**International Journal of Medical and Health Research**
**ISSN: 2454-9142**
**Received: 05-11-2018; Accepted: 07-12-2018**
**www.medicalsciencejournal.com**
**Volume 5; Issue 1; January 2019; Page No. 22-27**

# Anaesthesia for bilateral intranasal anthrostomy and bilateral partial turbinectomy in a patient with bronchial asthma: A case report

**Chinwe Edith Okoli[1*], Eze Onyegbule Okubuiro[2]**
[1] Consultant Anaesthetist, Federal Medical Centre Umuahia, Nigeria
[2] Consultant Anaesthetist, Marina Specialist Hospital, Port Harcourt, Nigeria

**Abstract**
AC, a 44 year-old male patient with history of bronchial asthma presented with complaints of bilateral nasal blockage, rhinorrhoea and cough of 4 weeks duration. He had suffered recurrences of these symptoms for about 15 years prior to presentation at the hospital. He had been on medications for bronchial asthma for 12 years and the last asthmatic attack occurred one month prior to presentation. Bilateral intranasal anthrostomy (BINA) and bilateral partial turbinectomy were performed under general anaesthesia with muscle relaxation. The postoperative period was uneventful. This case report discusses the anaesthetic management of this patient during this surgery.

**Keywords:** Anaesthesia, anthrostomy, turbinectomy, asthma

## Introduction
Bronchial asthma is a common disease which affects all age groups [1]. About 6.7 percent of all patients who present at University College Hospital Ibadan (UCH) Ibadan for surgery under anaesthesia and who have inter current medical illness are asthmatic [2]. The disease is characterized by episodic or chronic wheezing, cough and a feeling of tightness in the chest as a result of bronchoconstriction [3]. Patient's anxiety can precipitate asthmatic attack in the perioperative period [4]. Presence of foreign body in the airway such as endotracheal tube or oropharyngeal airway and use of drugs that release histamine can also precipitate attacks of bronchospasm in the asthmatic patient [5]. It is therefore vital for the anaesthetist to use perioperative measures that will promote brochodilation. Drugs that may release histamine and maneuvers that might irritate the airway should be avoided.
Otolaryngological procedures pose a unique challenge to the anaesthetist. The proximity of the surgical field to the air-way results in the surgeon competing with the anaesthetist for access to the air-way. This situation might be further compounded by possible air-way compromise from existing ear, nose or throat pathology. Consequently, the best possible surgical access should be created without further compromise to the air-way. The need for adequate monitoring cannot be over-emphasised, and plans should always be in place to deal with intraoperative difficulty.

## Case Presentation
AC, a 44-year-old male carpenter weighing 72 kg presented at the UCH with complaints of bilateral nasal blockage, mucoid nasal discharge and cough all of about 4 weeks duration. These symptoms had recurred many times over the previous 15 years and he had obtained relief by using triprolidine with pseudoephedrine (Actifed) tablets and xylometazoline (Otrivin) nasal drops. He had also suffered recurrent attacks of bronchial asthma in the previous 12 years and had been admitted on three occasions in a peripheral hospital. Each episode was characterized by cough, dyspnoea, wheezing and itching in the ears. The last attack occurred about one month before his presentation. The attacks were often precipitated by inhalation of dust or smell of cooking oil. He had been on regular treatment with oral salbutamol 4 mg three times daily and chlorpheniramine (piriton) one (4 mg) tablet twice daily. Occasionally he used puffs of salbutamol inhaler to gain relief from the asthmatic attacks. Systemic review revealed no other symptoms. He was married and had four children. He neither consumed alcoholic drinks nor smoked cigarettes.
Physical examination revealed a calm middle-age man who was not febrile, pale nor jaundiced. His respiratory rate was 18 breaths per minute, chest expansion was equal bilaterally and air entry into both lungs was good. No wheeze was detected during inspiration or expiration. He had bilaterally engorged inferior turbinates with mucoid discharge. No abnormality was found in the ear and throat. Pulse rate was 88 beats per minute, regular and of good volume and blood pressure was 130/80 mmHg. The heart sounds were normal with no added sounds. A diagnosis of chronic allergic rhinosinusitis in a known asthmatic was made.
An X-ray of the paranasal sinuses showed gross thickening of the maxillary anthral mucosa bilaterally with haziness of the sphenoidal sinus. Differential white blood cell count showed lymphocytosis (63%). The results of other investigations were as follows: Haemoglobin- 170g/L (normal), Urea/ electrolytes- within normal range, ECG- Normal sinus rhythm, Chest X-ray- Normal chest radiograph,

