# Pitfalls of Pediatric Anesthesia

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#### **Emergence Delirium**

Emergence delirium (ED) also referred to as emergence agitation (EA) is a well documented phenomenon occurring in children and adults in the immediate postoperative period.<sup>1</sup> With the recent popularity of the newer inhalation agents desflurane and sevoflurane, numerous clinical studies have been published questioning the association of these anesthetics with an increased incidence of emergence delirium.

Emergence delirium is defined as a dissociated state of consciousness in which the child is inconsolable, irritable, uncompromising or uncooperative, typically thrashing, crying, moaning, or incoherent.<sup>2-4</sup> Additionally paranoid ideation has been observed in combination with these emergence behaviors.<sup>4</sup> Characteristically these children do not recognize or identify familiar and known objects or people. Parents who witness this state claim the behavior is unusual and uncustomary for the child. Although generally self limiting (5-15 min) ED can be severe and may result in physical harm to the child and particularly the site of surgery.<sup>3,5</sup>

Emergence delirium is not a new phenomenon; it has been reported after the introduction of every new anesthetic including most inhalational agents and intravenous agents including midazolam, remiferitanil and propofol.<sup>6</sup>

Other drugs know to be associated with ED include 1) atropine or scopolamine, 2) ketamine, 3) droperidol, 4) barbiturates and possibly, 5) benzodiazepines.

The incidence of emergence delirium in all postoperative patients is 5.3% with a more frequent incidence in children (12-13%).<sup>7</sup> The incidence of emergence delirium after halothane, isoflurane, sevoflurane and desflurane ranges from 2-55%.<sup>8-12</sup>

It has been postulated that this phenomenon has become more acutely noticed with the increased use of agents such as sevoflurane and desflurane that have a rapid emergence profile due to their low blood gas solubility profile. It was thought that rapid emergence with lack of adequate pain control before emergence contributed or was the cause of this problem. This has been supported by Davis noting that ketorolac administration decreased the incidence of emergence delirium three to fourfold after myringotomy with either halothane or sevoflurane anesthesia.<sup>13</sup> Several studies have documented a reduction in ED with the administration of fentanyl 2.5 ug/kg intraoperatively or 2.0 ug/kg fentanyl administered intranasally, 1 ug/kg in each nostril after the induction of anesthesia.<sup>14-18</sup>

However, other studies have noted emergence delirium in children after sevoflurane anesthesia despite effective regional blocks to prevent postoperative pain.<sup>12</sup> These studies demonstrated that delirium occurred more frequently in preschool age children, 1 to 5 years of age, lasted 5-15 minutes in the recovery room and often resolved spontaneously.<sup>12,19,20</sup>

Studies that have also called into question whether adequate pain management is the cause of ED include sevoflurane vs halothane administration in patients undergoing MRI (no surgery) where the incidence of ED was 33% in sevoflurane treated patients versus 0% in halothane treated children.<sup>21</sup> But in patients undergoing MRI with sevoflurane anesthesia those that had a dose of intravenous fentanyl 1 ug/kg, 10 minutes before discontinuation of the anesthetic had an incidence of emergence agitation of 12% versus 56% in the placebo group.<sup>22</sup> An unanswered question was although discharge time was similar in both groups did the administration of fentanyl cause a slower awakening time?

In addition, studies have also called into question pain and rapid emergence as a cause for ED, since in these studies pain was well controlled and patients still exhibited signs of ED.<sup>23,24</sup> Murray demonstrated that preemptive oxycodone reduced post anesthesia agitation for children who received halothane but not for those who received sevoflurane.<sup>25</sup>

It must recognize in these studies it may be difficult to differentiate pain related agitation from other sources. Although pain cannot be entirely excluded as a contributing factor for ED the data does suggest the influence of another mechanism. Consideration of pain as a potential factor for agitation is important particularly in children undergoing short surgical procedures for which peak effect of analgesias may be delayed until they are well awake.

