Implications of anesthesia for the asthmatic patient

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The anesthetic management of an asthmatic patient can be challenging for the anesthetist. Serious complications, of which 75% are pulmonary, can occur both intra- and post-operatively. The author reviews these complications and offers several methods for administering anesthetic agents to asthmatic patients. Also included is a discussion of the autonomic nervous system.

Asthma runs the spectrum from being a nuisance to being a terrifying experience which, if it continues unabated to status asthmaticus, can lead to death. The disease, many times, is a difficult puzzle for the medical practitioner to solve.

If an asthmatic patient must undergo anesthesia, another spectrum of attitudes may be manifested by those of us who deliver the anesthesia care. They may range from a "blase cavalierism" based on an abject ignorance of the disease to "controlled panic" based on past experience with asthmatics. The intent of this article is to provide a bit of illumination on asthma. Also included are a few hints on how to anesthetize the asthmatic patient, whether he or she is presently asymptomatic or in the midst of a status asthmaticus siege.

Physiology of asthma

What is asthma? Gold defines asthma as a condition involving intermittent episodes of bronchospasm associated with wheezing, and symptom free periods. The patient usually has a personal or family history of allergy.

In true acute asthma, there is no infection. Therefore, when a suspected asthmatic has a chronic productive cough, purulent sputum or x-ray evidence of lung infiltration, it can be construed that the patient either has asthma complicated by an infection, chronic bronchitis, emphysema, pneumonia, or some combination of these conditions. Furthermore, it is inherent in the definition of pure asthma that there be symptom-free periods during which airway resistance is normal.

Characteristic symptoms in acute asthma are bronchoconstriction and mucus production with some vascular engorgement and inflammatory edema in the submucosa. The mechanism of bronchoconstriction is extremely complex and is not totally understood, although progress has been made in the last ten years.

Many researchers feel that the basic problem with asthma is an abnormal balance between the sympathetic and parasympathetic systems innervating the lungs. This concept is known as the autonomic imbalance theory.

The normal respiratory tree is maintained in a state of slight bronchoconstriction which provides a resting bronchomotor tone to the airways. Changes in the resting tone and the effects of various maneuvers and irritants are actively and finely adjusted in the normal lung. In the asthmatic lung, detrimental bronchospasm replaces the normal advantages of bronchomotor tone.
The control of bronchomotor tone and bronchial muscle responses is extremely complex. It involves afferent input from chemoreceptors and other receptors which activates a variety of reflex breathing responses. It is believed that the asthmatic lung has an inherent or acquired increase in bronchomotor tone and a heightened susceptibility to numerous and variable stimuli, resulting in reflex bronchospastic responses.

The receptor theory

This leads us to a brief discussion of the receptor theory. All muscle fibers and secretory cells can be affected by natural hormones or messengers and by a variety of administered drugs. The particular effect produced depends on the characteristic relationship between the stimulating molecule and the intrinsic organization of the cell. A specific agent may cause a precisely determined reaction by activating a series of events in specialized cells of certain organs. This is best explained by assuming that the cell has specific receptor sites to which activating messenger molecules with the appropriate configuration can become fixed. The messenger molecule is much like a specific key which fits into a specific lock.

The effects of autonomic nervous system stimulation can be explained by the receptor theory. The sympathetic nerve fibers are known to release the natural hormone transmitter norepinephrine and epinephrine while the parasympathetic fibers release acetylcholine. These agents have a specific action on bronchial muscle fibers. Epinephrine causes relaxation, as does norepinephrine though at a lesser degree, and acetylcholine causes contraction. These findings correlate with the receptor theory by ascribing the presence of both adrenergic and cholinergic receptors in the bronchial muscle tissue.

