A case of persistent bronchospasm after anesthesia induction

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ABSTRACT

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We report a case of persistent bronchospasm after anesthesia induction. The case refers to an elective surgery of an ulcerous formation in the intergluteal cleft. Bronchospasm is not an unusual event in the immediate intubation period, especially in patients with respiratory disease, but in most cases resolves uneventfully. In this patient, despite thorough treatment in the operation room, ausculatory findings remained unchanged, with progressive worsening of arterial blood gases. After this event, the surgery was postponed and the patient was transferred to the ICU for further management. In this article we describe the steps that were taken in order to manage this adverse event and ensure patient's safety and successful outcome.

INTRODUCTION

Bronchospasm is the clinical feature of exacerbated underlying airway hyperreactivity. Hyperreactive airway refers to patients who exhibit heightened airway reactivity to normal or lower level of physical, chemical, or pharmacological stimulus to the airway. Hyperreactivity is characterized by intermittent bouts of exaggerated airway narrowing[1]. In the perioperative period, it usually arises during anesthesia induction but may also be detected at any stage of the anesthetic course.

Bronchospasm encountered during the perioperative period, especially after anesthesia induction and intubation, may involve an immediate hypersensitivity reaction including IgE-mediated anaphylaxis or a nonallergic mechanism triggered by factors such as mechanical (i.e. intubation-induced bronchospasm) or pharmacologic-induced (via histamine-releasing drugs such as atracurium or mivacuri-

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um) bronchoconstriction in patients with uncontrolled underlying airway hyperreactivity[2,3]. Chest auscultation should be done to confirm wheezing, whereas decreased or absent breath sounds suggest critically low airflow. The differential diagnosis includes inadequate anesthesia, mucous plugging of the airway, esophageal intubation, kinked or obstructed tube/circuit, and pulmonary aspiration. Unilateral wheezing suggests endobronchial intubation or an obstructed endotracheal tube by a foreign body (such as a tooth, mucous plug etc.). If the clinical symptoms fail to resolve despite appropriate therapy, other etiologies such as pulmonary edema or pneumothorax should also be considered[4].

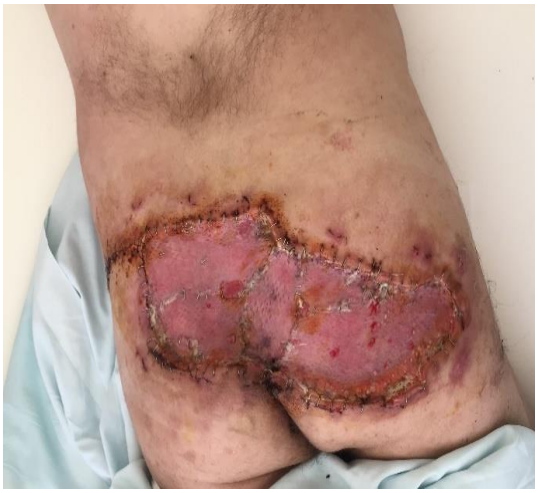
Predisposing factors for the development of perioperative bronchospasm are acute or chronic airway diseases. The acute causes include upper and lower respiratory tract infections due to viral or bacterial infections, which commonly occur in children, but may also occur in adults. Chronic diseases such as allergic rhinitis, chronic bronchitis, emphysema, and asthma are common in adults. Smoking is an important factor, which may exaggerate reactive airway. Also, history of wheezing during previous anesthesia and drugs like b-blocking agents consist risk factors[1].

In this report we present the sequel of events for the management of a patient, with no previous history of respiratory disease, who developed bronchospasm after anesthesia induction that did not resolve during the initial standard treatment.

CASE REPORT

Our case was a 49 year old male, who was scheduled for plastic surgery regarding of an ulcerous formation of the intergluteal region (Figure 1).

Figure 1. Lesion area



As standard procedure, a preoperative evaluation was performed and the patient was classified according to the ASA physical status scale as ASA class II. He declared being a smoker (1.5 packs per day for 30 years), social drinker and his past medical history included two previous surgeries for pilondinal cyst under local anesthesia. He had a BMI of 37, height 1.68m and weight 107kg and his functional capacity was more than 6 METS. General physical examination was unremarkable (ECG: sinus rhythm, no signs of uncompensated heart failure, normal heart and lung auscultation). Pa-

tient's airway assessment revealed Mallampati score IV with a normal mouth opening and neck movements. Instructions regarding premedication with nebulized Salbutamol sulfate, Ipratropium bromide along with budesonide bid and diazepam 5mg per os were given 1h before surgery.