Other anesthetic techniques have been associated with decreased incidence of ED. Preschool children receiving a propofol versus sevoflurane anesthetic had a 38% incidence of ED with sevoflurane versus 0% with propofol. However, sevoflurane still provided a shorter PACU stay than propofol.<sup>24</sup> This was also confirmed by Cohen who had an incidence of 23.1% of patients with ED who received sevoflurane versus 3.7% with propofol.<sup>23</sup>

Another agent clonidine given intravenously at a dose of 2 ug/kg after induction of anesthesia in 40 male children age 2-7 years undergoing circumcision decreased emergence agitation to 2 patients in the clonidine group compared with 16 patients in the placebo group, being severe in 6 patients. Pain control was a penile nerve block before surgery.<sup>26</sup>

What about premedication in relation to ED - does it make it better or worse? Preoperative administration of midazolam has reduced the incidence of ED as compared with placebo<sup>27</sup> as has clonidine<sup>28</sup> however, this may be due to slower awakening rather than anxiety.

Oral premedication with the midazolam syrup 0.2 mg/kg had an incidence of emergence agitation that was seen in 47% of patients as compared to 81% who received a saline placebo.<sup>29</sup> In addition the midazolam premedication did not prolong the PACU stay. Sevoflurane has been reported at high concentrations to potentiate and at low concentrations to block gamma-aminobutyric acid A (GABAA) receptor mediated inhibitory postsynaptic currents (IPSC<sub>s</sub>).<sup>30</sup> Whether the biphasic effects of sevoflurane on GABA<sub>A</sub> receptor mediated IPSC<sub>s</sub> contribute to sevoflurane-induced EA remains unclear. Evidence that induction of sevoflurane anesthesia with propofol results in calmer recovery results in calmer recovery<sup>31</sup> does support that potentiation of GABA<sub>A</sub> receptor - mediated IPSC<sub>s</sub> may improve the recovery quality of sevoflurane. It is postulated that benzodiazepines such as midazolam potentiate the inhibitory effects of GABA<sub>A</sub> receptors<sup>32</sup> and therefore could be promising agents for improving the recovery quality of sevoflurane anesthesia. This study also confirmed that children younger than five years of age are more likely to have EA after sevoflurane anesthesia.<sup>33</sup> Ben-Ari et al indicated that the GABA<sub>A</sub> receptor could be excitatory rather than inhibitory in the early postnatal period<sup>34</sup> with the development of excitatory inputs the GABA<sub>A</sub> receptor becomes inhibitory as the child ages.<sup>34</sup> This development in the GABA<sub>4</sub> receptor is due to a switch from high to low chloride content in the neurons.<sup>35</sup> Developmental differences in the neurotransmitters and neuromodulators may account for the age-related differences.

Other data have contradicted this notion.<sup>36-39</sup> One study found children who received midazolam experienced ED more frequently than those who did not and observed the agitation lasted longer.<sup>30</sup>

Although Kain et al<sup>38</sup> demonstrated a decreased incidence of maladaptive behaviors at postoperative weeks after midazolam there was no difference in ED between children who received midazolam and those who did not. Furthermore benzodiazepines themselves have been associated with paradoxical reactions and agitation that have been reversed with flumazenil.<sup>40</sup>

Therefore other causes of emergence delirium must be searched for. It may be due to misperception of environmental stimuli with paranoid ideation,<sup>4</sup> differing CNS effects<sup>4,14,41</sup> sympathetic activation or some type of psychomotor effect.<sup>14</sup> It is known that the EEG pattern with sevoflurane differs from that of patients anesthetized with halothane.<sup>42</sup>

Although we may not know the cause of emergence delirium a recent prospective cohort study of emergence agitation in the pediatric post anesthesia care unit may give us the most insight into patients at risk for this phenomenon.<sup>43</sup>

Five hundred twenty-one children age 3-7 years who were undergoing general anesthesia for elective outpatient procedures were included. Ninety-six (18%) had emergence agitation. The agitation lasted up to 45 minutes in some cases with mean of 14 minutes. Fifty-two percent of children with agitation required pharmacologic intervention with prolonged post anesthesia care unit stay of 16 minutes over non-agitation children. Ten factors were associated with EA including 1) younger age (4.8 vs 5.9 years), 2) no previous surgery, 3) poor adaptability, 4) ophthalmology and 5) otorhinolaryngology procedures, 6) sevoflurane, 7) isoflurane, 8) sevoflurane/isoflurane, 9) analgesics and 10) short time to awakening. Of these otorhinolaryngology procedures, time to awakening and isoflurane were shown to be independent risk factors. Emergence agitation was associated with 5 adverse events, increased bleeding from surgical site (1) pulling out a surgical drain or an IV (2) increased pain at the operative site and minor injury of a nurse.