The nature of the response of the adrenergic receptors to stimulation by epinephrine and similar adrenergic (sympathomimetic) agents is complex. Therefore, these receptors can be placed into at least three categories: alpha, beta, and beta2. The cholinergic receptor is stimulated by acetylcholine and other parasympathomimetic agents. The variations in responses allows further categorization of receptors: nicotinic and muscarinic. The bronchial muscle tissue is classified as muscarinic.*

In addition to alpha, beta and cholinergic receptors, bronchial muscle tissue provides specific receptors for other mediators such as histamine (histaminergic receptors) and serotonin (serotoninergic receptors). The most important receptors in the treatment of bronchospasm are the adrenergic receptors which are found in most tissues of the body as well as in bronchial muscle. Because of their widespread location, most drugs used to treat bronchospasm usually exert numerous side effects in other tissues.

Pharmacologic studies have concluded that bronchial muscle has two different types of adrenergic receptors, alpha and beta2. These receptors also exist in other tissues; alpha receptors are also found in mucosal blood vessels. Beta2 receptors are distributed principally in the peripheral blood vessels of bronchial muscle, peripheral limb muscles, other smooth muscle, and the central nervous system (CNS). A similar class of receptors, the beta1 receptors, are found principally in heart muscle and in adipose tissue. Alpha receptors generally mediate excitatory effects, whereas beta receptors mediate inhibitory effects (except in the myocardium).

Alpha receptors are stimulated by a variety of agents generally classified as mucosal vasoconstrictants, since their principal effect is on the alpha receptors located on the blood vessels of mucous membranes. Bronchial muscles contain a relatively small supply of alpha receptors and their stimulation can result in only mild bronchoconstriction.

Beta1 receptors in the heart are stimulated by many agents, including drugs used principally as bronchodilators. The effect on the heart tends to bring unwanted side effects. Stimulation of the cardiac beta1 receptors results in chronotropic and inotropic effects such as increased cardiac output, tachycardia and a tendency toward arrhythmias.

Beta2 receptors in the lung induce bronchial muscle relaxation. Stimulation of beta2 receptors in mucosal and peripheral blood vessels results in vasodilation. Beta2 receptor stimulation also affects the nervous system and peripheral muscles, resulting in anxiety, nervousness, insomnia and tremors. One of the metabolic effects of stimulation of beta2 receptors in muscles and in the liver is glycogenolysis, by which glycogen is converted to glucose and energy is made available.

A drug such as epinephrine stimulates all three types of adrenergic receptors (alpha, beta1 and beta2) and thus causes a considerable number of reactions in addition to bronchial muscle relaxation. The actual effects on an individual patient vary, depending on the dosage of the drug, route of administration, and individual variability.
of administration, prior exposure to adrenergic drugs, individual variation and reflex responses. The search for new pharmacologic bronchodilators has been directed at the development of potent and selective beta₂ stimulators. Further attempts are being made to eliminate the unwanted beta₂ effects on blood vessels and the nervous system.

In recent years, attention has been directed to cholinergic pharmacology. Consequently, the role of the vagus in mediating both normal bronchomotor tone and bronchospasm has come to light. There is now evidence that many of the allergic and other reactions in the lung that cause bronchospasm do so by stimulating the mast cells and inducing the release of mediators such as histamine. These molecules then act on muscles cells to cause a minor degree of bronchospasm.

The major bronchospastic response, however, is indirect, resulting from the effect of the mediators on parasympathetic vagal stimulation. The ensuing reflex results in a release of acetylcholine at the neuromuscular junction, and this transmitter, in turn, acts on the muscle cells to cause a more profound degree of contraction. Such a bronchoconstrictive reflex can be blocked by anticholinergic drugs, such as atropine, which potentiate bronchial muscle relaxation or at least cause inhibition of bronchial muscle contraction.

It is probable that other receptors in lung tissue, such as the histaminergic, serotoninergic and prostaglandin receptors, play a role in bronchospasm, but their full effect is not yet fully understood.

**Mediators of bronchospasm**

Asthma has an allergic basis in many patients. This implies that a particular antigen to which the patient has been sensitized (such as pollen components) reacts with a specific antibody. Immunoglobulin (Ig), the main class of antibody that participates in the allergic reaction, in most cases appears to be reagin (IgE). Allergic subjects produce a specific IgE which then becomes attached to the surface of tissue mast cells.

