Additional risk factors include a history of asthma, previous or current eczema, a family history of eczema, asthma or hay fever in two or more relatives, wheezing occurring more than three times during the previous 12 months, and exposure to passive smoking (von Ungern-
Sternberg et al., 2015). The incidence of perioperative bronchospasm in children without asthma is 0.2-4.1% and in children with asthma, the incidence increases to 2.2-5.7% (Regli & von Ungern-Sternberg, 2014). As anesthesia practitioners, it is important to remember that regardless of the patient’s medical history, severe bronchospasms can still occur despite the presence or absence of risk factors. However, awareness and identification of these risk factors along with pretreatment when applicable could result in fewer perioperative adverse respiratory events.

**Case Report**

A 7 month old, 66 cm, 8.6 kg male patient with no prior surgical history presented for a hypospadias repair. Medical history included hypospadias, cephalohematoma at birth, umbilical hernia, and born preterm at 35 weeks. Patient had no known drug allergies. Patient was not taking any medications. No labs were obtained and the airway classification was unable to be determined prior to the procedure due to the patient’s age and cooperativeness. The patient was classified as an ASA 1.

The patient did not receive any medication in the preoperative period. Upon arriving to the operating room, standard monitors were applied. An inhalational induction was utilized with 70% nitrous oxide, 30% oxygen, and sevoflurane at 8%. After the patient was asleep, nitrous oxide was discontinued and sevoflurane was reduced to 4-5%. An IV was inserted at this time. The patient was a very difficult IV start due to excess subcutaneous tissue. Fentanyl 10 micrograms and propofol 10 milligrams was administered intravenously before intubation. A Mac 1 blade was utilized along with a 3.5 cuffed endotracheal tube. A grade 1 view of the vocal cords was easily obtained and the endotracheal tube was passed through the cords via one attempt and secured at the lips at 11 centimeters. Endotracheal tube placement was verified by end-tidal carbon dioxide, bilateral breath sounds, bilateral chest rise, and tube condensation. A
leak test was performed and a leak was identified at 16 cm H2O. The eyes were taped and sevoflurane with oxygen/air mixture was used to maintain the anesthetic. Expiratory sevoflurane concentration ranged from 2.5-3.5% throughout the case. Mechanical ventilation was utilized using pressure control ventilation. The peak inspiratory pressures were adjusted to receive a tidal volume of 50-70 milliliters and the respiratory rate was set at around 32 breaths per minute. The patient’s tidal volumes and respiratory rate were adjusted to maintain normocapnia. Before the procedure began, ondansetron 1 milligram and cefazolin 245 milligrams was administered intravenously. A penile block was performed by the surgeon at the start of the case. No intraoperative issues were encountered. The patient received 250 milliliters of Lactated Ringer’s and required no additional narcotics.

Upon completion of the procedure, the inhalational agent was discontinued and the patient was placed on 100% FiO2. Within seconds after discontinuing the inhalational agent, the patient’s oxygen saturations decreased from 100% to 75%. Circumoral cyanosis was present and a generalized pale color was noted. The patient was unable to be manually ventilated. Anesthesia practitioners determined that the patient was experiencing a bronchospasm due to the rapid decline in oxygen saturations. Epinephrine 10 micrograms IV push was given and the patient was manually ventilated via the endotracheal tube. Oxygen saturations increased to around 94%. Approximately two minutes later, the patient again experienced a bronchospasm. The endotracheal tube was still in place at this time. Oxygen saturations quickly dropped to around 20% despite treatment with epinephrine. Anesthesia practitioners were unable to obtain an end-tidal carbon dioxide tracing and were unable to manually ventilate the patient. A code blue was called, no pulse palpable, and chest compressions were initiated. Throughout the code which lasted approximately 90 seconds, the patient received six boluses of epinephrine IV push (10
micrograms per bolus) and a single albuterol treatment. The endotracheal tube was also removed during the code because it was questioned whether the tube had become dislodged, a mucus plug present, or the endotracheal tube itself contributing to the bronchospasm. After 90 seconds, the patient was able to be manually ventilated, a pulse was palpable, and the patient was maintaining oxygen saturations around 92%. The patient was kept in the OR for approximately 30 minutes after the event in order to evaluate and treat the patient’s condition.

Once the patient was awake and able to maintain his own airway and oxygen saturations were stable, the patient was transferred to the PACU. The patient had no further respiratory issues. He was then admitted to the PICU where he was given 4 milligrams of decadron for intermittent stridor and started on ceftriaxone. It was then noted that the patient had been treated two weeks prior for bilateral otitis media that was unresolved after a week of treatment. At this point, he was started on amoxicillin. It was also discovered that both parents were smokers. A chest x-ray was obtained that showed moderate, bilateral bronchial wall thickening, indicating inflammation in the airways. After treatment with IV antibiotics and decadron and observation in the PICU for 24 hours, the patient was discharged from the hospital the following day without any complications.

Discussion

Bronchospasm

“The pathophysiology of bronchospasms is complex and involves airway smooth muscle contraction, airway edema, and an increase in airway secretions (McCall, 1992, pg. 202).” The concentrations of two chemical compounds, cyclic adenosine 3’5’-monophosphate (cAMP) and cyclic guanosine 3’5’-monophosphate (cGMP), regulate airway smooth muscle tone (McCall,
As the concentration of cAMP increases, smooth muscle tone decreases and bronchodilation occurs (McCall, 1992). As the concentration of cGMP increases, smooth muscle tone increases and bronchoconstriction develops (McCall, 1992). It is believed that the muscle tone is altered because cAMP and cGMP modify the amount of calcium available for muscle fiber contraction, influencing mediator release from sensitized mast cells or by hyper or hypopolarizing the cell, which alters the cell’s responsiveness to stimuli (Stratelak, 1990).

