Chapter 17
Respiratory System

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A 3-year-old male, 15 kg, is scheduled for functional endoscopic sinus surgery, bilateral myringotomy and tubes, and nasal ciliary biopsy. Vital signs are BP 90/60 mmHg; P 125 bpm, as palpated by his PMI in the right precordium; and T 37.2 °C. His hemoglobin is 13.0 gm/dL. He has a productive cough and mild expiratory wheezing, does not have a runny nose, and is completing a 14-day course of ampicillin. He uses a beclomethasone (Qvar) inhaler twice daily.

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R.S. Holzman et al. (eds.), *Pediatric Anesthesiology Review*,
DOI 10.1007/978-3-319-48448-8_17

Preoperative Evaluation

Questions

1. Why is this patient wheezing? How would you like to further evaluate the wheezing? Why? How will it affect anesthetic management?

2. What are the implications of dextrocardia for this patient?

3. Is this patient adequately treated with his current medication regimen? What would you add? Why? Any specific implications for anesthetic management? Are there significant neuroendocrine effects to be expected from the use of beclomethasone through the inhalation route?

Preoperative Evaluation

Answers

1. Wheezing, in the setting of asthma or other diseases with bronchoreactivity, is a result of airway narrowing due to inflammation and the accumulation of airway secretions. In chronic conditions, the inflammatory response, particularly in children, may result in tracheo- or bronchomalacia, worsening the wheezing because of the loss of integrity of the cartilaginous matrix of the airways and airway collapse with increased work of breathing. The increased work of breathing exacerbates wheezing by producing more turbulent flow and worsens the underlying metabolic homeostasis of the patient because the harder they work to breath, the less efficient their breathing becomes. They therefore have to work harder and utilize more calories to breathe, worsening their failure to thrive.

   The evaluation of wheezing is initially clinical, with attention to the respiratory rate, utilization of accessory muscles of respiration such as intercostal muscles and neck muscles, and gross movement of the entire rib cage. In addition, intercostal retractions may be seen. Tachypnea is the first sign of respiratory distress unless the patient is already exhausted. The wheezing may be inspiratory, expiratory, or biphasic, and this will lead to a further refined diagnosis of supraglottic, infraglottic, or mixed airway obstruction. A careful history and the patient's responsiveness to bronchodilators will provide further information as to the etiology of the wheezing. There may be cardiovascular consequences as well, including pulmonary hypertension and right heart failure. Anesthetic management should be directed to optimal preinduction medical management through sympathomimetics, anticholinergics, and leukotriene modifiers, with additional steroids as needed. Theophylline is used less often.

2. The dextrocardia may be an isolated finding but is more likely related to Kartagener's syndrome, which is characterized by ciliary dysmotility and abnormal polymorphonuclear (PMN) leukocyte motility. Patients develop chronic otitis media, sinusitis, recurrent respiratory infections, and bronchiectasis. Sterility in males is due to abnormal spermatozoa motility. Situs inversus or dextrocardia is associated with the syndrome.

3. Single drug medical management is probably inadequate; beclomethasone is acceptable as one component of long-term management, but does not completely address all of the mechanisms of chronic airway reactivity or acute exacerbations. Sympathomimetic and anticholinergic therapy may be required for the unstable preoperative patient. An albuterol inhaler and ipratropium may be appropriate additional medications for achieving short-term control. Leukotriene modifiers (montelukast, zafirlukast) will become effective more slowly. The beclomethasone can have systemic effects, although these may not be clinically significant.

4. Should this patient be premedicated? With what? Why? What about a parent-present induction?

Intraoperative Course

Questions

1. How will you set up the ECG monitor? Why? Would an arterial line be a good idea Why/why not? Would you place it preinduction? Is a sidestream capnometer more accurate than a mainstream capnometer for this case? What about transcutaneous CO_2 analysis?

2. Would an anticholinergic be helpful prior to induction? Would you administer? Why/why not? Is this patient at risk for inspissation of secretions as a result? How can you minimize airway reactivity during the induction of anesthesia? How will you know when he is "deep" enough? Should any specific intravenous agents be avoided? Why/why not? Is ketamine better than propofol for induction?