Interestingly enough children who received a combination of sevoflurane and isoflurane for induction and maintenance were twice as likely to have EA compared with any other anesthetic regimen.

Almost all children (98%) who had EA received intraoperative analgesics as compared with 86% of nonagitated children. There was no difference in the duration of anesthesia in the two groups but time to awakening was shorter in the EA group  $14 \pm 14$  min versus  $26 \pm 23$  min.

Why the increased incidence in otorhinolaryngology procedures? Eckenhoff et al<sup>44</sup> and Bastron and Moyers<sup>45</sup> speculate a "sense of suffication" may contribute to EA in patients undergoing head and neck procedures but there is no scientific data to support this. As far as premedication with preoperative midazolam there was similar incidence in EA in those who received midazolam as those who did not (15% versus 19% respectively).

Temperament of the child with low adaptability has also been associated with sedation failure.<sup>46</sup> Kain has demonstrated children who were not enrolled in day care, those with no sibling and those who were very impulsive were at a greater risk for developing negative behavior changes such as separation anxiety, nightmares and bedwetting at two or more weeks after surgery.<sup>47</sup> More study definitely needs to be done in this area of temperament and emergence outcomes.

Treatment of emergence delirium can include opioids, midazolam, propofol and flumazenil. Propofol (0.5 mg/kg IV) or midazolam (0.02 mg/kg IV) have both been used successfully in treating ED in children.<sup>14</sup> When deciding on a therapy remember the occasional child may have a paradoxical reaction to midazolam, characterized by agitation combativeness or inability to be consoled. If the reaction is suspected consider the use of flumazenil 0.01 mg/kg IV, (max 0.2 mg/dose) at 1-2 min intervals to a maximum dose of 1 mg. In children greater than 12 years of age, give 0.2 mg/dose at 1-2 min intervals up to a total dose of 1 mg.

It is important to remember that several life threatening considerations (eg. hypoxia, severe hypercarbia, hypotension, hypoglycemia, increased intracranial pressure) may also result in disorientation and altered mental status. These entities must be diagnosed and treated promptly. Bladder distention may also yield a similar clinical picture.

## **Airway/Respiratory Complications**

#### **Upper Airway Obstruction**

Transient upper airway obstruction is relatively common in the immediate recovery period. These patients will have a paradoxical respiratory pattern with a sucking in of the chest and distention of the abdomen with inspiration. The incidence is higher in pediatric patients than adults because of the larger amount of airway soft tissue in children (tonsils and adenoids) as well as the residual effects of potent vapor anesthetics. Residual inhalational anesthetic causes persistence of hypotonia of the pharyngeal muscles and posterior displacement of the tongue.

Careful patient positioning placing the patient on their side and airway maneuvers can decrease this obstruction. But which airway maneuvers are the most effective in relieving this obstruction? Two recent articles give us insight into these maneuvers. Video analysis of airway obstruction in children showed that chin lift and jaw thrust do not always create a completely patent pharynx and laryngeal aperture. Adding CPAP of 10 cm of H<sub>2</sub>O to either of these maneuvers improved upper airway patency and significantly decreased the stridor score.<sup>48</sup> Also in children with adenoidal hyperplasia, although chin lift may improve airway patency and ventilation, jaw thrust with or without CPAP 5 cm H<sub>2</sub>O was the most effective maneuver to overcome airway obstruction in these children.<sup>49</sup>

## Laryngospasm

Laryngospasm can occur in the PAR especially after transport of the patient following "deep extubation."

Two areas have recently come into focus as relative to the risk of development of laryngospasm. First of 15,183 children in a day surgery unit who had general anesthesia, those who developed laryngospasm were 2.05 times more likely to have an active upper respiratory infection (URI). They were also more likely to be younger and be undergoing airway surgery.<sup>50</sup> Bronchospasm has been shown to be higher in intubated patients with a URI.<sup>51,52</sup> LMA use instead of intubation has shown that laryngospasm was equal in both groups but the incidence of mild bronchospasm was higher in the intubated group. Therefore the LMA may be a suitable alternative if the decision is made to proceed with anesthesia in a child with a URI but will not decrease the risk of laryngospasm.<sup>53</sup>