Production of cAMP and cGMP are regulated by input from the branches of the autonomic nervous system, which are the sympathetic and parasympathetic branches (Grossman & Porth, 2014). When the sympathetic nervous system is stimulated, catecholamines are released causing either excitation or inhibition of smooth muscle contraction depending on the site, dose, and type of receptor present (Grossman & Porth, 2014). Norepinephrine has potent excitatory activity and low inhibitory activity (Grossman & Porth, 2014). Epinephrine has potent excitatory and inhibitory activity (Grossman & Porth, 2014). In vascular smooth muscle, when the alpha receptors are stimulated, vasoconstriction occurs. On the other hand, when beta receptors are stimulated, vasodilation occurs (Grossman & Porth, 2014).

When alpha adrenergic receptors are stimulated by norepinephrine, cAMP production is altered resulting in an excess of cGMP leading to bronchoconstriction (Stratelak, 1990). This exaggerated alpha receptor response may be associated with the bronchospasm associated with asthma; however, this mechanism is least involved when dealing with perioperative bronchospasms (Stratelak, 1990). When beta 2 adrenergic receptors in the lungs are stimulated by epinephrine, there is a conversion of adenosine triphosphate to cAMP (Stratelak, 1990). As a result, the concentration of cAMP increases and bronchodilation develops. Certain medications, such as some beta-blockers, can prevent this beta 2 receptor stimulation and result in a
bronchospasm. There is also an enzyme known as phosphodiesterase that can inactivate cAMP (Grossman & Porth, 2014). If cAMP is inactivated, the concentration of cGMP increases and a bronchospasm can develop (Grossman & Porth, 2014).

As described previously, norepinephrine and epinephrine both have a role in altering cAMP and producing either bronchoconstriction or bronchodilation. However, these are not the primary mechanisms leading to bronchospasms. During anesthesia, it is believed that the primary mechanism of bronchospasm involves parasympathetic nervous system stimulation (Stratelak, 1990). When pulmonary stretch receptors, irritant receptors, or interstitial juxtapulmonary-capillary receptors are stimulated, activation of the parasympathetic nervous system occurs (Stratelak, 1990). These three receptors communicate with the vagus nerve resulting in release of acetylcholine at the post synaptic muscarinic receptors in the bronchial walls of the central airway (Stratelak, 1990). Stimulation of these muscarinic receptors with acetylcholine produces airway narrowing, edema, and mucus production (Stratelak, 1990).

The irritant receptors, which are located beneath the tight junctions of the bronchiolar epithelium, can be activated by a variety of stimuli such as mechanical stimulation associated with surgical procedures, intubation, secretions, and aspiration of gastric contents or blood (Stratelak, 1990). These receptors can also be stimulated by exposure to anesthetic gases (Stratelak, 1990). When patients have an upper respiratory tract infection, morphological changes in the respiratory epithelium occur which allow for greater penetration of anesthetic vapors which activate the irritant receptors and can cause bronchospasms (Stratelak, 1990).

Other mechanisms by which bronchospasms can occur include biochemical mediators which are released from mast cells located throughout the lungs (Stratelak, 1990). Examples of these biochemical mediators include histamine, leukotrienes, platelet activating factor,
eosinophil, and neutrophil chemotactants (Stratelak, 1990). These mediators increase smooth muscle tone by directly acting on the smooth muscle or through stimulation of the parasympathetic nervous system (Stratelak, 1990).

**Risk Factors**

“Despite the development of guidelines for anesthesia management, perioperative adverse respiratory events remain one of the major causes of morbidity and mortality during pediatric anesthesia (von Ungern-Sternberg et al., 2010, pg. 773).” These respiratory events can precipitate hypoxemia and lead to life-threatening events especially in children who have a limited oxygen reserve (von Ungern-Sternberg, Habre, Erb, & Heaney, 2009). It is unclear if children with multiple risk factors are being identified in clinical practice. The child’s medical history, the surgical procedure itself, and the anesthetic management can influence and contribute to adverse respiratory events during anesthesia. Bronchial hyperreactivity is a significant risk factor for the occurrence of perioperative adverse respiratory events (von Ungern-Sternberg et al., 2009). Bronchial hyperreactivity is often present in those with asthma, a respiratory tract infection, and those exposed to passive smoking. These underlying diseases lead to airway inflammation with subsequent alteration of the autonomic nervous system and enhancement of airway responsiveness to different stimuli encountered during anesthesia (von Ungern-Sternberg et al., 2009).

As previously mentioned, the surgical procedure and anesthetic management can be independent risk factors for perioperative adverse respiratory events. The incidence of bronchospasms is increased when an endotracheal tube is used versus a laryngeal mask airway (Looseley, 2010). According to Becke (2012), any manipulation of the airway, such as an endotracheal tube or a bronchoscopy, increases a child’s risk for perioperative adverse
respiratory events. Surgeries near the airway, such as ENT procedures or eye surgeries, along with surgeries that impair pulmonary function, such as cardiac or upper abdominal surgeries, increase the risk of perioperative adverse respiratory events (Becke, 2012).

**Smoking**

Second-hand smoke is a product of the burning end of a cigarette, known as sidestream smoke, which contains high concentrations of more than 6,000 substances, including ammonia, cyanide, benzene, nicotine, heavy metals, and carbon monoxide as well as many carcinogens (Lyons, 2011). According to Jones and Bhattacharyya (2006), many of these substances are toxic to the health of the passive smoker and many respiratory and non-respiratory diseases have been linked to passive smoking. Children are extremely vulnerable to passive smoke exposure because they are unable to escape the smoke. Also, due to their smaller size and developmental stage, children may be more susceptible than adults to the harmful byproducts of sidestream smoke (Lyons, 2011). The total deposition of particles in the lungs of children may be 50% greater than in adults who are exposed to second-hand smoke because of the differences in airway caliber (Lyons, 2011).