4. Premedication may be helpful for several reasons; it will lessen anxiety (for the parent as well as the child) and will also decrease the secretion of endogenous catecholamines and lower the risk of induction laryngospasm. In addition, lower anxiety will decrease crying and aerophagia, which may increase the chance of aspiration. A benzodiazepine alone should not shift the CO_2 response curve and cause significant respiratory depression. A more complete "preinduction" premed such as a benzodiazepine + ketamine + an anticholinergic orally will serve several purposes: amnesia, anxiolysis, bronchodilation, and a decrease in secretion volume.

Intraoperative Course

Answers

1. The ECG monitor should be configured for dextrocardia with a mirror image of the usual lead placement. An arterial line is probably not necessary for this type of surgery, which should be relatively short, without a large blood loss. On the other hand, if the patient's respiratory status deteriorates as the case proceeds, one should have a low threshold for arterial line placement in this patient with known severely reactive airways. Sidestream and mainstream capnography differ because the sidestream capnograph aspirates a bulk sample of respiratory gases continuously, while the mainstream capnograph detects respiratory gases by an infrared beam transmitted through the gases in the endotracheal tube without bulk gas sampling. The main disadvantage of the mainstream capnometer in children is that the detector is bulky relative to the diameter of the endotracheal tube. Under the drapes, in a warm patient, there is a tendency for the weight of the mainstream capnometer to kink the endotracheal tube. The aspiration side channel in the sidestream capnometer is much lighter and does not kink the tube. Transcutaneous CO_2 analysis can be helpful when skin blood flow can be relied upon; in cold patients and low cardiac output states, it becomes less reliable. General anesthetics can also alter skin blood flow, but children preserve their cutaneous blood flow better than adults, and therefore, in the warm child, transcutaneous CO_2 analysis is relatively reliable.

2. It is worthwhile administering an anticholinergic prior to induction; however, it is important to be aware of the dose administered. The most logical choice is a reasonable dose of glycopyrrolate intravenously (if an IV is present) or inhalation administration of ipratropium bromide. Inspissation, or drying, of secretions is not a significant concern, because anticholinergics change the volume of elaborated secretions but not their water content [1–3]. In general, patients should be deeply

3. What is your choice for anesthetic maintenance? Why? How would the use of sevoflurane for maintenance compare to isoflurane? Would desflurane be a good choice after induction and during maintenance? Is MAC different in this age group? In what way? A colleague suggests that he would avoid nitrous oxide in this patient? Do you agree? The surgeon would like to apply 4 % cocaine to the nose and mucosa of the maxillary antrum for topical analgesia and vasoconstriction? Is this reasonable? How much will you tell him he can use?

4. What muscle relaxant would you choose? Why? What are the varying histamine-release properties of the relaxants you typically use? Which releases the least amount of histamine?

5. Are there advantages to allowing this patient to breath spontaneously? Would you do it? Why/ Why not? What breathing circuit would you choose? Why?

anesthetized as well as adequately treated with sympathomimetics, anticholinergics, and steroids, in order to additionally attenuate their bronchoreactivity. The usual clinical signs should be followed for depth, such as heat rate, blood pressure, and abdominal muscle tone. The eye signs, particularly pupillary dilatation, may be less reliable because of the administration of the sympathomimetics and anticholinergics both causing mydriasis. Intravenous induction agents do not need to be avoided as long as you remember that they are primarily hypnotics and anesthetic depth is more influenced by the volatile agents and narcotics. Ketamine may also act synergistically for depth of anesthesia and sympathomimetic effects. The choice of intravenous induction agent makes relatively less difference as long as an adequate depth of general anesthesia is achieved by one of the above strategies.