**Pulmonary Function Test**

**Table 1**

| | Predicted | Test Result | % |
|---|---|---|---|
| **Pre-bronchodilation** | | | |
| FVC (L) | 4.2 | 4.0 | 95.2 |
| $FEV_1$ (L) | 3.4 | 2.9 | 85.3 |
| $FEV_1$/FVC% | 81 | 72.5 | |
| PEFR (L/min) | 559 | 470 | 84 |
| Post-bronchodilation | | | |
| FVC (L) | 4.20 | 4.0 | 95.2 |
| $FEV_1$ (L) | 3.40 | 2.95 | 86.76 |
| $FEV_1$/FVC% | 81 | 73.8 | |
| PEFR (L/min) | 559 | 473 | 84.5 |

**Anaesthetic Management**

At preoperative review, the history, physical examination and investigations were as earlier reported. Airway assessment revealed Mallampati class III category and physical status based on the American Society of Anesthesiologists (ASA) classification was class II. The likely presence of a nasal pack during emergence from anaesthesia which would require him to breathe through the mouth was explained. Informed consent was obtained from the patient for anaesthesia and surgery while salbutamol inhalers, ampoules of 2.5% solution of aminophylline and vials of hydrocortisone were provided for use in the preoperative period. Oral intake was stopped about 8 hours before surgery. A request was made for two units of cross matched and screened fresh whole blood for intraoperative use. Oral salbutamol 4 mg and oral chlorpheniramine 4 mg were prescribed to be administered orally with a sip of water on the morning of surgery.

Prior to the arrival of the patient in the theatre, routine checks were carried out on the anaesthetic machine. Equipment for difficult intubation including a range of non-kinkable cuffed endotracheal tubes of sizes 7.0, 7.5, and 8.0 were selected and tested for leakage. The tubes were also lubricated with lidocaine gel. A size 3 laryngeal mask airway was lubricated and kept on the anaesthetic machine. Drugs for anaesthesia including aminophylline, lignocaine, hydrocortisone and adrenaline were drawn up in labelled syringes.

On arrival in the theatre, the pre-induction blood pressure was 120/70 mmHg and pulse rate was 80 beats per minute. The monitor also showed normal ECG waves and arterial oxygen saturation ($SpO_2$) of 98% while the patient was breathing room air. A stethoscope attached to his precordium revealed normal heart sounds and normal breath sounds. A 16-gauge cannula was placed in a vein on his left forearm through which an intravenous infusion of normal saline was started.

The patient was preoxygenated for 5 minutes during which intravenous atropine, 0.6 mg, intravenous pethidine 50 mg, intravenous plain lignocaine 70 mg, and 2 puffs (200 mg) of salbutamol aerosol were administered. Anaesthesia was induced with intravenous ketamine 150 mg given slowly over 60 seconds. Intravenous suxamethonium chloride 100 mg was administered to facilitate laryngoscopy and intubation of the trachea with a size 8.0 mm non-kinkable cuffed orotracheal tube. Intubation was carried out when adequate muscle relaxation had been obtained. The cuff of the orotracheal tube was inflated and it was secured in place with adhesive tapes when correct placement had been confirmed by auscultation of the lungs.

Ventilation was manually-controlled and anaesthesia maintained with 100% oxygen using a fresh gas flow of 6 litres per minute through a Bain's breathing system. Halothane 0.75% was added to the gas mixture. Six milligrams (6mg) of pancuronium bromide was administered intravenously to achieve muscle paralysis and 2 additional boluses of 1 mg each were administered before the end of operation. The oropharynx was packed with wet sterile gauze. Additional 50mg of pethidine was administered intravenously, 40 mins after the initial dose. The patient's condition remained stable throughout the 95 minutes of intraoperative period. The blood pressure was in the range of 120-140mmHg systolic and 60-100mmHg diastolic. The heart rate ranged between 70 and 120 beats per minutes. The ECG waves remained normal while the $SpO_2$ was between 98% and 100%. Continuous auscultation with the precordial stethoscope showed normal breath sounds and heart sounds.