In the asthmatic patient, large numbers of primed cells are located in the immediate proximity of bronchial muscle cells. The importance of IgE in allergic asthma is suggested by the fact that such patients often have a serum concentration of reagin that is 5-7 times the normal level. Few non-asthmatic people have elevated IgE levels and not all asthmatics have increased levels.

When an inhaled or blood-borne antigen reacts with the specific fixed antibody, the process results in a series of reactions, including a cation-dependent activation of serine esterase, that results in the breakdown of mast cell granules and the release of extremely potent mediators. These molecules diffuse into surrounding tissues, activating the responsive tissue cells and evoking vagal reflex.

The result of this action are the characteristics of asthma: bronchospasm, viscous mucous secretion, eosinophilia, vasodilation and edema of the airways. Mediator release, causing similar reactions, may also occur from other tissue cells that participate in immunologic reactions such as the granule containing basophils and eosinophils. However, the role of these blood cells in the sequence of events leading to bronchospasm is not clearly established.

This complex process resulting in mediator release may occur in as much as 50% of patients who have asthma. It appears to be the mechanism which accounts for the most important forms of allergic asthma.

**Interaction of mediators and vagus**

It has been known for some time that various forms of lung stimulation cause a release of the mediators and that these mediators can stimulate vagal activity. Gold has shown that antigen—antibody reactions in asthmatic patients result in the release of mediators. These in turn, stimulate irritant receptors in the lung which trigger vagal reflexes. This results in bronchoconstriction produced by the action of acetylcholine on the muscle cells.

Furthermore, vagal stimulation of mast cells in the presence of the antigen potentiates the release of more histamine and other mediators. Obviously, this is a very complex mechanism with multiple interrelated actions which causes bronchial muscle overactivity.

Autonomic impulses, mediators and drugs act directly or indirectly on airways to affect the contraction of bronchial muscle. The mechanism that is activated or inhibited within the cells involves a series of biochemical reactions in which the cyclic nucleotide 3'5' adenosine monophosphate (c-AMP) is pivotal.

Cyclic AMP has been called the "second messenger" because, once it has been produced (as a result of the stimulatory effect of the first messenger), it affects the activities of enzymes in a large variety of cells. Once c-AMP accumulates intracellularly, the effect is bronchial muscle relaxation, although the exact mechanism is unclear.

A second system exists that counteracts the effect of cyclic AMP in a "yin-yang" fashion.
Cyclic 3'5' guanosine monophosphate (c 3'5' GMP) acts to produce physiologic effects opposite to those induced by c-AMP. It is believed that c-AMP and c-GMP have a more important role in controlling muscle tone within the muscle cell. Bronchodilators such as sympathomimetics and phosphodiesterase inhibitors appear to act directly on the muscle cell where other drugs such as corticosteroids have a greater effect on mast cells.

At this point, it is apparent that the mechanisms of asthma are not simple, nor are they essentially clear. Therefore, treatment of asthma is not simple. A number of medications can be administered that act at multiple points in the chain of events, causing the bronchial muscle to go into spasm.

The following (Table I) is a review of the pharmacology of these medications.

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**Table 1.**

Drugs used in the treatment of asthma

1. **Bronchodilators**
   - Beta-2 adrenergic receptor stimulators
   - Phosphodiesterase inhibitors
   - Anticholinergic drugs
   - Prostaglandins
   - Other bronchial muscle relaxants

2. **Antimediator drugs**
   - Antihistamines
   - Corticosteroids
   - Bischromones
   - Immunosuppressive drugs
   - Anti-inflammatory drugs and analgesics
   - Diethylenediamine

3. **Agents affecting alpha receptors**
   - Alpha-adrenergic receptor stimulators
   - Alpha-adrenergic receptor blockers

4. **Miscellaneous drugs**
   - Mucokinetic agents
   - Antimicrobial agents
   - Cough medicines
   - Tranquilizers
   - Anesthetics (local and general)
   - Narcotic analgesics
   - Gases:
     - Oxygen
     - Helium
     - Carbon dioxide
   - Others:
     - Phenytoin
     - Alcohol

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**Bronchodilators**

Most of the bronchodilators used are either beta₂ stimulators or phosphodiesterase inhibitors. Others include the anticholinergics, prostaglandins and other bronchial relaxants.