3. A balanced anesthetic with a significant component of deep inhalation anesthesia with a volatile agent would probably be best. The opioids are excellent for providing analgesia and rapid attenuation of surgical stress, while the potent volatile agents are excellent bronchodilators because of their effect on airway smooth muscle. At equi-MAC concentrations, the volatile agents are almost all equally efficacious at bronchodilatation. Desflurane, because of its pungency, has been associated with laryngospasm during induction of anesthesia in children, but has certainly been employed successfully as an intraoperative maintenance agent following a sevoflurane induction, with the added benefit that as an insoluble anesthetic, the emergence is more rapid. Ketamine may be a reasonable choice because of the reasons outlined above. Avoiding nitrous oxide allows you to use a higher F_iO_2 and may also be important if the pulmonary vascular resistance is elevated and pulmonary hypertension is present. Cocaine is an excellent topical vasoconstrictor, but it is unnecessary to use a 4 % concentration in this age patient; 1–2 % would be enough for topical analgesia and vasoconstriction, up to 3 mg/kg. This is a relatively uncommon practice in pediatrics, however, where oxymetazoline has been found equally effective.

4. A variety of muscle relaxants can be chosen as long as their histamine release and potential for allergic reaction are minimal. Atracurium (mixed cis and trans enantiomers) and rocuronium are associated with histamine release, but vecuronium and cisatracurium are minimal histamine releasers.

5. There may be an advantage to spontaneous breathing due to less turbulent airflow distal to an area of obstruction, according to the Hagen-Poiseuille equation. For the majority of patients, muscle relaxation with positive pressure ventilation will provide optimal operating conditions and maximize patient safety because of less chance for movement. For severely obstructed patients, the spontaneous breathing strategy may become necessary. I would choose a circle absorption system for the economy of fresh gas flow. An alternative would be a variation of the Mapleson circuits, either a D (Jackson Rees modification of Ayre's T) or an A (Magill system, optimal for spontaneous breathing). However, both of these Mapleson systems require a much higher fresh gas flow, with its attendant loss of temperature and wastefulness of volatile agent, than a circle absorption system, which can have fresh gas flows lowered to within very low flow or closed circuit range.

6. Shortly following the application of 4 % cocaine, the patient's blood pressure is noted to be 180/110, with a heart rate of 220 bpm. As the surgeon begins, it increases to 210/120 with a heart rate of 230. Why? What else is in your differential? What would you do? Should you attempt to slow the heart rate? Why? What will you do to slow the heart rate? Any potential problems with that?

7. A sudden increase in peak airway pressure and desaturation (bronchospasm): the patient desaturates to 87 % over 30 s, and diffuse biphasic wheezing develops with a rise in peak inspiratory pressure to 55 cm H_2O. What would you do first? and next? Why? Where could the problem be? If this is truly bronchospasm, what could you do to fix it? Pharmacologically? Mechanically?

Postoperative Course

Questions

1. When would you extubate? Why? Would you extubate deep? Is this patient at risk for postextubation croup? More so than anyone else? For postextubation laryngospasm? More so than anyone else? Tracheomalacia?

2. What are the risks to this patient of reversal of neuromuscular blockade? Why? What are the alternatives? Would you avoid a reversal agent? Is neostigmine better/worse than pyridostigmine or edrophonium?

6. The direct effect of absorbed cocaine is the etiology of these findings. There are two options – if the cocaine is packed in the nose and is continuing to be delivered, the packing should be removed and suction applied to remove any trace quantities pooled at the mucosal level. Medications that act directly as antihypertensives can be administered if these expectant measures are ineffective, such as sodium nitroprusside (Nipride) in doses ranging from 0.5 to 10 mcg/kg/min and esmolol or propranolol, also in small divided doses and titrated to effect.

7. There should be a logical and rapid progression through a differential diagnosis of mechanical obstruction at the level of the breathing circuit and endotracheal tube and then to the level of the patient's trachea and major conducting airways. Such obstruction can be due to a kinked tube or a tube obstructed with secretions or a mucus plug or the airway obstructed with a mucus plug. If there is no obvious kink in the endotracheal tube, then a suction catheter can be passed through the endotracheal tube until you are convinced that the tube is clear. If mechanical obstruction is ruled out, then diffuse inspiratory and expiratory wheezing can be accounted for by bronchospasm. Albuterol can be delivered via the endotracheal tube, but it may be more effective if the bronchospasm is severe to deliver dilute epinephrine (i.e., 0.1 mcg/kg) intravenously to see if the bronchospasm will break. Anesthetically, the patient should probably be deepened with the volatile agent; ketamine and intravenous glycopyrrolate may also be effective. In order to promote exhalation due to the impaired expiratory flow rate, the expiratory phase of the I: E ratio should be lengthened; otherwise there may be breath stacking.