Intraoperative blood loss assessed by visual inspection of soaked swabs, drapes and contents of the suction bottle was 500 ml. A total of 1.5 litres of 0.9% saline was infused. Blood transfusion was not found necessary. At the end of the surgical procedure, halothane was discontinued. The oropharyngeal pack was removed and the oropharynx cleared of secretions with a sterile catheter. Residual neuromuscular blockage was reversed with intravenous administration of 1.2 mg atropine followed by 2.5 mg of neostigmine. The patient was turned on to a left lateral position. When he was fully awake and spontaneous ventilation was adequate, the endotracheal tube was removed. Oxygen (100%) was administered by face mask for five minutes after termination of anaesthesia. Further observation showed that no bronchospasm or airway obstruction had occurred as he breathed through his mouth. He was transferred to the recovery room in the left lateral position on a trolley.

**Surgery and Findings**

A bilateral intranasal anthrostomy and bilateral partial turbinectomy were done and operative findings included; engorged inferior turbinates, mucoid nasal secretions, dry anthral aspirate and minimal collection of anthral mucus.

**Postoperative Management**

In the recovery room, oxygen was administered by a disposable face mask at a flow rate of 2 litres per minute for 30 minutes. He was nursed in the left lateral position and infused with 5% dextrose saline at the rate of 1 litre 8 hourly. Spontaneous ventilation remained adequate. His clinical condition remained stable and he was discharged to the ward after about one hour stay in the recovery room. Postoperative analgesia was achieved with intravenous tramadol 100mg 8 hourly in the first 48 hours and thereafter, tramadol tablet 100 mg was continued orally every 8 hours for the next 3 days. At the ear, nose and throat ward, he received chest physiotherapy as part of his postoperative treatment. He had an uneventful recovery and was discharged home in good health on the 7$^{th}$ day after surgery.

**Discussion**

The epidemiology of bronchial asthma has been changing worldwide. Increases in prevalence, mortality rates, and hospitalization rates have been reported [6, 7, 8]. The reasons for these increases are not clear but these certainly affect the practice of anaesthesia [9]. The fundamental cause of bronchial asthma is still unknown despite intensive research. However, three abnormalities are present: airway obstruction that is at least partially reversible, airway inflammation and airway hyper-responsiveness to a variety of stimuli [10]. Some of these stimuli will have little or no effect on non-asthmatics with normal airway [5, 11]. The airway obstruction is caused by smooth muscle spasm, mucous plugs and bronchial oedema. Asthma may be classified into two types: Extrinsic where an external allergen is demonstrable as in this case and intrinsic where there is none. Intrinsic asthma tends to occur in adults, is more chronic and continuous and often requires long term steroid therapy [1, 12]. Chronic airway inflammation involving many cell types and inflammatory mediators accompanies the bronchial hyper-responsiveness of bronchial asthma [13]. However, the precise relationship of the inflammatory cells and their mediators to airway hyper-reactivity is not fully understood.

Sinus infection may substantially contribute to the frequency and severity of asthma attacks. Sinusitis and asthma are both inflammatory diseases that arise or aggravated when mucociliary clearance is impaired [14]. According to the Asthma and Allergy Foundation of America, half of all people with moderate to severe asthma also have chronic sinusitis [15]. Sinusitis and asthma have common triggers which include cold, viral infection, air pollution, airborne allergens and dry or cold air. A multitude of factors associated with anaesthesia may provoke bronchospasm in a bronchial asthma patient. Local irritation from inhalational anaesthetics or foreign bodies in the airway such as endotracheal tubes or oropharyngeal airways and suction catheters predispose to bronchospasm [5]. Histamine release from anaphylactic reactions, blood transfusion reactions, d-tubocurarine, metocurine, atracurium and barbiturates may include bronchospasm. Stimulation of the upper or lower airways by blood or vomitus, as well as surgical stimulation may result in bronchospasm. Drugs that act by increasing acetylcholine concentration, such as neostigmine may cause bronchospasm. Despite this theoretical possibility, reversal of neuromuscular blockade is not usually associated with exacerbation of bronchoconstriction. When atropine, a mild bronchodilator is used prior to administration of cholinesterase inhibitor, it may help protect against the potential for bronchospasm.