According to Lyons (2011), in 2005 the World Health Organization estimated that 57.2% of children were exposed to second-hand smoke in the home. Children who are exposed long term to second-hand smoke are 3.5 times more likely to have airway and pulmonary complications such as laryngospasm, bronchospasm, wheezing, coughing, stridor, increased mucus production, and oxygen desaturation following anesthesia (Lyons, 2011).

“In healthy lungs, the bronchial epithelium is an integrated structure that consists of ciliated cells, goblet cells, and basal cells with each of these cells having a role in the body’s
defense against infection (Lyons, 2011, pg. 21).” When tobacco smoke comes in contact with the bronchial epithelium via inhalation, the basal cells release inflammatory cytokines that may result in systemic inflammation (Lyons, 2011). Additional respiratory changes that occur as a result of exposure to second-hand smoke include hypersecretion of mucus, impaired tracheobronchial clearance, and small airway narrowing with increased closing capacity (Lyons, 2011). The reflex sensitivity of the lower and upper airways may also be increased as a result of second-hand smoke exposure. The direct effects of the harmful substances listed above include airway irritation involving immunologic mechanisms and mutagenesis (Jones & Bhattacharyya, 2006). Several otolaryngologic diseases have been linked to passive smoke exposure including chronic otitis media with effusion, chronic rhinitis, and adenotonsillar hypertrophy (Jones & Bhattacharyya, 2006).

In a study performed by Jones and Bhattacharyya (2006), 405 children age 0-18 were monitored through the perioperative period and the incidence of breath holding, laryngospasm, bronchospasm, hypersecretions, and airway obstructions was determined. Out of the 405 children, 168 of them had been exposed to second-hand smoke. Second-hand smoke exposure was defined as either one or both parents smoking at least one cigarette per day within the past twelve months. During anesthesia, the incidence of bronchospasm in patients not exposed to second-hand smoke compared to those exposed to passive smoke increased from 0.8% to 8.3%. In the recovery room, the incidence of bronchospasm in patients not exposed to second-hand smoke versus those exposed to passive smoked increased from 1.3% to 6.5%. The incidence of breath holding, laryngospasm, hypersecretions, and airway obstruction were all increased in the group exposed to second-hand smoke. As determined by this study, pediatric patients exposed to second-hand smoke are at a significantly increased risk for perioperative adverse respiratory
events. In order to prevent complications perioperatively, a preoperative assessment that includes screening for passive smoke exposure should be performed.

**Asthma**

The incidence of asthma and bronchial hyperreactivity is increasing in the pediatric population (Regli & von Ungern-Sternberg, 2014). According to Regli and von Ungern-Sternberg (2014), asthma is one of the most common chronic diseases in the world with a prevalence of 1-20%. Children who have asthma are at an increased risk of perioperative adverse respiratory events. Western lifestyles and obesity are associated with a higher incidence of asthma, bronchial hyperreactivity, other atopic diseases, and upper respiratory tract infections (Regli & von Ungern-Sternberg, 2014).

Regli and von Ungern-Sternberg (2014) define bronchial asthma as a variable and often reversible airflow obstruction and bronchial hyperreactivity. Bronchial hyperreactivity describes the increased ease and the higher degree of which airway narrowing occurs when stimulated while the increased likelihood of developing respiratory complications, particularly bronchospasm and laryngospasm, is known as airway susceptibility (Regli & von Ungern-Sternberg, 2014). In patients with acute and chronic airway inflammation, airway susceptibility can be increased leading to a higher occurrence of perioperative adverse respiratory events (Regli & von Ungern-Sternberg, 2014). Even without asthmatic or respiratory symptoms, airway susceptibility and bronchial hyperreactivity can still be present (Regli & von Ungern-Sternberg, 2014).

The degree of airway inflammation and remodeling of the airways varies in each patient who suffers from asthma. “While airway inflammation is characterized by infiltrations of
eosinophils, mast cells, and T-helper lymphocytes into the peripheral airways, the histological characteristics of remodeling are a thickening of the reticular basal membrane of the epithelial cells, airway smooth muscle and goblet cell hypertrophy and hyperplasia, and angiogenesis (Regli & von Ungern-Sternberg, 2014, pg. 289).” “Raised levels of interleukins caused by airway inflammation can increase immunoglobin E production and activated eosinophils can release leukotrienes and platelet activating factor associated with bronchoconstriction and mucus secretion (Regli & von Ungern-Sternberg, 2014, pg. 289).” Airway inflammation, remodeling, bronchial hyperreactivity, and wheeze are not symptoms that are just specific to asthma; they can occur in other respiratory diseases such as upper respiratory tract infections, cystic fibrosis, and bronchopulmonary dysplasia (Regli & von Ungern-Sternberg, 2014).

Due to several reasons, the incidence of perioperative adverse respiratory events are increased in children with asthma compared to adults with asthma. Increased airflow obstruction can occur due to reduced airway diameter (Regli & von Ungern-Sternberg, 2014). Children also tend to have mucous secretions that are more viscous and increased when compared to adults. In addition, epithelial damage along with shedding can plug large and small airways (Regli & von Ungern-Sternberg, 2014).

**Upper Respiratory Tract Infection**

Children who present for surgery with an upper respiratory tract infection have an increased risk for perioperative adverse respiratory events. According to Nagelhout and Plaus (2014), in children with an upper respiratory tract infection, the risk of developing adverse respiratory events, such as bronchospasm, laryngospasm, hypoxemia, atelectasis, croup, and stridor, is increased by as much as two to seven times. A child with an upper respiratory tract infection is at risk for perioperative adverse respiratory events because of the changes that occur
in the airway. An upper respiratory tract infection causes increased airway reactivity, a propensity for the development of atelectasis and mucous plugging of the airways, and an increased likelihood for developing postoperative arterial hypoxemia (Nagelhout & Plaus, 2014).