The prototype drug in this category is epinephrine, which is a natural hormone and a first messenger. There is a tendency toward tachyphylaxis or refractoriness, whereby excessive use of a bronchodilator leads to a loss of the effectiveness of the agent.

**Phosphodiesterase inhibitors.** The second messenger c-AMP undergoes enzymic breakdown by phosphodiesterase. Intracellular c-AMP can be maintained by interfering with the inactivation process.

The most significant of these agents are the methylxanthines. Caffeine, theobromine and theophylline are in this classification, but only theophylline is powerful enough to be of pharmacologic significance.

**Anticholinergic drugs.** Atropine has long been used in the treatment of asthma. (For many years, asthmador cigarettes were available, the principal component of which was atropine.) Therapeutic selectivity is obtained by local application of the drug. Inhibition of bronchospasm can be effectively achieved by nebulization of atropine. Aerosol administration of atropine does not seem to effect the bronchial glands; though systemic administration causes vagal inhibition, resulting in a decrease in secretion by these glands and a consequent drying of the respiratory mucosa.

Atropine prolongs the effect of c-AMP stimulators such as isoproterenol. Atropine has also been shown to reverse asthmatic bronchospasm by drugs that block beta receptors such as propranolol. There are some newer aerosol anticholinergics, for example, ipratropium bromide, which are even more powerful bronchodilators.

**Prostaglandins.** These hormonal agents have been shown to have a bronchodilator and bronchoconstrictor effect on lung tissue. Prostaglandins have been broken down into a number of subcategories. The varied properties of these agents suggest that they will have a role in the future management of asthma, but it is difficult to predict what that role will be.

**Other bronchial muscle relaxants.** Khellin, a drug extracted from a plant found in Mediterranean countries, has some bronchial dilatory properties, but it is also an antimediator acting through the c-AMP mechanism. Cromolyn sodium is a potent prophylactic drug which interferes with the antigen antibody reaction.
Marijuana has some value in the relief of bronchospasm. Tetrahydrocannabinol, the major active ingredient in marijuana, relaxes the bronchomotor tone of the normal airways and causes bronchodilation in the asthmatic lung. The exact mechanism of this effect is still unknown.

**Antimediator drugs**

Many causes of asthma are clearly related to the actions on bronchial muscle by mediators that are released from mast cells and basophils. It is disappointing to find that pharmacologic agents, which are specific antagonists of individual mediators, have relatively little value in asthma. In contrast, nonspecific agents such as corticosteroids and cromolyn may have greater success with an allergic etiology. The following agents vary in value in the treatment of asthma.

*Corticosteroids* such as prednisone are of established value in acute therapy and in prophylaxis. They have various actions on mast cells, beta<sub>2</sub> receptors and at other sites. Antihistamines are of minor value in allergic asthma, but much work needs to be done to evaluate these agents which have many differing sites of action.

Bischoromones-cromolyn stabilize mast cell surface. As stated, this category of drugs has been established as being valuable in prophylaxis of allergic asthma and also exercise-induced asthma.

*Immunosuppressive agents* are being researched, but at this time, no value has been established for their use in asthma therapy.

Anti-inflammatory agents such as aspirin and phenylbutazone may have a prophylactic value in some patients, however, they cause asthma in hypersensitive patients. They work by interfering with the prostaglandin PFG<sub>2</sub> which causes bronchoconstriction.

Diethylenecarbamide is an antifilarial agent which may be of value in tropical asthma. Aerosol administration may prevent exercise-induced asthma.

**Agents affecting alpha receptors**

Alpha receptors are distributed throughout the lungs, and a wide variety of results can be obtained by using drugs that are alpha adrenergic agonists or antagonists. Mast cells and bronchial muscle cells have a sparse supply of alpha receptors, compared with beta<sub>2</sub> receptors. Stimulation of the alpha receptors causes the bronchospasm with an increase in airway resistance.