Preoperatively, evaluation of asthmatic patients should determine the recent course of the disease and to ascertain that the patient is in optimal condition. Patients with bronchial asthma presenting for a surgical procedure may be classified into three groups preoperatively based on data from history, physical examination and laboratory investigations [16]. Group 1 includes patients with a history of wheezing but who are not presently wheezing and have not had recent bouts of bronchospasm. They are not taking medication and clinical history, physical examination and pulmonary function tests show little or no abnormality. Such patients require no additional work-up or intervention prior to anaesthesia and surgery. Group 2 patients are those who are free of wheezing but who report recurrent bouts of bronchospasm. They may be taking prophylactic bronchodilator as noticed in this case. If their $FEV_1$ and other lung function tests results are greater than 50% of predicted value or unchanged from previous testing, present medications are continued and anaesthesia might be administered as scheduled. If these pulmonary function tests are less than 50% of predicted or have deteriorated from previous testing, such patients are reclassified into group 3. Patients in group 3 are those who are present with wheezing, or clinical deterioration from normal status. Their surgery should be delayed until bronchospasm is optimally controlled. Pulmonary function testing should be performed with and without bronchodilators in order to evaluate the potential for reversibility. Airway obstruction is considered reversible if a 15% increase in tests results is achieved with bronchodilators. An increase in $FEV_1$ of 15% or more following Bronchodilation treatment suggests that benefits may be achieved from additional bronchodilator therapy in the perioperative period.

Asthma is characterized by recurrent airway obstruction and inflammation. The difference between anaesthetizing an asthmatic patient with clearly audible preoperative wheezing and one without wheezing may be a potentially life-threatening anaesthetic experience versus a totally uneventful one. Preoperative improvement of patient's condition is mandatory. Two major groups of drugs may be used in the management of asthmatic patients. Bronchodilators are employed to reverse or prevent airway obstruction and inhibitors of inflammation are often indicated. The bronchodilators include sympathomimetic, theophylline derivatives and anti-cholinergic while the anti-inflammatory drugs include corticosteroids, sodium cromoglycate (cromolyn sodium), nedocromil and leucotriene modifiers (montelukast, zafirlukast, pranlukast, zilueton).

Corticosteroids are potent drugs that are useful in the treatment of chronic asthma as well as during acute attacks [17]. Their mechanism of action is not completely understood, but all preparations are thought to act in the same way by modifying and reversing the asthmatic process.[16] Hydrocortisone is the steroid preparation most frequently used in the perioperative period [10]. Methylprednisolone can also be used. Aerosols of corticosteroids have a similar onset of action and duration of activity and offer no additional benefit in the acute situation. Cromolyn sodium acts by preventing the release of mediators from mast cells and perhaps other cells as well [18]. It is valued solely as prophylaxis and has little use in the acute management of bronchospasm. Although chemically unrelated, Nedocromil is more potent but shares similar clinical profile with cromolyn sodium [18]. Compelling evidence supports the role of the cysteinyl leukotrienes in the pathophysiology of asthma [19]. Leucotriene modifiers (montelukast, zafirlukast, pranlukast, zilueton) act by antagonizing cysteinyl receptors [18].

Sympathomimmetic drugs are agonists at beta-adrenergic receptors have been used in the treatment of asthma. They produce relax bronchospasm by stimulating adenylcyclase to catalyse the production of cyclic adenosine monophosphate (c-AMP) which inhibits release of bronchoconstrictor substances. Examples of beta agonists include adrenaline,

isoprenaline, ephedrine, terbutaline and albuterol (salbutamol). Selective beta$_2$-agonists are preferable to less selective beta-adrenergic agonists with both beta$_1$ and beta$_2$-agonists effects [20]. Isoprenaline, a non-selective beta receptor agonist is often associated with undesirable tachyarrhythmias. Selective beta$_2$- agonists such as salbutamol, terbutaline and feneterol differ from isoprenaline in having increased duration of action, greater beta-selectivity and fewer side effects. Longer acting beta2-adrenergic agonists that are inhaled such as formoterol [21]. and salmeterol are at different stages of clinical trials.

Aminophylline, a water-soluble salt of theophylline is commonly used in the management of bronchospasm [18]. Theophyllines are phosphodiesterase inhibitors that inhibit c-AMP breakdown into 5-AMP thereby causing increased c-AMP concentration. C-AMP inhibits bronchoconstrictor substance release. The therapeutic range for serum concentration of aminophylline is 10-20mg per ml. Levels higher than this are associated with features of aminophylline toxicity such as tachyarrhythmias, hypotension and convulsions [9]. ECG monitoring to detect tachyarrhythmias is usually beneficial in the elderly. Ipratropium bromide, a quartenary anticholinergic compound has been released for clinical use as a metered dose inhaler [18]. It prevents the cellular build-up of c-GMP within the bronchiolar smooth muscle. Ipratropium bromide is not indicated in the treatment of severe acute bronchospastic episodes because peak bronchodilating action occurs after 30-90 minutes following its administration.