According to Tait, Voepel-Lewis, and Malviya (2000), approximately 95% of respiratory infections seen in children presenting for surgery are viral in origin. When the virus penetrates the epithelium and mucosa, the development of airway inflammation, edema, and bronchoconstriction occurs (Becke, 2012). This makes the airway more sensitive to secretions and volatile agents. “The viral infection interacts with the autonomic nervous system because viral neuraminidases can inhibit the cholinergic muscarinic M2 receptors, which is followed by an increased release of acetylcholine and consecutive bronchoconstriction (Becke, 2012, pg. 334).” “Bronchial hyperreactivity, which can persist for up to six weeks, may result from the virally induced liberation of tachykinin and neuropeptidases with a constriction of smooth muscles in the respiratory tract for weeks (Becke, 2012, pg. 334).”

There is still much debate regarding how long to postpone surgery after an upper respiratory tract infection. A study performed by von Ungern-Sternberg et al., (2010) provided evidence that the high risk for perioperative adverse respiratory events is limited to the first two weeks after an upper respiratory tract infection and there is no increase in the incidence of perioperative adverse respiratory events in children with upper respiratory tract infections more than two weeks before the procedure. According to Becke (2012), it is no longer necessary to postpone surgery six weeks; when clinical signs of an acute infection are present, a delay of at least two weeks is appropriate. According to Nagelhout and Plaus (2014), infectious nasopharyngitis that does not involve the lower respiratory tract requires postponing surgery for two weeks. Surgery should be postponed for four to six weeks when the child shows signs and
symptoms of lower respiratory tract involvement (Nagelhout & Plaus, 2014). Waiting this amount of time minimizes airway hyperactivity.

**Other Risk Factors**

A study performed by von Ungern-Sternberg et al., (2010) aimed to identify any associations between family history, anesthetic management, and occurrence of perioperative adverse respiratory events by assessing children preoperatively with an adapted version of the International Study Group for Asthma and Allergies in Childhood (ISAAC) questionnaire. The study included all children presenting for general anesthesia. The ISAAC questionnaire was used to record upper respiratory tract infections, including time of the infection (present, less than two weeks prior, or two to four weeks prior) and the symptoms involved (clear or green runny nose, fever indicated by a body temperature greater than 38 degrees Celsius, and a dry or moist cough), asthma and wheezing in the past twelve months or wheezing with exercise, nocturnal dry cough persisting for more than two weeks in the past year, present or past hay fever, eczema, or allergy, passive smoke exposure, and the occurrence of asthma, eczema, or hay fever in first-degree relatives. Perioperative adverse respiratory events were classified as laryngospasm, bronchospasm, airway obstruction, oxygen desaturation (less than 95%), and severe cough. All adverse respiratory events were documented along with when they occurred. A total of 9297 ISAAC questionnaires were available for analysis.

Findings from the study performed by von Ungern-Sternberg et al., (2010) showed that factors easily obtained from a preoperative assessment, including respiratory symptoms, eczema, or a family history of asthma, rhinitis, eczema, or exposure to second-hand smoke, were associated with an increased risk for the occurrence of perioperative adverse respiratory events. A positive respiratory history was more predictive for bronchospasm than for other perioperative
adverse respiratory events. The risk of bronchospasm was ten times higher in patients with a nocturnal dry cough than in patients without. The incidence of bronchospasm in children with a history of respiratory problems increased by 8% versus children with no history. When desflurane was used to maintain anesthesia, the incidence of bronchospasm was increased by 10% versus if sevoflurane was used. There was no difference in the incidence of bronchospasm if sevoflurane or propofol was used to maintain general anesthesia. A personal history of eczema increased the relative risk for bronchospasm. This might be explained by the fact that eczema, especially in older children, is frequently associated with atopy, present wheeze, and asthma. A personal history of hay fever was also associated with an increased risk for bronchospasm, which underlies the potential association between atopy and perioperative bronchospasm. A history of wheezing with exercise or more than three episodes in the past 12 months was associated with a greater risk of perioperative bronchospasm compared with the presence of a recent upper respiratory tract infection. This could be due to the presence of increased airway sensitivity caused by chronic airway inflammation. In this study, the presence of asthma in at least two family members was associated with a significantly increased relative risk of bronchospasm. Also, eczema, rhinitis, or asthma in at least two family members increased the risk of potentially life threatening complications, such as laryngospasm and bronchospasm, by nearly three times. This study also showed that a family history of asthma is an independent risk factor for perioperative adverse respiratory events.

**Etiology**

When diagnosing and treating a bronchospasm, it is of utmost importance to think of what could be causing the bronchospasm. Offending agents need to be identified promptly so the situation does not continue to deteriorate and proper treatment can begin. The anesthesia
practitioner should consider all potential causes such as anaphylaxis to a medication that was given, a reaction to blood products, a possible latex allergy, inadequate depth of anesthesia, or manipulation of the airway during times of light anesthesia (Nagelhout & Plaus, 2014). Airway soiling due to secretions, blood, or aspiration can also cause a bronchospasm (Loosely, 2010).