Secondly, children exposed to environmental tobacco smoke (ETS) were studied. Of those exposed to ETS, 9.4% developed laryngospasm whereas those without domestic ETS exposure, only 0.9% developed laryngospasm. All occurred on emergence from general anesthesia.<sup>54,55</sup>

# Management

Laryngospasm may present as complete or incomplete airway obstruction. If incomplete airway obstruction is present sounds should be present - eg. grunts, expiratory squeaks. Complete or incomplete airway obstruction should have initial treatment with jaw thrust and chin lift. The middle finger of each hand should be placed in the "laryngospasm" notch. It is behind the lobule of the pinna of each ear and is bounded anteriorly by the ascending ramus of the mandible adjacent to the condyle, posteriorly by the mastoid process of the temporal bone and cephalad by the base of the skull. Press firmly inward toward the base of the skull with both fingers at the same time lifting the mandible at the right angle to the plane of the body (jaw thrust or forward displacement of the mandible). This will convert laryngospasm within one or two breaths to laryngeal stridor and then to unobstructed respirations.<sup>56</sup> This should include administration of 100% O<sub>2</sub> with gentle positive pressure. It must be remembered that incomplete airway obstruction may rapidly become complete.

Complete airway obstruction shares many of the signs of incomplete airway obstruction - tracheal tug, retractions of the chest wall and marked abdominal respiration, but there is no sound present. Positive pressure will not "break" laryngospasm in the presence of complete airway obstruction.<sup>57</sup> It may worsen it by forcing supraglottic tissues downward into the glottic opening. High pressure generated by the flush valve may dilute anesthetic gases, and lead to a lighter level of anesthesia. It may also force gas down the esophagus into the stomach making ventilation more difficult. If IV access is present succinylcholine 0.5-

1 mg/kg with atropine 0.02 mg/kg can be given. If no IV access is present IM succinylcholine 4 mg/kg is recommended to be given in the deltoid muscle. Intralingual atropine and succinylcholine can cause ventricular arrhythmias in patients anesthetized with halothane.<sup>58</sup> If laryngospasm is sustained and the child is extremely hypoxic it may be necessary to intubate without muscle relaxants.

## **Post Intubation Croup**

Post intubation croup is more common than in adult cases and seen in between 1-6% of pediatric cases. Factors that contribute to post intubation croup are traumatic or repeated intubations, coughing "bucking" on the tube and changing the patients position after intubation. Other factors include increased incidence in patients with trisomy 21, surgery greater than 1 hour, surgery of the head and neck and a tight fitting endotracheal tube (air leak >25 cm  $H_2O$ ).

Post intubation croup usually becomes symptomatic within the first hour after extubation with maximum edema usually occurring at 4 hours after extubation and resolving by 24 hours. Initial treatment is humidified oxygen by mask adding racemic epinephrine (0.25-0.5 ml of a 2.25% solution in 3 ml of normal saline) administered by nebulization oxygen mask. Edema may rebound after treatment with racemic epinephrine so patients must be checked after one hour and overnight admission must be considered in outpatients. Dexamethasone may be prescribed (0.3-0.4 mg/kg) however whether steroids are effective are controversial.<sup>59,60</sup>

In patients with subglottic stenosis treatment with a helium/oxygen mixture (70%/30%) may avoid intubation.

The following tables may guide the treatment of post intubation croup.

#### **Table 1 Croup Score**

	Score				
Criteria	0	1	2	3	
Stridor	None	Only with agitation	Mild at rest	Severe at rest	
Retractions	None	Mild	Moderate	Severe	
Air entry	Normal	Mild decrease	Moderate decrease	Severe decrease	
Color	Normal	Not applicable	Not applicable	Cyanotic	
Level of consciousness	Normal	Restless when disturbed	Restless when undisturbed	Lethargic	

Adapted with permission.<sup>61</sup>

Total Score	Degree	Management
≤4	Mild	Outpatient; give mist therapy
5-6	Mild to moderate	Outpatient if child improve in emergency room/PAR after mist, is greater than 6 months old, and has a reliable family
7-8	Moderate	Admit; give racemic epinephrine
>8	Severe	Admit; give racemic epinephrine, oxygen, and intensive care therapy

#### Table 2 - Treatment of Croup Using Croup Score

Adapted with permission.61

Discharge to home is reasonable if both the physicians and parents are in agreement as to the ability of the parents to care for and observe the child closely at home and return if there are signs of respiratory distress.