Pre-medication for asthma patients should consist of anti-cholinergic drugs such as atropine to block vagal reflex-induced bronchospasm and a sedative agent. Pethidine and promethazine are considered satisfactory [1]. An inhaled beta$_2$agonist such as salbutamol should be administered just before induction of anaesthesia to minimize the risk of bronchospasm [4, 22]. Local anaesthetic agents administered either systemically or as aerosols have been used to treat bronchospasm for many years. The mechanism involved includes direct effects on airway smooth muscle, inhibition of mediator release and interruption of reflex arcs. Plain lignocaine given prior to intubation is often useful in preventing reflex bronchoconstriction provoked by instrumentation [9].

Anaesthesia for bilateral intranasal anthrostomy and partial turbinectomy in a patient with bronchial asthma will require a smooth anaesthetic technique and a clear airway as coughing and staining will result in venous congestion which may persist during surgery and cause increased bleeding. Measures to maintain a clear airway are essential as partial obstruction of the airway may lead to hypoxaemia, hypercapnia and unduly light anaesthesia [23]. Anaesthetic depth should be adjusted to stimulation to avoid bronchospasm. Any particular choice of anaesthetic agent is not as important as achieving deep anaesthesia prior to intubation and surgical stimulation. Ketamine produces bronchodilation through mechanisms that might include inhibition of vagal pathways, direct relaxation of smooth muscle and increase in endogenous catecholamines which relax the airway via beta$_2$-adrenoceptors on airway smooth muscle [22]. It is therefore beneficial in asthmatic patients. Thiopentone sodium is associated with dose related broncho-constriction [24]. Thiopentone in doses used clinically does not reliably protect against reflex-induced brochospasm. On the other hand, thiopentone is not necessarily contra-indicated in asthmatic patients, particularly if instrumentation of the airways is delayed until adequate anaesthesia has been accomplished with inhaled agents. It has been suggested that if tracheal intubation is accomplished with the patient "deeply anaesthetised" then the choice of an induction agent becomes unimportant [9]. Propofol compared to thiobarbiturate or oxybarbiturate has less incidence of wheezing during induction [23]. It is widely documented that propofol has properties beneficial to patients with asthma. Pederson et al. showed that propofol directly relaxes guinea pig tracheal tissue and was more potent than ketamine [25]. Cigarini et al. found that propofol prevented fentanyl-induced bronchoconstriction in surgical patients [26]. Pederson reported that sedation doses of propofol inhibited postoperative bronchospasm in two patients with hyperreactive airway disease [25]. Propofol (where available) is the induction agent of choice for asthmatic patients.

Suxamethonium chloride stimulates muscarinic receptors and release of histamine which may provoke bronchospasm in asthmatic patients [22]. However, the use of suxamethonium was indicated in this case because of anticipated difficult intubation. It has rapid onset of action and provides optimal muscle relaxation which is necessary for smooth intubation. Whenever possible, endotracheal intubation in the asthmatic patient should be avoided since instrumentation of the airway is a major trigger for wheezing during anaesthesia [28]. To this end, local anaesthesia or general anaesthesia with face mask is preferable. Endotracheal intubation with a cuffed tube to ensure a patent and protected airway was however unavoidable in this case as the site of surgery was the upper airway. A pre-formed oral tube may be used when a non-kinkable tube is not available as it moves the tube connection to the breathing system away from the operating field [28].

Halothane may prevent the development of brochospasm by blocking airway reflexes, directly relaxing smooth muscle of the airway and inhibiting mediator release [22]. It is therefore an inhalational agent of choice in asthmatic patients. Isoflurane and enflurane have been shown in an earlier study to be as effective as halothane at equivalent MAC multiples in dogs at antagonizing vagally mediated bronchoconstriction [29]. However, an animal (dog) study of histamine-induced bronchospasm found that at doses less than 1.7 MAC, halothane is a better bronchodilator than isoflurane [30]. Non-depolarising muscle relaxant associated with histamine release like curare, atracurium, and mivacurium are often contraindicated. Most of the commonly used neuromuscular blocking agents are preferable hence the use of pancuronium bromide in this patient. Blood, secretions and vomitus trickling down the upper airway may provoke bronchospasm in asthmatic patients [5]. Packing of the pharynx with wet gauze after orotracheal intubation was intended to prevent this since the surgery involved the upper end of the airway.