**Diagnosis**

A bronchospasm encountered during the perioperative period and especially after induction/intubation may involve an immediate hypersensitivity reaction including IgE-mediated anaphylaxis or a non-allergic mechanism triggered by factors such as mechanical or pharmacologic-induced (Dewachter, Mouton-Faivre, Emala, & Beloucif, 2014). Manifestations of bronchospasms can vary significantly depending on the severity of the spasm. Some bronchospasms may manifest as an audible wheeze giving practitioners a clue that there is increased resistance in the airways. If the patient is on the ventilator, an upward slope of the end-tidal carbon dioxide monitoring with a rise in end-tidal carbon dioxide may develop (Nagelhout & Plaus, 2014). Many bronchospasms will cause a decrease in oxygen saturations along with a decrease in tidal volume. Manually ventilating these patients can become extremely difficult due to the bronchoconstriction that develops in the smooth muscle of the airways. Other signs indicating a bronchospasm include prolonged expiration and increased peak inspiratory pressures (Nagelhout & Plaus, 2014).

**Differential Diagnosis**

Differential diagnoses should include endobronchial intubation, obstruction of the endotracheal tube secondary to increased secretions or blood, pulmonary aspiration, or a kinked endotracheal tube (Linck, 2007). When diagnosing a bronchospasm, practitioners need to also
rule out a laryngospasm. In non-intubated patients, acute laryngospasms can mimic bronchospasms and produce upper airway noise, reduced breath sounds, and difficulty in ventilation (Looseley, 2010). If clinical symptoms fail to resolve despite appropriate therapy, other etiologies such as pulmonary edema or pneumothorax should also be considered (Dewachter et al., 2014).

**Treatment**

If the anesthesia practitioner suspects that the patient is experiencing a bronchospasm, the first intervention includes removing the offending agent and placing the patient on 100% FiO\(_2\). Wheezing may be noted either audibly or through auscultation. If a bronchospasm develops during a procedure and if it is severe, the surgeon should be notified and additional staff called for assistance (Nagelhout & Plaus, 2014). The anesthetic depth should be increased with a volatile agent, ketamine, propofol, or a combination of these (Nagelhout & Plaus, 2014). After the anesthetic depth is increased, verify correct placement of the endotracheal tube and determine if it has migrated distally to possibly cause irritation in the lungs. If the patient is on the ventilator, the mode should be switched to manual and the patient ventilated by hand. Manually ventilating the patient allows the provider to evaluate pulmonary compliance and to identify all causes of high-circuit pressure (Nagelhout & Plaus, 2014).

**Anesthetic Agents**

The anesthetic depth should be increased either by increasing the volatile anesthetic or administering a bolus of propofol. The volatile anesthetics are useful due to their potent bronchodilatatory effect, with sevoflurane having the greatest effect (Regli & von Ungern-Sternberg, 2014). Desflurane should be avoided because it increases bronchial smooth muscle tone and
airway resistance (Regli & von Ungern-Sternberg, 2014). According to Regli and von Ungern-Sternberg (2014), desflurane should be avoided in pediatric patients, especially in those with asthma, because it is associated with an increased risk of perioperative adverse respiratory events, particularly bronchospasm.

Administering a bolus of propofol can help treat a bronchospasm by relaxing the smooth muscles of the airway. Propofol has a fast onset of action providing a smooth induction and an improved quality of emergence from anesthesia (Becke, 2012). As compared to the volatile anesthetics, propofol is superior in blunting airway reflex bronchoconstriction but has inferior bronchodilating properties (Regli & von Ungern-Sternberg, 2014). However, Becke (2012) claims that the bronchodilating effects of propofol are similar to that of the volatile agents.

**Inhaled Agents**

Albuterol is the preferred selective beta 2 adrenergic agonist for the treatment of acute bronchospasm. The beta 2 adrenergic agonists are a class of drugs that bind with high specificity to beta adrenergic receptors found on human airway endothelial and epithelial cells, airway smooth muscle cells, as well as immune cells such as T lymphocytes and eosinophils (Fitzgerald, 2006). All beta agonists are structural analogs of human epinephrine and produce airway muscle relaxation and bronchodilation and promote mucociliary transport (Fitzgerald, 2006). Albuterol is most often administered through a metered-dose inhaler, producing about 100 micrograms per puff (Stoelting & Hillier, 2006). For patients who are intubated, albuterol puffs can be delivered via the endotracheal tube and manual breaths given to deliver the agent to the lungs. The duration of action of an inhaled dose is about four hours; however, significant relief of symptoms may persist for up to eight hours (Stoelting & Hillier, 2006). When combined with volatile anesthetics, there is an additive effect on bronchomotor tone (Stoelting & Hillier, 2006).
In the presence of life-threatening bronchoconstriction, racemic albuterol should be given at a dose of 0.15 milligram per kilogram every 20 minutes for three doses, then 0.15-0.30 milligrams per kilogram up to 10 milligrams every 1 to 4 hours as needed (Fitzgerald, 2006). If needed, continuous nebulization of albuterol should be started at 0.5 milligrams per kilogram per hour (Fitzgerald, 2006). When large doses of albuterol are used, the practitioner should be aware of the possible side effects, such as tachycardia and hypokalemia. Despite these side effects, large doses of albuterol may be necessary due to decreased deposition at the site of action secondary to small tidal volumes and narrowed airways. For asthmatic patients and those with a reactive airway, inhaled albuterol blunts airway responses to tracheal intubation (Stoelting & Hillier, 2006).

According to Dewachter et al., (2011), short-acting beta 2-selective agents, such as terbutaline and salbutamol, are key drugs for the fast relief of bronchoconstriction and there is no difference in efficacy between the two. The onset of action for these drugs occurs within five minutes, peak effect is within 60 minutes, and the duration of action is four to six hours (Dewachter et al., 2011). They should be immediately administered via a nebulizer (8–10 puffs) to achieve appropriate therapeutic levels (Dewachter et al., 2011).