Upon arrival at the operation room, standard monitoring which consisted of ECG 3-lead continuous recording (HR), noninvasive blood pressure (BP) measurement and pulse oximetry (SpO_2) was connected to the patient. The following vitals were recorded prior to anesthesia induction BP= 162/94mmHg, HR=79bpm and SpO_2=100%. Two 18G IV cannulas and Bispectral index monitoring were placed and Ringer's Lactated Solution was being administered. General anesthesia induction was performed by the co-administration of Fentanyl 0.25mg, Lidocaine 100mg, Propofol 200mg and Rocuronium 70mg. Patient was successfully intubated with the aid of an intubating stylet without vigorous effort. The subsequent glottis view was Cormack Lehane grade II. Patient's surgery was scheduled in theprone position, so a spiral endotracheal tube was inserted. A mild tachycardiac response (HR=85bpm) to laryngoscopy was recorded while BP remained stable.

While auscultating for the proper placement of the endotracheal tube, inspiratory, expiratory wheezing and rales were identified equal at corresponding sites over both lungs. At that time end tidal CO_2 ($ETCO_2$) was 35 mmHg, with capnography curve showing no evident signs of obstruction and SpO_2 was 100%, at FiO_2 = 0.5. Concurrently, an erythema of the left upper limb appeared, originating at the IV catheter used for the administration of fluids and drugs. In a few minutes a flush at patient's face was noted. 10 puffs of Salbutamol spacer -100mcg/dose- were delivered through the endotracheal tube, 2 mg of midazolam IV were given to deepen anesthesia, while Sevoflurane was initiated for anesthesia maintenance. Hydrocortisone 200mg, Dimetindene maleate 4mg/100ml IV infusion and Ranitidine 50mg IV were also administered. All this time vital signs were within normal range (BP=110/70mmHg, HR=70bpm, SpO_2=99%) and ventilation was adequate. Subsequent repeated doses of 10 puffs back to back of Salbutamol were given with a total of 30 puffs through the endotracheal tube with no signs of improvement. An arterial line at the radial artery and a central femoral venous catheter were placed. Pressure control ventilation mode was selected in order to avoid high peak airway pressure. However, airway resistance was evident from the beginning and increase in PIP (Peak inspiratory pressure) was necessary to keep $PCO_2$and tidal volume within acceptable values (Table 1).

Table 1. Patient's arterial blood gas (ABG) samples and ventilation related data in the Operating Theater

	1h	2h	3h	4h	5h
Ventilation modes	PCV	PCV	PCV	PCV	PCV
FiO_2/$ETCO_2$	0.5/35	0.5/38	0.6/38	0.7/40	0.7/35
PIP cmH_2O	25	27	27	28	28
RR Breaths/ /min	12	13	13	14	14
PEEP cmH_2O	5	5	5	5	5
PaO_2 mmHg	168	135	104	83	95.5
$PaCO_2$ mmHg	37.4	43.9	48.1	53.1	45.7
pH	7.39	7.32	7.3	7.27	7.225
BE/HCO_3	-0.5/23.6	-3.4/21.6	-2.8/23.4	-3.2/23.9	-2/22.3
SpO_2%	100	97	97	96	96

FiO_2: Fraction of inspired oxygen, $ETCO_2$: end-tidal CO_2, $PaCO_2$: arterial partial pressure of carbon dioxide, PaO_2: arterial partial pressure of oxygen, pH: potential of hydrogen, BE: base excess, HCO3: bicarbonate, SpO_2: oxygen saturation, PIP: Peak inspiratory pressure, RR:respiratory rate, PEEP: positive end-expiratory pressure, PCV: Pressure control ventilation.

Magnesium sulfate 2g IV and ketamine initially as two bolus doses of 20 mg followed by IV continuous infusion (2mg/kg/h) were also given in order to tackle the bronchospasm outbreak. Only few secretions were aspirated after trachea suctioning. The administration of nebulized adrenaline 3mg, through the breathing circuit, in order to manage inspiratory wheezing was futile.