Controlled studies involving narcotics in asthmatic patients undergoing anaesthesia are rare. In the few *in vitro* studies found in the literature, the effects of opioids on airway reactivity in asthmatic subjects appear too small and inconsistent [26]. However, morphine administration is

associated with "increases in plasma levels" of histamine.[31] Asthmatics react by bronchoconstriction to very low concentrations of histamine; narcotics that have minimal histamine-releasing properties such as pethidine and fentanyl would be preferable [9]. Pethidine was administered just before induction of anaesthesia to augment the analgesic and narcotic effects of other drugs used. The clustering of the surgeon, scrub nurse and instruments about the patient's head usually forces relocation of the anaesthetist and anaesthetist's equipment towards the foot of the operating table. The use of Bain's breathing system with regulatory valve close to the anaesthetic machine was therefore desirable. Inadvertent extubation, migration of the endotracheal tube and disconnections in the breathing system is common during ear, nose and throat (ENT) procedures [28]. This may not be easily detected after the patient has been draped. Precordial stethoscope can be used for continuous monitoring of air entry into the lungs. Inadequate filling of the reservoir bag during ventilation may also signal a discontinuity in the breathing system and should be monitored. Adequate intraoperative monitoring of the asthmatic patient is mandatory. Precordial stethoscope may also be used to detect intraoperative wheezing in asthmatic patients. While life threatening bronchospasm under anaesthesia is relatively uncommon, mild wheezing and elevation of peak inspiratory pressure occurs more commonly [28]. The pulse oximeter is also indicated as it may show a dropping arterial oxygen saturation when bronchospasm occurs intra operatively. This is usually due to airway closure by bronchospasm and secretions and under-ventilation of perfused alveoli. The end-tidal carbon dioxide tension ($ETCO_2$) may show a decrease while the $PaCO_2$ increases during intraoperative bronchospastic episodes. This is because differences in resistance result in over-distension of some lung units, over-ventilation of others, and an overall increase in ventilation-perfusion mismatch. Over-distended alveolar may not be perfused at all, especially in the face of hypotension resulting in large increases in dead space [30]. The ECG as used in this case can detect the presence of arrhythmias which occur commonly during operations on the face [23]. Arrhythmias may also occur if excessive amounts of adrenaline are absorbed from the soaked gauze used to pack both nasal cavities especially while halothane was being used. However, no arrhythmias were observed, probably because the dose of adrenaline used was within the safe limit as suggested by Katz and Katz [32].

When brochospasm occurs intraoperatively, the first remedial step is to deepen the level of anaesthesia and increase the $FiO_2$ [33]. This is usually effective because the most common cause of asthmatic attack during surgery is inadequate anaesthesia [33]. Blood pressure is monitored to ensure normotension as anaesthesia is deepened by increasing the concentration of the inhalational agent. An incremental dose of ketamine could maintain blood pressure, rapidly deepen anaesthesia and avoid the problem of delivering inhaled anaesthetic to a patient with poor ventilation. The cardiovascular responses to ketamine administration are secondary to central sympathetic stimulation or to the inhibition of catecholamine uptake in postganglionic neurons. Since halothane sensitizes the myocardium to endogenous and exogenous catecholamines, simultaneous use of halothane and ketamine may provoke cardiac arrhythmias. Oxygenation can be improved by increasing the concentration of inhaled oxygen. Any mechanical obstruction of the airway should be relieved by passing a catheter through the endotracheal tube (ETT) to suction secretions and to determine whether there is any other mechanical obstruction or kinking of the tube. The cuff of the ETT can be deflated; the tube moved back 1cm or 2cm and the cuff re-inflated. This will relieve stimulation of the carina by the tip of the ETT. The 'gold standard' of treatment of intraoperative bronchospasm is inhalation of a $beta_2$agonist such as salbutamol; unlike theophyllines, salbutamol induce bronchodilation even in the presence of adequate halothane anaesthesia as demonstrated in both animal models and humans [4, 3, 4]. However, recommended therapy also included intravenous aminophylline [21]. Intraoperative bronchospasm was not observed in this patient and these measures did not become necessary. Intraoperative blood loss of 500 ml was replaced with 1.5 litres of crystalloid fluids. Although two units of fresh whole blood had been cross-matched intraoperatively, blood transfusion was not found necessary.