However, stimulation of the alpha receptors that supply pulmonary blood vessels results in vasoconstriction. If the mucosa is inflamed and "boggy," the resulting mucosal shrinkage may outweigh the alpha adrenergic bronchospasm, and result in a balance that decreases airway resistance. Thus, alpha receptor stimulators may actually improve airway dynamics in bronchial asthma.

Alpha adrenergic receptor blockers have been used by investigators for the treatment of asthma. The blockers result in a decrease of alpha adrenergic tone in bronchial muscle, and can therefore result in a decrease in airway resistance.

Thymoxamine is one of the most specific of the alpha receptor blocking drugs. Inhalation of this agent can result in marked potentation of the bronchospasmolytic effect of beta<sub>2</sub> stimulating drugs. Phentolamine (Regitine®) has also been used in the treatment of asthma.

It cannot be emphasized enough that classification of drugs requires further study. When an adrenergic drug with potent beta<sub>2</sub> activity, negligible beta<sub>1</sub> properties, marked alpha blocking activity and an anticholinergic effect is produced, it will mark a major step forward. This combination of properties in a medication will be valuable in the treatment of asthma with a clear autonomic imbalance.

**Miscellaneous drugs used in treating bronchospasm**

A large variety of drugs is administered to patients with respiratory disease. The primary focus of these medications is aimed at the nonbronchospastic components of the pulmonary problem. Some of these agents have a direct and beneficial effect on asthma, and are used as adjuncts in the management of the asthmatic patient.

Mucokinetic agents, such as potassium iodide solution (SSKI™) or sodium iodide are useful to treat the patient with acute asthma who has difficulty in clearing tenacious mucous.

Antimicrobial agents. Infection can act as a nonspecific irritant thereby triggering bronchospasm. Many physicians prescribe prophylactic antibiotic therapy in the treatment of acute asthma. Two antibiotics have been reported to have an intrinsic antibronchospastic effect. They are troleandomycin (TAO™), and erythromycin. When troleandomycin is given, it potentiates the effects of steroids. Therefore, the steroid dosage should be reduced or the patient will have a greater chance of developing Cushing's syndrome.

Cough medications have also been used for the treatment of asthma, but they are categorically of questionable value.
Tranquilizers. Acute asthma is usually accompanied by severe anxiety. Alleviating this factor can result in a physiologic improvement in airway mechanics. Anxiety can be greatly reduced by simple reassurance provided by a competent practitioner. Many times the busy physician prescribes pharmacologic reassurance in the form of “that little pill.” This certainly may improve the status of the emotional, panic-stricken asthmatic, but should not be relied upon exclusively.

Diazepam (Valium®) is very popular, but can cause depression of the respiratory center. This can be particularly disadvantageous if the patient is hyperventilating. This drug can be given safely if prescribed judiciously.

Hydroxyzine (Atarax®, Vistaril®) is the one tranquilizer that has definite bronchodilatory properties. Besides being a tranquilizer, hydroxyzine is an anticholinergic, antihistamine and antimetic. Some anesthesia practitioners utilize only hydroxyzine as a preoperative medication.

Barbiturates. Phenobarbital has been used widely as an adjunct in asthma preparations when combined with ephedrine and theophylline. Barbiturates can adversely affect the respiratory system by causing respiratory depression. Thiopental (Pentothal®) can cause or potentiate bronchospasm. A further disadvantage of barbiturates is that they induce the formation of microsomal enzymes in the liver that hydrolyze corticosteroids. Therefore, a steroid-dependent asthmatic receiving a preparation with a barbiturate may experience reduced therapeutic value from the steroids.

Gases

Oxygen is often required in bronchospastic patients for relief of hypoxemia. It does not appear to have any intrinsic bronchodilatory properties.

Helium has been used in the past because of its relatively low density. It has since lost its therapeutic value because of the more sophisticated drugs now available.

Carbon dioxide is a bronchodilator that has been advocated in the use of asthma. It has been proven that carbon dioxide causes relaxation of peripheral and central airways in young asthmatics. Carbon dioxide should be used cautiously to avoid possible complications such as respiratory depression and central nervous system narcosis.