Within the first hour, a chest X-ray was performed, that revealed no specific findings except for a suspicion of a consolidation of the left costophrenic angle. The new ECG was similar to the preoperative one.

Two hours after anesthesia induction, systemic blood pressure fell at 70mmHg and IV adrenaline was administrated. After a total of 100mcg adrenaline IV, IV Noradrenaline16mg/250ml N/S 0,9% ,at low doses, was initiated in order to maintain a mean blood pressure > 70mmHg. Hypotension could be justified by the anesthetic level (BIS 40-44) and lack of surgical stimulation. Neither inspiratory nor expiratory findings were ameliorated and considering the lack of improvement and the worsening of ABG findings, with gradual increasing difficulty to keep $PaCO_2$ within normal range ($ETCO_2$=38 mmHg and $PaCO_2$= 48 mmHg, 1mmHg at hour 3), it was decided that the surgery should be postponed and the patient to be transferred to the Intensive Care Unit (Table 1). It should be underlined that because of lack of available ICU beds, it was necessary to remain in the operation theatre for a total of 4,5hours. The alternative option was the patient to be transferred in ICU of another hospital, which was evaluated as risky for the patient's safety. Interestingly, the capnography displayed only mild upsloping unlikely to the typical "sharkskin" observed in obstructed airway.

At the ICU the patient received maximum treatment with bronchodilation agents, antibiotics, corticosteroids that included salbutamol, ipratropium, beclometasone, magnesium sulfate, aminophylline, methylprednisolone, ciprofloxacin, clindamycin and vitamin complex. Sedation was maintained with IV infusion (IVI) of midazolam, fentanyl and cisatracurium in order to sustain the best possible ventilatory conditions. Vasopressor support with Noradrenaline 32mg/250ml N/S 0,9% at 0,5 ml/h was disconnected by day 2 in the ICU, as mean pressure was > 90mmHg.

After communication with the patient's relatives, it was found that he smoked 2 packs per day for 35 years and consumed alcohol daily and activity level estimated below 6 METS. They also mentioned an incident of bee sting that resulted in him "nearly dying", which indicated an allergic background. This information diverge from our preoperative interview with the patient but anesthetic management would have not been altered dramatically as no signs of active or unstable cardiac disease and no respiratory disease was found with clinical examination[5].

On day 4 in the ICU alkalization of the urine was added because of CPK level at 1295. Diuresis was adequate through his clinical course. Undiagnosed hypertension was identified and controlled with antihypertensive agents on the grounds that pain was excluded, as the patient was receiving IVI Fentanyl, adjusted according to requirements, since day 1in ICU. Noteworthy is that if any effort would be made to lower the infusion of muscle relaxant respiratory failure occurred and subsequent inability to ventilate (Table 2, ABG day 4 in ICU).

Table 2. Patient's ABG samples and ventilation related data during ICU stay.

	Day 1	**Day 2**	**Day 3**	**Day 4**	**Day 5**
Ventilation modes	PRVC	PRVC	PRVC	SIMV VC	VC
FiO_2	0.6	0.6	1	0.65	0.6
TV*RR ml*Breaths/ /min	550*17	650*14	650*14	650*15	630*15
PEEP cmH_2o	6	6	6	6	6
PaO_2 mmHg	72.4	76.7	118	70.3	74.9
PaCO2 mmHg	46.7	48	48	72	49.2
pH	7.31	7.35	7.35	7.26	7.409
BE/HCO3	-2.8/23	0.4/26	0.6/26.1	2.5/31.2	5.3/30.5
SpO_2 %	93.5	95	98.1	93	94.5

FiO_2: Fraction of inspired oxygen, TV: tidal volume, $PaCO_2$: arterial partial pressure of carbon dioxide, PaO_2: arterial partial pressure of oxygen, pH: potential of hydrogen, BE: base excess, HCO3: bicarbonate, SpO_2: oxygen saturation, RR: respiratory rate, PEEP: positive end-expiratory pressure, PRCV: Pressure-Regulated Volume Control ventilation, SIMV: Synchronized intermittent mechanical ventilation, VC: volume control ventilation

In spite of all treatment, clinical features of wheezing were evident with no sign improvement.

Other factors that were considered as trigger

points were pain from the ulcerous formation and reaction due to mechanical irritation from the spiral endotracheal tube (HUDSON RCI SHERIDAN/SPIRAL-FLEX® made of PVC with an integral steel reinforcing wire). It is also worth mentioning that the ulcer was characterized by signs of malignancy, which mandated further prompt investigation.