Terbutaline is a predominantly beta 2 adrenergic agonist. It can be administered orally, subcutaneously, or by inhalation to treat bronchospasms (Stoelting & Hillier, 2006). If given by the subcutaneous route at a pediatric dose of 0.01mg/kg, it will produce responses that resemble those of epinephrine with a longer duration of action (Stoelting & Hillier, 2006). Each metered dose puff delivers about 200 micrograms (Stoelting & Hillier, 2006).
Intravenous Treatment

Pediatric patients have a limited respiratory reserve, thus they will not be able to tolerate periods of apnea. Therefore, it may be necessary to intervene with epinephrine sooner than you would if treating an adult with the similar diagnosis (Nagelhout & Plaus, 2014). Epinephrine 5-10 micrograms per kilogram should be used as the initial subcutaneously injected bronchodilator in children (Nagelhout & Plaus, 2014). In life threatening emergencies, dilute a 1-milligram vial of epinephrine in a 10-milliliter syringe, give one to two milliliters (100-200 micrograms) IV push, in increments, for a maximum dose of 0.5 milligrams per dose of a 1:1000 solution and wait for the child’s reaction (Nagelhout & Plaus, 2014).

Epinephrine is a catecholamine that is released from the adrenal medulla. Its functions include regulation of myocardial contractility, heart rate, vascular and bronchial smooth muscle, glandular secretions, and metabolic processes such as glycogenolysis and lipolysis (Stoelting & Hillier, 2006). Epinephrine activates beta 1 and beta 2 receptors. Activation of beta 2 receptors results in bronchial smooth muscle dilation. When beta 2 receptors are activated, there is an increase in intracellular concentrations of cAMP (Stoelting & Hillier, 2006). As a result, a reduction in vasoactive mediators occurs. The end result is relaxation of the smooth muscle in the airway responsible for bronchoconstriction (Stoelting & Hillier, 2006).

Steroids

Another key medication in the treatment of bronchospasms is parenteral steroids. They decrease airway inflammation and help speed resolution of exacerbations. However, their effect is not immediate and therefore, they should not be used as a first line treatment (Dewachter et al., 2011). Regli and von Ungern-Sternberg (2014) recommend that preoperative oral steroids be
reserved for children with severe bronchial hyperreactivity. Preoperative oral steroids should only be used when the benefits outweigh the risk of potential side effects. Asthmatic patients should continue all asthma medications including long-term oral corticosteroids throughout the perioperative period (Regli & von Ungern-Sternberg, 2014). If steroids are used, improvement in symptoms can be seen as early as six hours after initiation but the majority effect will occur after 48 hours (Regli & von Ungern-Sternberg, 2014).

**Recommendations**

As anesthesia practitioners, it is likely that we will be challenged on a day to day basis with pediatric patients presenting for surgery with either an active upper respiratory tract infection, asthma, or with other risk factors increasing their chance for developing an adverse respiratory event such as a bronchospasm. For these patients at risk, canceling the procedure will likely not be an option due to several reasons. Upper respiratory tract infections are common in the pediatric patient and cancellations can place an emotional and economic burden on families due to the fact that parents have to miss work to stay at home and care for their child. For the patients who are at risk due to multiple risk factors which do not include an upper respiratory tract infection, these risk factors will likely always be present regardless of when the procedure is performed. Choosing an anesthetic strategy that minimizes the risk of perioperative adverse respiratory events is key in these patients.

**Preoperative Interventions**

For those patients who have multiple risk factors for developing a bronchospasm or those with an active upper respiratory infection, premedication might help prevent an adverse respiratory event. According to von Ungern-Sternberg et al., (2009), premedication of the child
with a beta 2 agonist such as salbutamol has been demonstrated to be effective in preventing increases in total respiratory resistance and in decreasing the incidence of perioperative bronchospasm. Von Ungern-Sternberg et al., (2009) performed a prospective randomized controlled study to investigate the impact of salbutamol premedication on the incidence of perioperative laryngospasm, bronchospasm, desaturation (<95%), and severe coughing in children with a recent upper respiratory tract infection. The study consisted of 600 children age 0-16 undergoing general anesthesia with either a laryngeal mask airway or endotracheal tube. The 600 children were divided into three different groups. Four hundred of the children had an upper respiratory tract infection within the past two weeks; however, no active infection was present. The study defined upper respiratory tract infection as having a moist cough. The other 200 children had not suffered from an upper respiratory tract infection within the past four weeks.

Two hundred children with a respiratory tract infection received preoperative salbutamol (2.5 mg if weight <20 kg, 5 mg if weight >20 kg) through a nebulizer 10–30 minutes prior to surgery while 200 children with a respiratory tract infection did not receive salbutamol. The exclusion criteria for the study consisted of airway malformations, major airway reconstructive or cardiothoracic surgery, and/or a current respiratory tract infection. Either sevoflurane or propofol were used for induction and anesthesia was maintained with sevoflurane in all the cases. All adverse respiratory events, such as laryngospasm, bronchospasm, oxygen desaturation (<95%), and severe coughing, in the perioperative period were recorded.

The 200 children who were healthy presented with the lowest rate of respiratory complications regardless of whether salbutamol was given preoperatively. However, children who presented with a moist cough in the two weeks prior to surgery who received a
premedication with salbutamol demonstrated a significant reduction in the incidence of perioperative bronchospasm (5.5% vs 11%) and severe coughing (5.5% vs 11.5%) compared with children who had a respiratory tract infection but did not receive salbutamol. The results of this study suggest that the incidence of adverse respiratory events might be significantly reduced if premedication with salbutamol is used in children with a recent respiratory tract infection. Premedication in children who have other risk factors besides an upper respiratory tract infection might be beneficial and could possibly reduce the incidence of perioperative adverse respiratory events.

