Anesthesia for the Asthmatic Patient
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This article provides a review of the physiology of the asthmatic lung and explores the options open to the anesthetist faced with the asthmatic patient.

The question of how to best manage the asthmatic patient under anesthesia has lingered for years, and though it has been partially resolved from time to time by the discovery of new agents or techniques, it still remains. The purpose of this article is not to unequivocally answer that question but to give the practitioner additional insight with which to deal with it.

In review, a balance is struck between adrenergic and cholinergic activity upon the pulmonary system, which maintains homeostasis within. Beta adrenergic stimulation causes bronchodilation, while alpha adrenergic stimulation causes bronchoconstriction. Cholinergic stimulation also causes bronchoconstriction. Two types of beta receptors have been discovered: beta1 and beta2. Beta1 receptors cause cardiac stimulation, while beta2 receptors cause bronchodilation and also vasomotor depression.

In the early 1960's, Szentivanyi advanced the theory of beta adrenergic blockade as a unifying mechanism in asthma. In the words of Wilson and Galant:

"This theory proposes that many of the stimuli which result in the clinical syndrome of asthma do so by causing the release of potent chemical mediators of inflammation such as the kinins, slow reacting substance of anaphylaxis (SRS-A), histamine, serotonin and acetylcholine. These mediators, in turn, result in bronchoconstriction and secretion of epinephrine as a homeostatic response. If beta adrenergic receptors are normally responsive, activation of these by epinephrine will balance alpha adrenergic and cholinergic receptor stimulation and airway patency will be maintained. However, if beta receptors are defective, bronchoconstriction is favored."²

Is asthma, then, a phenomenon of autonomic imbalance caused by defective beta receptors? The published evidence points to this conclusion.

At this time the beta adrenergic receptor is thought to incorporate adenylyl cyclase, an enzyme catalyst which converts adenosine triphosphate (ATP) to cyclic 3', 5' adenosine monophosphate (CAMP) in its structure. A second enzyme, phosphodiesterase, reduces CAMP to inactive 5' adenosine monophosphate (AMP).³

The presence of increased levels of CAMP influences bronchial smooth muscle in two ways. It inhibits (lung mast cell) the release of mediators of bronchoconstriction following antigen exposure, and also exerts a direct influence on smooth muscle favoring relaxation. An opposite effect, favoring...
broncho-constriction is achieved by increased levels of guanosine 3', 5' monophosphate (cyclic GMP) which is thought to be produced by cholinergic stimulation. This can be avoided by blocking vagal pathways with anticholinergics.

The success of the drugs used in treating asthma is explained by the resulting CAMP levels. The catecholamines stimulate adenyl cyclase conversion of ATP to CAMP. The methyl xanthines (aminophylline), on the other hand, inhibit phosphodiesterase reduction of CAMP.4

Preoperative evaluation of asthmatic patients

Preoperative evaluation of asthmatic patients is most important because of the great variation in signs, symptoms and complications found in those patients labeled asthmatic. A complete history and physical are important for establishing: (1) the frequency and severity of attacks; (2) the presence of wheezing; and (3) those medications used chronically or acutely. Procedural information and assurance are important in preparing all surgical candidates but are especially so for the apprehensive asthmatic.

While the area of premedication has not been investigated under controlled conditions, it is an essential part of every anesthetic. Recent reports5,6,7 have recognized hazards of barbiturate administration in the long-term treatment of asthmatics due to liver enzyme induction, which increases hydrolysis of theophylline and corticosteroids; the result being reduced biological activity of both substances.