#### Bronchospasm

Bronchospasm can be a significant life threatening event at any time in the postoperative period. At increased risk are known asthmatics, children with acute upper respiratory tract infections (especially with intubation)<sup>62</sup> and ex-premature infants with bronchopulmonary dysplasia. It can also be seen in anaphylaxis, histamine release, mucous plugging and aspiration.

Pretreatment with inhaled ipratroprium or albuterol in patients with a URI within the preceding 6 weeks or an active URI in the preceding 7 days did not demonstrate a decrease in airway problems (desaturation, laryngospasm, and bronchospasm).<sup>63</sup> Treatment of bronchospasm includes oxygen, nebulized  $\beta$  agonists (albuterol or metoproterenol 0.5 ml + 2.5 ml saline), terbutaline (MDI or SC), epinephrine 0.01 mg/kg/IV and steroids depending on the severity of the bronchospasm. In severe cases reintubation may be necessary.

## Aspiration

A previous study reported that the incidence of anesthesia related pulmonary aspiration in a university affiliated pediatric hospital has been shown to be 0.10% (twice that reported in adults.<sup>64</sup> In a new report the pediatric groups at risk have been identified in a retrospective review by Warner.<sup>65</sup> The data included 56,138 infants, children and adolescents younger than 18 years of age who underwent 63,180 general anesthetics. Anesthesia for elective procedures comprised 93.5% of all cases. The overall frequency of perioperative pulmonary aspiration was 1:2,632 anesthetics, 24 patients (0.04%). Emergency procedures were associated with a greater frequency of aspiration than were elective procedures (1:373 vs 1:4,544 anesthetics, p <0.001). There was no significant difference in frequency of pulmonary aspiration across different ages or ASA Physical Status Classification. Seven of the 24 patients were ASA PS 1 & 2 children undergoing elective procedures (1 in 8,000 cases); the same ratio as the adult population. None of these aspirations resulted in serious pulmonary complications.

Fifteen of the 24 children did not develop respiratory symptoms within 2 hours. Five of the other nine needed respiratory support with 3 needing mechanical ventilation greater than 48 hours. None died from the pulmonary aspiration. If patients were asymptomatic within 2 hours of the aspiration they were sent home (if they were ambulatory surgery patients) or to a regular nursing unit. If they were symptomatic and had a  $pO_2 > 90\%$  on room air or supplemental oxygen they were sent to a regular nursing unit. All others were admitted to an intensive care area.

The majority of patients with serious aspiration had a bowel obstruction or ileus especially those <3 years of age. Possibly these children have a decreased effectiveness of the lower esophageal sphincter.<sup>66,67,68</sup> Younger children have more air in the stomach due to swallowed air and higher intraabdominal pressure due to crying or gagging. Also the ability to apply effective cricoid pressure may be more difficult in infants and toddlers especially when gagging occurs. These patients who aspirated, gagged or coughed during induction or airway manipulation in which paralysis with muscle relaxants was not present or insufficient to prevent a gag or cough. Unlike adults in which 1/3 of patients aspirated at the end of anesthesia only 1 in 8 aspirations occurred at this time in children, possibly because children are usually awake with good gag reflexes before extubation.

# **Mediastinal Mass**

This is a pediatric anesthesia problem that is a Pandora's box of problems for the anesthesiologist. Usually these masses are tumors that have initial presentation as an anterior mediastinal mass in 1/25,000 children. In children with primary mediastinal tumors 45% were lymphomas (Hodgkins and non Hodgkins) 24% were germ cell tumors (eg. benign, teratomas, malignant teratomas and seminomas), 17% as thymic tumors (hyperplasia and thymomas) and 15% were mesenchymal tumors (tumors of fibrous adipose and muscle tissues, blood vascular origin and vascular).<sup>69</sup> In infants and children 0-2 years, neurologic tumors (benign and malignant) are most common, in children 2-10 years neurologic and lymphatic tumors are equally common and children > 10 years lymphoma and Hodgkins disease are most prevalent.<sup>70</sup>

The anterior mediastinum is bounded anteriorly by the posterior border of the sternum, posteriorly the heart, superiorly by the sternal angle and inferiorly by the fourth thoracic vertebrae. Structures within this space are the trachea and bronchi, superior vena cava (right atrium), main pulmonary artery and aortic arch. Survival of children with tumors in this area depends on early and correct diagnosis. The particular concern includes lymphoblastic (non-Hodgkins) tumors that double in size rapidly and the decision to add radiation therapy depends on the cell type.