Other agents

Other agents have been suggested as possible bronchodilators. Phenytoin (diphenylhydantoin, Dilantin®) was found to be helpful by one investigator in controlling asthma, but there has been no intensive study to prove its efficacy.

Alcohol. Since alcohol has been hailed as the “cure all of all problems,” it should also be mentioned. The relief found in the treatment of asthma with alcohol could very well have been achieved with reassurance alone.

Anesthetic agents

Anesthetic agents, both regional and general, have been found to be effective in reversing some forms of bronchospasm. There is a place for general anesthesia in the treatment of asthma. Anesthetists are sometimes called upon to give general anesthetics to patients in status asthmaticus. The administration of anesthesia in these cases is especially difficult.

How does the practitioner choose an anesthetic for the asthmatic patient? Following the selection process used by a number of practitioners at our institution (Wilford Hall USAF Medical Center) should provide a helpful step-by-step mechanism.

A very careful history must be elicited as a part of the preanesthetic assessment. When dealing with the patient who states a positive history of asthma, it is important to determine the severity of the disease. For example, you may find that the patient’s asthma may be a “runny nose” in November and December (in even years) or a slight cough in the morning, due to post-nasal drip.

It is imperative to elicit a history of wheezing and then determine the severity. Obviously, the patient with the occasional wheeze in the spring or fall who requires no therapy should be monitored, but not as closely as the “year round” asthmatic who is intractable on steroids and other asthma medications.

In choosing an anesthetic, the obvious question is, “Can it be done under some form of conduction anesthesia?” If it can and the patient can be emotionally supported to accept this procedure, conduction anesthesia is our first consideration. By not introducing noxious agents into the tracheobronchial system, we have eliminated a major problem area.

Emotional support of the patient cannot be emphasized enough. A suitably anesthetized patient, lying awake, listening to the basic discussion in the operating room and fearing the worse, may be psychogenically triggering a full blown asthmatic attack. It is imperative that the discussion be kept at a minimum and that the noise level is kept down. A continuous light conversation be-
tween the anesthetist and the patient also helps to alleviate problems.

If the patient with severe asthma requires general anesthesia, all attempts are made to have the patient in the best pulmonary shape possible. Pulmonary function tests and arterial gases are performed, and the patient is evaluated by the pulmonary medicine department.

It is important when administering general anesthesia to give as gentle and skillful an anesthetic as possible, in addition to giving a profound anesthesia, especially in cases where endotracheal manipulation and intubation are to be performed.

There is still a great deal of controversy as to whether or not thiopental should be used as an induction agent. Although it is recognized that thiopental and other thiobarbiturates can cause bronchoconstriction, those who advocate use of this agent with asthmatic patients state that if thiopental is to be used, a large dosage (5-8 mg/kg) should be given to obtund reflexes. The rationale for this is that complications are the result of an inadequate dosage, not because of the intrinsic properties of the drug.

Some viable alternatives as intravenous inducing agents are methohexital (Brevital®), diazepam (Valium®), and ketamine HCl. Methohexital and diazepam have the unpleasant side effect of causing vascular pain on injection and methohexital can cause hiccuping and coughing. Primary induction with halothane is a definite alternative to thiopental, especially with the asthmatic child.

Use of the laryngeal tracheal anesthesia (LTA) kit is felt to be indicated for the asthmatic patient who must be intubated. Some practitioners use two LTA kits—320 mg of 4% lidocaine to aid in obtunding laryngeal reflexes. A number of other local agents have been advocated but the availability, ease and reliability of the LTA kit has precluded the other agents (except for use of cocaine 4% to shrink nasal mucosa if a nasotracheal intubation is necessary).

Controversy still rages as to which inhalation agent is best. Many “old timers” still feel that there is nothing like diethyl ether to dilate the bronchi in the asthmatic, break up the mucous plugs and get them to the upper airway where they can be eliminated via suctioning. Looking to other agents, such as halothane and enflurane, provides other advantages, however.

Halothane is considered the agent of choice because it does relax bronchial musculature and does not irritate the respiratory mucosa as does ether. Our experience has shown that halothane can cause a problem, though. A patient who presents with some bronchospasm and is being given aminophylline intravenously will have serious ventricular arrhythmias under deep halothane.