Reversal of non-depolarising neuromuscular blockade with neostigmine can provoke airway constriction in bronchospastic-prone patients. Anticholinergic agents such as atropine or glycopyrrolate successfully prevent bronchospasm due to these cholinesterase inhibitors. It may be prudent to produce effective cholinergic blockade prior to administration of the anti-cholinesterase drug if the patient can tolerate tachycardia. Tramadol has been advocated as an analgesic without respiratory depression when used parenterally [35]. It has been found to be well tolerated and equipotent to pethidine in the treatment of postoperative pain following abdominal surgery [35]. Its use is therefore indicated. Although narcotic analgesics that do not have histamine releasing properties may be used for asthmatic patients, it should be ensured that respiratory depression does not occur postoperatively when they are used [9]. Commencement of oxygen therapy and chest physiotherapy on this patient in the postoperative period probably helped to prevent hypoxaemia and pulmonary postoperative complications. There is no loss of carbon dioxide response in asthmatic patients and high inspired oxygen concentrations are well tolerated. For patients with co-existing chronic obstructive pulmonary disease (COPD) the hypoxic drive might be removed by increased $FiO_2$. It is important to watch the patient's respiration very carefully during the oxygen therapy and use Venturi mask with FiO2 of 0.24-0.4 to administer controlled oxygen therapy to patients with COPD [36].

## Conclusion

Bronchial asthma is a syndrome characterized by tracheobronchial tree hyper-responsiveness to a variety of stimuli. This results in episodic partially reversible bronchconstriction. Whereas many of these stimuli occur during the perioperative period, the main challenges in the anaesthetic management of these patients is the prevention and prompt relief of bronchospasm and its sequelae of hypoxaemia and hypercapnia during this period. Satisfactory management for surgical procedures in patients with bronchial asthma can be accomplished if the pathophysiology of the disease is understood and conditions which precipitate

bronchoconstriction in the perioperative period are avoided.