According to Regli and von Ungern-Sternberg (2014), in children with a history of asthma, preoperative beta 2 agonists should be given because they have been shown to improve lung function and reduce perioperative adverse respiratory events including bronchospasms. In regards to reversing perioperative bronchoconstriction, beta 2 agonists are more effective compared to ipratropium bromide (Regli & von Ungern-Sternberg, 2014). According to Looseley (2010), the risk of bronchospasms can be reduced with pretreatment with an inhaled/nebulized beta agonist 30 minutes prior to surgery, induction of anesthesia with propofol, and adequate depth of anesthesia before airway instrumentation.

A discussion with the parents should take place and they should be made aware of the problems with respiratory tract infections and anesthesia. They should be questioned to determine if the child’s symptoms are acute or chronic. Symptoms that would suggest a delay in elective surgery are a fever greater than 38.4 degrees Celsius, malaise, productive cough, wheezing, or rhonchi (Nagelhout & Plaus, 2014). According to Nagelhout and Plaus (2014), four to six weeks is considered a reasonable period of delay for these types of symptoms. For symptoms considered mild, such as a nonproductive cough, sneezing, or mild nasal congestion,
surgery could proceed as long as regional anesthesia or general anesthesia without an endotracheal tube is performed (Nagelhout & Plaus, 2014). If the nasal or oropharynx were to be suctioned, it should be done under deep anesthesia before emergence. The practitioner should also be vigilant with hydration status, consider using airway humidification, and consider the potential benefit of pharmacologic agents such as anticholinergics and beta agonists to help with airway secretions and airway hyperreactivity (Nagelhout & Plaus, 2014). When the use of an endotracheal tube is required for anesthesia, especially in children less than one year old, practitioners need to identify risk factors such as passive smoke exposure and underlying conditions such as asthma or chronic lung disease because these children might benefit from a two to four week delay in surgery (Nagelhout & Plaus, 2014). For the patients who present for surgery with resolving respiratory tract infections with severe or mild symptoms, a delay in surgery should occur to minimize the risk of proceeding with surgery. Surgery should be postponed two to four weeks after resolution of a minor upper respiratory infection and four to six weeks after resolution of a severe upper or lower respiratory tract infection (Nagelhout & Plaus, 2014).

**Case Study**

In the aforementioned case study, the patient had several risk factors placing him at risk for perioperative adverse respiratory events. These risk factors included the child’s age, being born pre-term, recent ear infections, and exposure to second-hand smoke. Based on current evidence, the patient could have benefited from a preoperative albuterol treatment. In addition, the intravenous epinephrine dosing of 10 mcg should have been administered in increments closer to 100 mcg. The patient may have benefited from the utilization of the volatile agent that was initially discontinued at the end of the procedure right before the onset of the bronchospasm.
As recommended in the literature, the endotracheal tube was removed in order to rule out the possibility that it could have been contributing to the bronchospasm. Finally, the literature also recommends the initiation of steroids intra-operatively or in the PACU rather than in the PICU as was done in this case study.

Conclusion

Having a current or recent upper respiratory tract infection is a huge risk factor for perioperative adverse respiratory events such as a bronchospasm. However, it is not the only risk factor that anesthesia practitioners need to be aware of when determining the appropriate anesthetic management for each patient. Pediatric patients may present for surgery and appear perfectly healthy with the parents denying any recent colds or runny nose. However, anesthesia practitioners need to be extra vigilant and ask about other risk factors that could possibly cause perioperative problems, such as smoke exposure at home, wheezing, history of asthma, or a family history of asthma, eczema or rhinitis. An accurate assessment of the risk of perioperative adverse respiratory events during the preoperative assessment could enable anesthetic management to be tailored to reduce the likelihood of respiratory complications, such as bronchospasms.
Reference


Identifying Risk Factors for Bronchospasms in the Pediatric Patient

Kayla Stiles, SRNA

Adverse Respiratory Events

- 75% of all critical incidents and 33% of all perioperative cardiac arrests in pediatric anesthesia are caused by adverse respiratory events.
- Due to their limited oxygen reserve and increased oxygen demand, the pediatric population does not tolerate these adverse respiratory events.
- Identifying risk factors for perioperative bronchospasms along with being adequately prepared can help prevent life-threatening complications.

Pathophysiology of Bronchospasm

- Smooth muscle tone of the airway is regulated by two chemical compounds: cyclic adenosine 3’5’-monophosphate (cAMP) and cyclic guanosine 3’5’-monophosphate (cGMP).
- However, it is believed that the primary mechanism of bronchospasm involves parasympathetic nervous system stimulation.

Case Information

- Hypospadias Repair
- 7 month old
- 8.6 kg
- Male
- ASA 1

Pre-operative Evaluation

- Past Medical History: Hypospadias, cephalohematoma at birth, umbilical hernia, and born preterm (35 weeks)
- Surgical History: No prior surgical hx
- Airway Evaluation: Unable to be determined due to child’s age and cooperativeness
- Medications: No home medications
- Allergies: NKDA
- Labs/Tests: None drawn

Anesthetic Course

- No pre-op meds were given
- Induction: An inhalational induction was utilized with 70% nitrous oxide, 30% oxygen, and sevoflurane at 8%.
- 24 gauge IV started, gas reduced to 5% and 10 mcg of fentanyl and 10 mg propofol given.
- Intubated with a 3.5 cuffed ETT
- Maintenance: Expiratory sevoflurane concentration ranged from 2.5-3.5% throughout the case.
- IV utilized with tidal volumes ranging from 50-70 mL
- Intra-op Meds:
  - Zofran 1 mg
  - Avlocad 245 mg
  - LR 250 mL
- Surgeon performed penile block; therefore no additional narcotics needed.
Intraoperative Issues

- Upon completion of the procedure, sevo was discontinued and the patient was placed on 100% FiO2. Within seconds after discontinuing the sevo, the patient’s oxygen saturations decreased from 100% to 75%.
- It was determined that the patient was experiencing a bronchospasm and epinephrine 10 mcg IV push was given and the patient was manually ventilated via the ETT.
- Approximately two minutes later, the patient again experienced a bronchospasm. The ETT was still in place at this time. Oxygen saturations quickly dropped to around 20% despite treatment with epinephrine.
- We were unable to obtain an ETCO, tracing, unable to manually ventilate, a code blue was called, no pulse palpable, and chest compressions initiated.