Procedures that may need general anesthesia or sedation include CT scan, biopsy of cervical nodes and CVL catheter placement. In addition thoracotomy with removal of the tumor may be indicated if the tumor is not responsive to radiation or chemotherapy.

# **Preoperative Evaluation**

Preoperative evaluation is essential to successful management of these patients. The risk of cardiorespiratory collapse is greatest in untreated tumors. Presenting signs and symptoms are particularly important in two systems - cardiovascular and respiratory. The severity of these findings depends on the size of tumor and location in the mediastinum.

## Respiratory

Tumors may compress or soften long segments of the tracheobronchial tree. The severity of this is due to the weight of the tumor, duration of the pressure and position of the patient. The patients respiratory status may appear normal in the sitting, prone or decubitus but in the supine position with decreased volume of the lung and maximum gravity of the tumor will compress the airway - particularly during expiration when pleural pressure is close to 0. In a series of 50 children 60% had respiratory symptoms - 9 were severe and 13 had marked tracheobronchial compression (35-93%) by CT scan.<sup>70</sup>

## Cardiovascular

Various structures can be compressed or constricted by an enlarging mediastinal tumor. Pericardial encasement may produce constriction or effusion. Compression of atrium or pulmonary artery may be asymptomatic. However a Valsalva effect (which decreases venous return) may be associated with syncopal events. The lesion worsens with the effect of gravity. The aorta is much less involved since the wall has much thicker muscle and the pressure within the lumen can withstand more extrinsic pressure.

Superior vena cava compression presents with facial and periorbital edema, shortness of breath, engorgement of jugular veins and mild CNS symptoms (headache and visual disturbances). These symptoms are worse in the supine position.

# **Preoperative Studies**

Important findings in the history and physical exam may reveal respiratory problems. Tachypnea, orthopnea and sleep disturbances based on position may suggest a compressed airway. Asking patients to assume the supine position may illicit these symptoms.

Chest x-ray, CT scan and MRI are static pictures of airway compression and may not accurately quantitate the degree of compression. Airway obstruction with general anesthesia is likely to occur if the diameter of the trachea is decreased by 50%.<sup>71</sup> Flow volume loops sitting and supine may give a more dynamic assessment of the airway as well as flexible fiberoptic bronchoscopy done under local or sedation. This may not be possible in younger patients.

In a recent report of an ambulatory surgery patient presenting for removal of a neck mass, a chest x-ray 2 weeks prior to surgery was normal. After induction of anesthesia and intubation increased resistance to ventilation occurred and cardiac arrest ensued. Compression of the trachea from an external mass was discovered during bronchoscopy which was diagnosed as T-cell lymphoma with intensive involvement of the supraclavicular fossa, mediastinum and pericardium. Recommendation was due to the rapid growth of these tumors a chest x-ray within 24 hours prior to ambulatory anesthesia in a child with cervical adenopathy should be done.<sup>72</sup>

Cardiovascular evaluation must focus on restricted cardiac output and venous return. Orthopnea, pulsus paradoxus, and SVC syndrome are concerning. If any of these are present a cardiac echo and CT scan of the chest are indicated.

History	Physical	Lab
Respiratory		
Cough	Decreased breathe sounds	Chest x-ray
Cyanosis	Wheezing	Flow volume loops
Dyspnea	Stridor	CT scan
Orthopnea	Cyanosis	
Cardiovascular		
Fatigue	Neck or facial edema	Chest x-ray
Faintness	Jugular vein distention	Echo
Headache	Papilledema	CT scan
Orthopnea	BP instability	
Cough	Pulsus paradoxus	

#### Table 3 - Signs and Symptoms<sup>73</sup>

#### **Anesthetic Management**

In order to safely anesthetize these children we must be aware of any limitations in respiratory and cardiovascular reserves and anticipate further deterioration. We must also have a strategy to prevent cardiorespiratory collapse under anesthesia.

Lymph node biopsy and CVL placement can be done in older children under local or sedation. High-risk patients are those who are short of breath at rest or supine or with syncopal or SVC syndrome.