On day 6 in the ICU after taking into consideration both risks and possible benefits, minimal surgery procedure was decided to proceed and consent from the patient's relatives was obtained. Meticulous debridement was performed by surgeons and the endotracheal tube was uneventfully switched to a standard one (RUSCH® SAFETY CLEAR®), using an intubating stylet, to exclude the possibility of mechanical irritation by the spiral tube. Surgery was uneventful.

On day 7 in the ICU, lung auscultation was evidently improved and gradual reduction in muscle relaxants and sedation infusion rates was attempted and progressively extubation was eventually commenced without additional deterioration of clinical features [Day 7 (a,b,c,d), Table 3].

On day 9 in the ICU, the patient was transferred to Plastic surgery department for further management under bronchodilation therapy. Close respiratory monitoring and consultation by pulmonologists was provided.

Table 3. Patient's ABG samples and ventilation related data from day 6 in the ICU until patient's discharge from ICU.

	Day 6	**Day 7a**	**Day 7b**	**Day 7c**	**Day 7d**	**Day 8**
Ventilation modes	VC	VC	PS5	T-P	MO_2	Venturi
FiO_2	0.6	0.6	0.6	0.6	0.98	0.5
TV*RR ml*Breaths /min	620*15	620*15				
PEEP cmH_2o	6	6	6			
PaO_2 mmHg	69	75	74	89	82.9	65
PaCO2 mmHg	42.9	40	56	47	45.9	43.5
pH	7.49	7.49	7.37	7.42	7.42	7.46
BE/HCO3	8.8/32.9	7.3/30.8	5.6/32	5.1/30	4.8/29	6.2/30
SpO_2 %	94.1	95.1	93	96	95.1	91

FiO_2: Fraction of inspired oxygen, TV: tidal volume, $PaCO_2$: arterial partial pressure of carbon dioxide, PaO_2: arterial partial pressure of oxygen, pH: potential of hydrogen, BE: base excess, HCO3: bicarbonate, SpO_2: oxygen saturation, RR: respiratory rate, PEEP: positive end-expiratory pressure, PS: Pressure support ventilation, T-P:T-piece, MO_2: oxygen mask.

After an interview with the patient it was concluded that the patient was exposed to an unfavorable environment with increased tobacco use, the day before surgery. A random sample a few days later shows marked signs of improvement (Table 4). A spirometry was conducted two months after the event which revealed no signs of respiratory disease.

Table 4. Patient's room-air ABG samples 3 days after discharge from the ICU

	Room-airABG samples
FiO_2	0.21
PaO_2	73.3
$PaCO_2$	37.6
pH	7.483
BE/HCO3	27.9/4.6
SpO_2%	93.9%

FiO_2: Fraction of inspired oxygen, $PaCO_2$: arterial partial pressure of carbon dioxide, PaO_2: arterial partial pressure of oxygen, pH: potential of hydrogen, BE: base excess, HCO3: bicarbonate, SpO_2: oxygen saturation.

DISCUSSION

Bronchospasm can be a destructive event in the perioperative period. In our case it was the first time for the patient to undergo an operation, so no previous medical history was available. Clinical examination was normal, but significant for chronic smoking, which can be a triggering factor for the development of bronchospasm.

According to Looseley, bronchial hyperreactivity is also associated with preoperative exposure to tobacco smoke, upper respiratory tract infection (URTI) and a history of atopy[6]. The first and the third factor were present to our patient.

The occurrence of perioperative bronchospasm has been reported up to 9% of asthmatic patients given general anesthesia, mainly after endotracheal tube insertion[7]. Smoking, as mentioned above, represents a major risk. Compared with nonsmokers, the relative risk of perioperative bronchospasm in smokers appears higher in females and in young smokers (16–39 yrs old) and is higher in patients with chronic bronchitis than in asymptomatic patients[8].