Post-op

- No further issues encountered in PACU.
- Patient admitted to PICU where he was given 4 mg of decadron for intermittent stridor and started on ceftriaxone.
- It was then noted that the patient had been treated two weeks prior for bilateral otitis media that was unresolved after a week of treatment. At this point, he was started on amoxicillin.
- It was also discovered that both parents were smokers.
- A chest x-ray was obtained that showed moderate, bilateral bronchial wall thickening, indicating inflammation in the airways.

Risk Factors for Perioperative Bronchospasm

- Higher ASA classification
- Recent upper respiratory tract infection
- Prematurity
- Low birth weight
- Children age 0 to 9 years; with highest incidence occurring in children 0 to 3 months old
- History of asthma

Risk Factors for Perioperative Bronchospasm

- Previous or current eczema
- Family history of eczema, asthma or hay fever in two or more relatives
- Wheezing occurring more than three times during the previous 12 months
- Exposure to passive smoke
- Type of anesthetic
- Type of procedure

Passive Smoke Exposure

- 3.5 times more likely to have airway and pulmonary complications
- When tobacco smoke comes in contact with the bronchial epithelium via inhalation, the basal cells release inflammatory cytokines that may result in systemic inflammation. Additional respiratory changes include hypersecretion of mucous, impaired tracheobronchial clearance, and small airway narrowing with increased closing capacity.
- During anesthesia, the incidence of bronchospasm in patients not exposed to second-hand smoke compared to those exposed to passive smoke increased from 0.8% to 8.3%. In the recovery room, the incidence of bronchospasm in patients not exposed to second-hand smoke versus those exposed to passive smoke increased from 1.3% to 6.5%.

Asthma

- One of the most common chronic diseases in the world with the incidence increasing
- Defined as a variable and often reversible airflow obstruction and bronchial hyperreactivity.
- The degree of airway inflammation and remodeling of the airways varies in each patient who suffers from asthma.
- The incidence of perioperative adverse respiratory events are increased in children with asthma compared to adults with asthma:
  1. Increased airflow obstruction can occur due to reduced airway diameter and 2. Children tend to have more mucus secretions compared to adults.
Upper Respiratory Tract Infection

- Causes increased airway reactivity, a propensity for the development of atelectasis and mucus plugging of the airways, and an increased likelihood for developing postoperative arterial hypoxemia
- In children, a URI, the risk of developing adverse respiratory events, such as bronchospasm, laryngospasm, hypoxemia, atelectasis, cough, and stridor, is increased by as much as two to seven times
- Most URIs are viral in origin; when the virus penetrates the epithelium and mucosa, the development of airway inflammation, edema, and bronchoconstriction occurs making the airway more sensitive to secretions and volatile agents
- How long to postpone surgery???

Other Risk Factors

- The presence of asthma in at least two family members was associated with a significantly increased relative risk of bronchospasm
- Eczema, rhinitis, or asthma in at least two family members increased the risk of potentially life threatening complications, such as laryngospasm and bronchospasm, by nearly three times

Etiology of Bronchospasm

- Anaphylaxis to a medication that was given
- Reaction to blood products
- Possible latex allergy
- Inadequate depth of anesthesia
- Manipulation of the airway during times of light anesthesia
- Airway soiling due to secretions, blood, or aspiration

Diagnosis of Bronchospasm

Manifestations can vary significantly depending on the severity of the spasm
- May manifest as an audible wheeze giving practitioners a clue that there is increased resistance in the airways
- An upward slope of the ETCO2 tracing with a rise in ETCO2 may occur
- A decrease in oxygen saturations along with a decrease in tidal volume
- Manually ventilating these patients can become extremely difficult due to the bronchoconstriction that develops in the smooth muscle of the airways
- Might also see prolonged expiration and increased peak inspiratory pressures

Differential Diagnosis

- Endobronchial intubation
- Obstruction of the ETT secondary to increased secretions or blood
- Pulmonary aspiration
- Kinked ETT
- In non-intubated patients, acute laryngospasms can mimic bronchospasms and produce upper airway noise, reduced breath sounds, and difficulty in ventilation
- Other causes such as pulmonary edema or pneumothorax should also be considered
BRONCHOSPASM IN PEDIATRICS

Treatment
1. Remove offending agent and start 100% FiO₂
2. Call additional staff if needed
3. Increase anesthetic depth via propofol, ketamine, or volatile agent
4. Verify correct position of ETT. Did it migrate distally?
5. Manually ventilate patient

References

Recommendations
- Choosing an anesthetic strategy that minimizes the risk of perioperative adverse respiratory events is key in patients with multiple risk factors.
- In one study, premedication of a child with a beta 2 agonist such as albuterol was effective in preventing increases in total respiratory resistance and in decreasing the incidence of perioperative bronchospasm.
- Children who presented with a moist cough in the two weeks prior to surgery who received a premedication with albuterol demonstrated a significant reduction in the incidence of perioperative bronchospasm (5.5% vs 11%) and severe coughing (3.5% vs 11.5%) compared with children who had a respiratory tract infection but did not receive albuterol.

Conclusion
- It is important to remember that regardless of the patient’s medical history, severe bronchospasms can still occur despite the presence or absence of risk factors. However, awareness and identification of these risk factors along with pretreatment when applicable could result in fewer perioperative adverse respiratory events.

References