A viable alternative under these circumstances is enflurane (Ethrane®). Its bronchodilatory properties have not as yet been determined, but there is no evidence of its having any bronchospastic properties. In the cases where we experienced arrhythmias through the use of halothane, a switch was made to Ethrane®, and the arrhythmias cleared.

Methoxyflurane (Penthane®) is also a potent agent which may have bronchodilatory properties. However, it has a very slow induction time which can induce a coughing, bucking laryngospasm.

The one agent universally contraindicated is cyclopropane. It does exacerbate bronchospasm and with sympathomimetic drug therapy, there can be severe arrhythmias.

Nitrous oxide is a natural agent which assists in getting the volatile agents into the patient via the second gas effect.

Narcotic agents are also generally contraindicated because of a possible intrinsic bronchoconstrictive property and a reduced postoperative respiratory drive. Morphine is particularly contraindicated because of the release of histamine from tissues.

Handling intraoperative emergencies

In the event of an intraoperative asthmatic emergency, we have developed the following procedures at our institution (though not necessarily in this order depending upon the patient’s etiology.)

1. The anesthetic machine is checked for valve malfunctions. There may very well be a problem in the machine; use an Ambu Bag® to attempt to ventilate the patient.
2. The endotracheal tube is checked. Possibly inject an LTA through the tube.
3. The patient’s anesthetic status is determined. You may need to switch from halothane to enflurane or vice versa, or give epinephrine 1:1000 .5 cc subcutaneously and/or .2 cc via the endotracheal tube.
4. Two puffs of an ISO Medihaler® are placed down the endotracheal tube; an isoproterenol drip may possibly be started.
5. Benadryl® (diphenhydramine) 25-50 mg IV is administered.
6. Hydrocortisone (Solu-cortef®) up to 1000 mg IV (push) is administered.
7. Aminophylline 5 mg/kg IV is administered.
Conclusion

There is no question that asthmatic patients are difficult to anesthetize. Complications can be terrifying if they occur. The overall incidence of intra- and post-operative complications are increased in the asthmatic patient. Seventy-five percent of these complications are pulmonary.

By giving the safest, gentlest anesthetic possible to asthmatic patients and by giving them emotional support pre- and post-operatively, we as anesthesia practitioners are doing our part to help minimize potential complications.

REFERENCES

In addition to the references listed below, much of the information presented in this article was obtained from unpublished materials and from conversations the author had with experts in the field. Although not presented in the context of the article itself, these references are offered for further study and reading.


AUTHOR

Lt. Colonel Salvatore LoPalo, CRNA, BSN, MS, received his RN from Craig Colony Hospital School of Nursing in Sonyea, New York, and graduated from St. Mary's Hospital School of Anesthesia in Rochester, New York in 1960. He received his BSN from the University of Nebraska in 1967, and his MS in Education from the University of Southern California in 1971.

He entered the Air Force in 1961, serving as Chief Nurse Anesthetist at Schilling AFB in Kansas before serving overseas in Vietnam and Spain. He has received the Bronze Star Air Medal and the Air Force Commendation Medal with Two Oak Leaf Clusters. He became a staff instructor in the Nurse Anesthetist Residency Program, Wilford Hall USAF Medical Center, Lackland AFB, Texas in 1975; Associate Director in 1978; and is currently Program Director there. He also serves as Consultant in Nurse Anesthesia to the Surgeon General of the United States Air Force.

Lt. Col. LoPalo was an On-Site Visitor for the Council on Accreditation of Nurse Anesthesia Programs from 1976 to 1978, and became a Senior On-Site Visitor in 1978, a position he presently holds. He is an Educational Consultant to the American Association of Nurse Anesthetists, and has published and presented numerous professional papers.

Lt. Col. LoPalo has presented this paper on several occasions throughout the country; it is the basis of his presentation at the 47th AANA Annual Meeting and Professional Sessions being held in Atlanta, Georgia in September, 1980.

It should be noted that the opinions stated in this article are those of the author and are not reflective of the official opinions of the Department of Defense and U.S. Air Force.