Of the 4,000 incidents reported in Australia, 103 reports of perioperative bronchospasm (3%) showed that an allergic mechanism was less frequently involved (21%) than a nonallergic mechanism (79%)[9]. Among these nonallergic cases, 44% occurred during the induction of anesthesia, 36% during the maintenance phase and 20% during the emergence/ recovery stage. During anesthesia induction, bronchospasm was mainly related to airway irritation (64%), whereas remaining causes were due to tube misplacement (17%), aspiration (11%), and other pulmonary edema or unknown causes (8%). During anesthesia maintenance, allergy (34%), endotracheal tube malposition (23%), airway irritation (11%) and aspiration with a laryngeal mask airway (9%) together accounted for almost 80% of the occurrences of bronchospasm. During induction or maintenance of anesthesia, bronchospasm caused by airway irritation occurred more frequently in patients who had one or more predisposing factors such as asthma, heavy smoking, or bronchitis. Others showed that an allergic mechanism accounted for 60% of the cases

in patients experiencing bronchospasm during induction of anesthesia[3]. A previous history of asthma was present in 50% and 60% of patients with nonallergic and allergic bronchospasm, respectively. Thus, uncontrolled asthma/chronic obstructive pulmonary disease is frequently involved with either pathophysiologic mechanisms (allergic vs. nonallergic), regardless of the stage of anesthesia (induction or maintenance).

The chronology of evolving clinical features is crucial to understand the pathophysiologic mechanism of an immediate hypersensitivity reaction.

IgE mediated anaphylaxis is characterized by cardiovascular manifestations. Bronchospasm coexists in 19-40% of cases and patients with a history of asthma or chronic obstructive pulmonary disease are more prone to it[2]. The occurrence of drug-induced anaphylactic bronchospasm is not related with intubation[3].

Latex-induced anaphylaxis develops up to 30-60 minutes after surgical incision and is associated with a history of atopy[10-12]. In our patient, except for bronchospasm no other distinct clinical features could imply an anaphylactic reaction. The delayed transient mild hypotension (1 hour after induction) was not attributed to an allergic reaction, but was justified by the effects of the anesthetic agents.

It is worth mentioning that bronchospasm can appear as acute reactivity from airway maneuvers and is not accompanied by cardiovascular compromise until the point when pathophysiologic mechanisms affect the preload through increased intrathoracic pressures and severe hypoxia with resulting respiratory failure happens[13].

The anesthetic plan should balance suppression and prevention of bronchospasm with the usual targets of patient safety, comfort and a quiet surgical field.

PREOPERATIVE MANAGEMENT

Going back and after considering information from both the patient, some would suggest that a spirometric exam should be performed. Noteworthy, according to guidelines from the European Society of Anesthesiology and the Hellenic Society of Anesthesiology, spirometry is not indicated as a routine exam in non-cardiac surgery. Results cannot alter anesthetic management and high risk patients can be identified upon auscultatio[5]. Furthermore, if evaluated far enough in advance, the patient should be advised to stop smoking at least 2 months before surgery[14]. Oral methylprednisolone 40 mg for 5 days before surgery has been proven to decrease post-intubation wheezing in recent diagnosed or poorly compliant patients with reversible airway obstruction[15]. If the patient is first evaluated immediately before operation and steroids are indicated, then i.v. corticosteroids may be useful. Short-acting bronchodilator therapy given prophylactically has

likely benefit. An optimal premedication relieve anxiety, improves work of breathing, and possibly prevent the induction of bronchospasm, while avoiding oversedation and respiratory depression. No ideal drug or drug combination exists for this. The α-2 agonist dexmedetomidine has a favourable profile, including anxiolysis, sympatholysis and drying of secretions without respiratory depression. Although there are numerous reports of its benefits in awake intubation and anaesthetic emergence[16], there are no data on its role in the asthmatic patient. By drying secretions and suppressing upper airway vagal responses, anticholinergic agents such as atropine or glycopyrrolate can decrease airway reactivity and should be considered.

INTRAOPERATIVE BRONCHOSPASM

A number of perioperative medications can protect from bronchospasm. Propofol appears to be superior to thiopental and etomidate in constraining increases in airway resistance, but there have been case reports of its association with bronchospasm in susceptible patients[17-19]. In heavy smokers undergoing anesthesia, a propofol formulation that uses metabisulphite induces higher airway resistance than that preserved with calcium edetate[20], an observation that should be taken into consideration in asthmatics as well. Ketamine has excellent induction characteristics and induces bronchodilation, possibly by interfering with the endothelin pathway[21].Lidocaine can prevent bronchospasm by attenuating sensory responses to airway instrumentation or irritation. Intravenous injection of lidocaine quickly achieves adequate airway anaesthesia. Inhalation anaesthetic induction should be considered if circumstances allow. Sevoflurane is well tolerated as an inhalational induction agent and has good bronchodilatory effect.