**References**


1. Grant IS, Nimmo GR. Intercurrent disease an anaesthesia in Aitkenhead A.R., Smith G. (eds). Textbook of Anaesthesia. 3rd Edition. Churchill- Livingstone, Edinburgh, 1996, 651-686.
2. Soyannwo OA, Bamgbade OA, Odutola OO: Medical Diseases and Anaesthesia. Afr J Anaesth intens care. 1996; 2:51-56.
3. O'Donnell RA, Frew AJ. Is there more than one inflammatory phenotype in asthma? Thorax. 2002; 57(7):566-568.
4. Bishop MJ, Cheney MD, Frederick W. Anesthesia for Patients with Asthma: Low Risk but Not Any Risk. Anesthesiology. 1996; 85(3):455-456.
5. Kumeta Y, Hattori A, Mimura M, Kishikawa K, Namiki A: A survey of perioperative bronchospasm in 105 patients with reactive airway disease. Masui J. 1995; 44:396-401.
6. McFadden ER. Acute Severe Asthma. Am J Respir Crit Care Med. 2003; 168(7):740-759.
7. Weiss KB, Gergen DK. Changing patterns of asthma mortality: identify target population at high risk. JAMA. 1990; 264:1683-7.
8. Mitchell EA. International trends in hospital admission rates for asthma. Arch Dis Child. 1985; 60:376-378.
9. Hirsham CA. Perioperative management of the asthmatic patient. Can J Anaesth. 1991; 38:R26-R32.
10. Markey J. Immuno pharmacology of asthma. Immunol Today. 1993; 14:3179.
11. Pizov R, Brown RH, Weiss YS, Baranov D, Hennes H, Baker S, *et al*. Wheezing during induction of general anesthesia in patients with and without asthma. Anesthesiology. 1995; 82:1111-1116.
12. Douwes J, Gibson P, Pekkanen J, Pearce N**.** Non-eosinophilic asthma: importance and possible mechanisms. Thorax. 2002; 57(7):643-648.
13. Fahy JV. Eosinophilic and Neutrophilic Inflammation in Asthma: Insights from clinical Studies Proceedings of the ATS. 2009; 6(3):256- 259.
14. Ikeda K, Tanno N, Tamura G. Endoscopic sinus surgery improves Pulmonary function in patients with asthma associated with chronic sinusitis. Ann Otol Rhinol Laryngol. 1999; 108(4)355-359.
15. Baroody FM. Mucociliary transport in chronic rhinosinusitis. Clin Allergy Immunol. 2007; 20:103-119.
16. Warner DO, Warner MA, Barnes RD, Offord KP, Schroeder DR, Gray DT, *et al*. Perioperative respiratory complications in patients with asthma. Anesthesiology. 1996; 85:459-67.
17. Maneechotesuwan K, Essilfie-Quaye KS, Kharitonov SA, Adcock IM, Barnes PJ**.** Loss of Control of Asthma Following Inhaled Corticosteroid Withdrawal Is Associated With Increased Sputum Interleukin-8 and Neutrophils**.** Chest. 2007; 132(1):98-105.
18. Falase AO, Akinkugbe OO. Respiratory system. In: a compendium of clinical medicine. Ibadan, 2003, 173-17
19. Thompson C, Cloutier CY, Bosse C, Poisson P, Larivee P, McDonald P, *et al*. Signaling by the Cysteinyl-Leukotriene Receptor 2: Involvement in chemokine gene transcription. J Biol Chem. 2008; 283:4.
20. Gordon JR, Swystun F, Li X, Zhang BE, Davis P, Hull VA, *et al*. Regular salbutamol use increases CXCL8 responses in asthma: relationship to the eosinophil response. Eur Respir J. 2003; 22(1):118-126.
21. Maneechotesuwan K, Essilfie-Quaye S, Meah C, Kelly S, Kharitonov A, Adcock IM, Barnes PJ. Formoterol Attenuates Neutrophilic Airway Inflammation in Asthma. Chest, 2005; 128:4.
22. Bishop MJ. Bronchospasm; avoiding an anaesthetic disaster. ASA Refresher course in Anesthesiology. 1991; 19:15-27.
23. Eames WO, Rooke GA, Wu RS, Bishop MJ. Comparison of the effects of etomidate, propofol, and thiopental on respiratory resistance after tracheal intubation. Anesthesiology. 1996; 84:1307-1311.
24. Pederson CM, Thirstrup S, Nielsen-Kudst JE. Smooth muscle relaxant effects of propofol and ketamine in isolated guinea-pig trachea. Eur J Pharmacol. 1993; 238:75-80.
25. Pederson CM. The effect of sedation with Propofol on postoperative bronchoconstriction in Patients with hyperactive airway disease. Intens Care Med. 1992; 18:45-46.
26. Cigarini I, Bonnet F, Lorino AM, Harf A, Desmonts JM. Comparison of the effects of fentanyl on respiratory mechanics under propofol or thiopental anesthesia. Acta Anaesthesiol Scand. 1990; 34:253-256.
27. Bishop MJ. New perspectives on the patient with reactive airways. IARS Review course lectures, 1997, 14-18.
28. O'Carroll TM. Anaesthesia for ENT Surgery in Aitken head AR Smith, G, text book of Anaesthesia 3rd Edition. Churchil Living stone, Edinbugh, 1996, 186-418.
29. Vetterman J, Beek KC, Lindahl SHE. Actions of enflurane isoflurane, vecuronium, atracurium and pancuronium on pulmonary resitance in dogs, Anesthesiology. 1988; 69:688-695.
30. Brown RH, Zerhouni EA, Hirshman CA. comparism of low concentration of halothane and isoflurane as bronchodilators. Anesthesiology. 1993; 78:1097-1101.
31. Roso CE, Moss J, Philbin DM, Savarese J. Histamine release during morphine and fentanyl anaesthesia. Anesthesiology. 1992; 56:93-96.
32. Katz RL, Kartz GJ Surgical infiltration of pressor drugs and their interacation with volatile anaesthetics. Br J Anaesth. 1966; 38:712-718.
33. Gal TJ. Bronchospasm: Physiology and pharmacological insights. IARS review course lectures, 1991, 1- 6.
34. Tobias JD, Kubos KL, Hirsham CA. Aminophylline does not attenuate histamine-induced airway constriction during halothane anaesthesia. Anesthesiology. 1990; 72:105-110.
35. Houmes RJM, Vocts MA, Verkaak A. Efficacy and safety of tramadol versus morphine for moderate and severe postoperative pain with special regards to respiratory depression. Anesth Analg. 1992; 75:510-514.
36. Shapiro BA, Kacmarck RM, Cane RD (eds). Clinical application of respiratory care. 4th edition. Mosby-Yearbook. St Louis, 1991, 421-432.