Postoperative Course

Answers

1. Arguments can be made for deep and awake extubation. I would extubate awake because there is a better assessment of oxygenation, ventilation, and other airway needs, even though some studies have shown a higher saturation with deep extubation [4]. Postextubation croup is probably more related to subglottic irritation from the intubation as well as a loose subglottic basement membrane. Postextubation laryngospasm is a much more likely possibility in any child with irritable airways.

2. Neuromuscular blockade and controlled ventilation often result in less efficient respiratory mechanics because of the cephalad migration of the diaphragm, decrease in FRC, and decreased efficiency of alveolar airflow because of positive pressure ventilation. The reversal of neuromuscular blockade with an anticholinesterase and anticholinergic may result in bronchospasm and increased salivation due to the anticholinesterase.

Additional Questions

Questions

1. What are the anesthetic induction considerations in the patient with pulmonary sequestration? What immediate interventions can be performed therapeutically for deterioration during induction?

2. Is infantile lobar emphysema a surgical emergency? Why/why not? Should the patient be bronchoscoped first? Why/why not? What muscle relaxant would you choose? Why? A colleague of yours suggests the avoidance of nitrous oxide; do you agree?

3. A frightened 8-year-old with a large anterior mediastinal mass presents for supraclavicular lymph node biopsy, and his parents want to know whether he can be asleep for the procedure? What does the answer depend on? Why? Under what circumstances would you want a perfusion team present?

Additional Questions

Answers

1. Pulmonary sequestration is the result of early isolation of pulmonary tissue from the developing lung bud. Sequestration may occur as intralobar or extralobar, depending on whether the abnormal tissue is located within the pleura or outside of it. The abnormal tissue may have cystic and solid areas with mixtures of air, rudimentary air sacs, and bronchi and chronically inflamed areas. Deterioration during the induction of anesthesia may be a result of communication of the sequestration with the respiratory or gastrointestinal tract, resulting in respiratory distress or GI symptoms. Spontaneous breathing may be more optimal in this regard until the communication can be identified, isolated, and ligated along with the blood supply to the sequestration. Extralobar sequestration, although extrapleural, generally presents at an earlier age with recurrent infections. Arteriovenous malformations are also commonly associated.

2. It may or may not be, depending on the timing and severity of presentation. A deficiency of bronchial cartilage, bronchial stenosis, or extrinsic vascular compression from the pulmonary artery may result in congenital lobar emphysema, characterized by overinflation and air trapping in the affected lobe with compression atelectasis of adjacent parenchyma and possible mediastinal displacement. Males are affected more often than females. There is progressive respiratory distress in the newborn period or in early infancy. Rapid deterioration requires urgent surgery, and when accompanied by a mediastinal shift, increased intrathoracic pressure, impaired venous return, and decreased cardiac output may result. The diagnosis is made by chest X-ray when bronchovascular marking is present in a hyperlucent area of the lung. Bronchoscopy should be considered in the older infant if there is a possibility of intraluminal obstruction causing lobar emphysema. Surgical excision in the neonate, however, may be an emergent procedure if the lobe is expanding rapidly. Vigorous positive pressure should be avoided during anesthetic induction, and spontaneous ventilation should be preserved. Nitrous oxide should be avoided, as should muscle relaxants.