TREATING PERIOPERATIVE BRONCHOSPASM

The aims of treatment are to relieve airflow obstruction and subsequent hypoxemia as quickly as possible.

When isolated perioperative bronchospasm occurs, oxygen concentration should be increased to 100%, pulmonary compliance should be monitored and all causes of high-circuit pressure should be identified[22]. Increased concentration of a volatile anesthetic (sevoflurane, isoflurane) is often useful[4] with the exception of desflurane because of its airway irritant effects, particularly in smokers[23]. Deepening anesthesia with an intravenous anesthetic (propofol) may be required because intubation-induced bronchospasm may be related to an inadequate depth of anesthesia.

Rapid-acting β_2-selective agonists are adequate drugs for the fast relief of bronchoconstriction. They should be immediately administered via a nebulizer (8 –10 puffs to achieve appropriate

therapeutic levels, may be repeated at 15- to 30-min intervals) or, if available, with a metered-dose inhaler (5–10 mg/h) connected to the inspiratory limb of the ventilator circuit. Salbutamol can be given also intravenously initially 250mcg slow and then at 5mcg/min titrated up to 20mcg/min IV infusion[6].

Systemic glucocorticosteroids should not be omitted because they speed resolution of exacerbations by decreasing airway inflammation. High dose methylprednisolone should be given, for example, 125 mg IV, with the understanding that it will take 4–6 h before they exert their beneficial effect.

The use of an antimuscarinic inhaled medication (e.g., ipratropium bromide) has been shown to attenuate reflex-induced bronchoconstriction with efficacy similar to inhaled β_2-agonists. Thus, combined nebulized ipratropium bromide (0.5 mg 4 – 6 times hourly) with a nebulized β_2-agonist produces greater bronchodilatation than a β_2-agonist alone and may be used to treat life-threatening bronchospasm or in those with a poor initial response to β_2-agonist treatment.

Magnesium plays a beneficial role in the treatment of asthma through bronchial smooth muscle relaxation, leading to the use of intravenous (single dose of magnesium sulfate: 2 g over 20 min) or inhaled preparations (doses from 110 mg to 1,100 mg) in patients with severe bronchospasm that fails to be relieved with β_2-agonists[24]. Epinephrine should be used in cases of associated cardiovascular collapse suggestive of IgE-mediated anaphylaxis[2,24]. Currently, no recommendations regarding epinephrine can be proposed[25], except that its use would be reasonable as a rescue therapy in patients with severe asthma complicated by hypotension that is not secondary to dynamic hyperinflation[26].

Helium–oxygen mixtures (heliox) have been used to maintain laminar flow in acute bronchospasm[27], but reports on its use perioperatively are limited[28]. A major limitation is that heliox mixtures can provide only 21–30% oxygen[29]. Helium facilitates ventilation but does not reverse the underlying bronchospasm, but it can provide an important bridge until corticosteroids take effect[30,31]. Nitroglycerin has been reported anecdotally to reverse acute bronchospasm, probably through direct smooth muscle relaxation[32,33].

Elective surgery should be postponed unless bronchospasm persists at baseline despite maximal medical optimization of the patient and further care provided in a monitored setting.

In spite of the aforementioned beneficial effects of lidocaine and propofol both drugs have been associated with perioperative bronchospasm. Lidocaine either IV or topically applied at the larynx has been linked with episodes of bronchospasm[34-36]. In such instances,

prompt drug discontinuation is needed to reverse clinical features.

Taking into account all available information this was a case of hyperreactive airway response triggered by mechanical irritation from the endotracheal tube.

CONCLUSION

Preoperative evaluation is the backbone of the anesthetic management. Predisposing factors for cardiovascular or respiratory complications should be meticulously recorded for the optimal preparation of the patient. Prompt interventions should be implementable as soon as possible to be beforehand with unexpected adverse events. Universal algorithms for emergencies in anesthesia provide a thorough step by step approach and promote patient safety. The foremost target for the anesthesiologist is to secure the patient's airway and avoid hypoxia. Bronchospasm can threaten this vital function and pose the patient at risk so it should be aggressively and thoroughly managed.

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