Younger children or larger surgeries will need general anesthesia. Even if a child appears free of cardiorespiratory symptoms the combination of placing the child supine, induction of anesthesia and positive pressure ventilation may lead to cardiorespiratory arrest.

Patients must be assessed in the supine position when awake but even if there is no distress when anesthesia is induced the patient may deteriorate. Placing in patient in the left lateral decubitus or sitting position may relieve the obstruction. Anesthesia may be induced in the sitting position but it is difficult to intubate and hypotension may occur precipitously. The left lateral decubitus or semi decubitus may be a better position.

Before surgery a planning and strategy conference should be held and this should include discussion of 3 modalities used to shrink the tumor before surgery and general anesthesia: chemotherapy, radiation and steroids. If the tumor can be treated and will decrease in size before surgery this is beneficial. However this must be balanced against obscuring the tissue diagnosis or treating a benign tumor with chemotherapy and radiation because of a wrong diagnosis. Both chemotherapy and radiation can obscure the tissue diagnosis and radiation is ineffective for non Hodgkins lymphoma. One viable therapy option that can shrink the tumor without obscuring the tissue diagnosis is 24-48 hours of steroids which is worth a try.

Ferrari and Bedford<sup>74</sup> concluded that in the absence of life threatening symptoms, general anesthesia could be safely performed in patients without prior radiation or chemotherapy to shrink the mass size. Despite successful outcomes using optimal management strategies the authors describe potentially fatal complications in 44% of the 9 patients who had preoperative symptoms and 1/3 were unable to be extubated at the end of the case due to airway insufficiency.

Premedication should be avoided and intravenous access should be present prior to induction, possibly lower extremity if SVC obstruction is present. Inhalation induction with maintenance of spontaneous ventilation at all times avoiding all muscle relaxants is indicated. In older children the airway may be secured with sedation with local anesthesia via fiberoptic bronchoscopy. An arterial line or a reliable noninvasive BP monitor must be present. If the child deteriorates during the induction turning the patient in the left lateral decubitus position may improve their status. Other options if collapse of the airway occurs is rigid bronchoscopy, pushing the ET tube past the obstruction and upward traction on the sternum to stent open the vessels. Cardiopulmonary bypass or veno-veno bypass may be necessary if either complete airway obstruction or vessel occlusion is anticipated. This must be planned and organized well in advance of the procedure. In addition extra long ET tubes in small sizes should be available to pass through fixed narrowed tracheas.

A recent interesting case report is of a 3 1/2 year old 16 kg child who presented with a large anterior mediastinal mass and asthma. The mass compressed the carina and left mainstem bronchus. Patients oxygen saturation was 76% so he was started on helium (30%) and oxygen increasing his saturation to 96%. He was also given IV steroids prior to induction because of intermittent steroid use for asthma. Patient was induced (after titration with IV midazolam 0.25-0.5 mg, total 1.5 mg) sitting with halothane in heliox maintaining spontaneous ventilation with 3.5 cm H<sub>2</sub>O of CPAP. At an inspired concentration of 2% halothane a number 2 LMA was inserted and 5 cm of CPAP was continued. Halothane 0.7-1.0% end expired was continued in heliox. Local anesthetic (lidocaine 1%) was used to assist in removal of supraclavicular node and CVP placement in external jugular vein via cutdown. Patient was then placed on his left side and LP and bone marrow biopsy were performed. The LMA was removed at the end of the case and patient was returned to recovery breathing heliox and in the semisitting position.<sup>75</sup>

Heliox has 1/3 the density of oxygen and 1/5 that of 30% oxygen and 70% nitrous permitting more laminar gas flow and decreasing resistance in the conducting airways. Heliox is stored in tanks with concentrations of helium 70% and oxygen 30%. It can be attached to the anesthesia machines air inlet but the rotameter will read inaccurately so one must read the FIO<sub>2</sub> with an oxygen analyzer. The lowest oxygen and the highest concentration of helium will give the best clinical effect because the decreased density is directly related to the amount of helium delivered. Patients oxygen saturations are the key to the optimum mixture.

Recovery room personnel must be notified on the effect of position on the patients cardiorespiratory status. Careful assessment planning, maintenance of spontaneous ventilation and proper positioning cannot be over emphasized in these cases.

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