3. An anterior mediastinal mass may or may not cause symptoms depending on its encroachment on the tracheobronchial tree and the right heart and pulmonary circulation. Physical diagnosis is not always helpful with regard to the severity of the chest disease, because even patients with more than 50 % airway narrowing may only be symptomatic with orthopnea; the superior vena cava syndrome is relatively rare in pediatric patients [5–7]. Pulmonary function tests may be helpful in demonstrating inspiratory and expiratory compromise. At 8 years of age, a MAC plus good local anesthesia by the surgeon is a very acceptable plan. The patient should also be placed in Semi-Fowler's position for optimal comfort for ventilation and gas exchange. Premedication with oral benzodiazepines may

4. A 14-year-old girl is admitted for evaluation of dysphagia, chest pain, and a weight loss of approximately 7 kg over the course of 3–4 weeks. At 5 years of age, she received an allogenic bone marrow transplant for aplastic anemia that was complicated by the chronic sclerodermoid form of severe graft vs. host disease with generalized skin involvement, muscle wasting, and restrictive lung disease requiring supplemental oxygen (0.5 L/min) and nocturnal biphasic intermittent positive airway pressure (BIPAP) ventilation. She is afebrile, with a heart rate of 110–140 beats per minute and a respiratory rate of 18–24 breaths per minute. Her room air oxygen saturation is 91 %, increasing to 98 % with 0.5 L/min of oxygen. The rigid chest wall shows almost no expansion during inspiration. Pulmonary function tests are:

FVC = 17 % of predicted		
FEV_1 = 18 % of predicted		
FEV_1/FVC = 96 % of predicted		
TLC = 64 %		
RV = 194 %		
RV/TLC = 78 %		
ABG: (0.5 L/min O_2) pH = 7.27, pCO_2	= 95 mmHg, pO_2	= 183 mmHg

Her ECG is unremarkable apart from a sinus tachycardia. Her echocardiogram ruled out right ventricular dysfunction but is suspicious for elevated right ventricular pressure. Her barium swallow demonstrated a web at the cervical esophagus with mild distal narrowing. She is coming to the OR for an endoscopic esophageal dilatation that the gastroenterologist's feel should take about 0.5 h to 45 min.

What are your plans for intraoperative anesthetic technique and postanesthetic care? What are the likely complications and pitfalls?

be a very rational plan and will lessen the patient's anxiety and improve the chances for a successful operating room course. For patients whose risks increase because of greater than 50 % airway encroachment or significant tumor impingement on the pulmonary circulation or the right ventricle, it is not a bad idea to have a rigid bronchoscope available. In the highest risk situations, when the patient has had episodes of syncopy which may be related to a drop in pulmonary blood flow as a result of right heart obstruction, it may be worthwhile to have both groins prepped and draped and have a perfusion team standing by to cannulate and go on to cardiopulmonary bypass immediately.

4. Because of the severe restrictive ventilatory defect and the esophageal obstruction, I would begin the induction of anesthesia in the sitting position while continuing spontaneous ventilation. The patient has severe CO_2 retention, and I would expect a severe decrease in the minute ventilation response to hypercarbia, so it might be worthwhile to consider a respiratory analeptic to shift the CO_2 response curve, such as doxapram [8]. It would probably be necessary to use a small amount of continuous positive airway pressure to maintain the FRC. As the patient lost consciousness, she could be placed in a more supine position, cricoid pressure applied, and the trachea could be intubated at that point. She is indeed a "full stomach," but, in my opinion, the risks of rapid sequence induction and commitment to positive pressure ventilation are outweighed by the advantages of spontaneous breathing with a careful and slow induction of anesthesia with the preservation of spontaneous breathing. Positive pressure may cause interventricular septal shifting that will significantly decrease her stroke volume and cardiac output, so I would be concerned about delivering positive pressure ventilation and committing her to controlled ventilation if it could be avoided. Even a small amount of controlled ventilation may be enough, particularly in this patient, to drive her CO_2 below apneic threshold (that CO_2 required for spontaneous breathing).

   She has a severe restrictive ventilatory defect with incipient changes in the pulmonary circulation and right heart. She has a "cuirass" type of pulmonary physiology which prevents deep breaths either during spontaneous ventilation or most importantly during controlled ventilation [9]. Because of the progressive vascular sclerosis that is characteristic of graft versus host disease, placement of peripheral arterial catheters may carry significant risk of ongoing vascular obstruction, even after the arterial catheter is removed. Notwithstanding her history (and anticipated complication) of elevated pulmonary vascular resistance and right heart problems, these are well understood and changes intraoperatively can be interpreted in light of these findings and supporting evidence from noninvasive monitors such as $ETCO_2$ and pulse oximetry [10].

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