A good night's sleep, however, is important pre-operatively and a sedative should be ordered. This sedative may be repeated parenterally on the day of surgery along with the administration of an anticholinergic which, as stated previously, has been shown to decrease airway resistance. Narcotics and tranquilizers remain controversial, as narcotics may cause a respiratory depression persisting into the recovery period while tranquilizers may cause circulatory depression, potentiation of central nervous system depression and an unpredictable duration of effect.8

Steroids are helpful to asthmatic patients for unknown reasons. It is suspected that the cell membranes are stabilized by steroids, thereby influencing the intensity of the release of the mediators of asthma.9 Steroid “preps” and coverage of those patients on steroid therapy should begin 12-24 hours preoperatively and continue 48 hours postoperatively. Hydrocortisone 1-2mg/kg/6-12 hours has been suggested.10

Anesthetic techniques

In Shnider and Papper’s research,11 the incidence of wheezing for a patient under general anesthesia without an endotracheal tube was the same as that of regional anesthesia. Nevertheless, regional anesthesia for surgery of an extremity, or the perineum and inguinal area is the technique of choice. Limitations to this technique are the possibility of peritoneal entry or surgery beyond the 2-3 hour duration of spinal anesthesia, which may necessitate the superimposition of a general anesthetic. According to Gold, the superimposition of a general anesthetic “may be difficult and in instances where the spinal anesthetic has been used for a specific purpose, such as in the patient with a full stomach, the very complication feared may be created.”11

Agents

Halothane is the agent of choice for general anesthesia, as its reputation as a bronchodilator is well known. Still, returning to Shnider and Papper,13 the most common single factor precipitating an acute attack of asthma was the introduction or presence of an endotracheal tube. Therefore, induction of anesthesia with the exception of the emergent full stomach condition, must be a prolonged and thorough procedure to assure that
the patient is at a satisfactory depth of anesthesia prior to the placement of an endotracheal tube. It is equally important to maintain an adequate depth of anesthesia throughout the procedure and to maintain a deep plane of anesthesia at extubation.

Thiopental and d-Tubocurarine are two commonly used drugs which have been implicated in asthmatic attacks/bronchospasm during general anesthesia. Yet, other reports indicate that these drugs "did not cause attacks of asthma out of proportion to the established overall incidence during all forms of general anesthesia." What then is the explanation for the seemingly contradictory reports about these agents? The histamine release associated with curare administration makes it suspect for the patient with reactive airways, despite reports of no adverse effects in defasciculatory dosages. Thiopental, which exerts a parasympathomimetic effect, could be implicated simply because a patient may appear to be asleep up until the moment of airway instrumentation, at which time the familiar bucking and coughing occur, leading to bronchospasm.

Ketamine has been advocated for the anesthetic management of asthmatic patients as a result of ketamine research in which asthmatic patients were included. In three instances under halothane anesthesia, bronchospasm was relieved by ketamine. Another study of airway resistance revealed that ketamine did not affect airway resistance in those patients without pulmonary disorders while making "dramatic" improvements in those patients with moderate to severe increased airway resistance. Though the mechanism responsible is not known, it is speculated that bronchiolar tone is reduced indirectly by the release of endogenous catecholamines associated with ketamine administration.

Enflurane exposure, in two recent reports, has been shown to cause bronchospasm. While one report describes a delayed asthmatic response following occupational exposure to enflurane in an anesthesia resident, the other report details the occurrence of bronchospasm in known asthmatics following enflurane administration. Although enflurane has been used successfully for asthmatic patients, these reports would indicate it be used with extreme caution for known asthmatics.

Muscle relaxants, other than the already mentioned d-Tubocurarine, do not appear to be contraindicated in light of their obvious advantages. Some sources contend that it is best to use a depolarizing agent only due to the necessity of prostigmine, a cholinergic agent, for reversal of nondepolarizers. Succinylcholine, gallamine, and pancuronium bromide have all been administered without adverse effects.

Complications

When bronchospasm does occur during general anesthesia (true bronchospasm is a rare occurrence), the usual cause is inadequate anesthesia, and the anesthetist might first administer a dose of succinylcholine. If this relieves the bronchospasm, the indication is that it was not a true bronchospasm in the first place, but rather an attempt by the lightly anesthetized patient to cough out the endotracheal tube (that is, laryngospasm). If the spasm persists, commence halothane to begin treatment of true bronchospasm.

In years past, additional treatment of further persistent bronchospasm was limited to isoproterenol and/or aminophylline, both of which had the potential to cause sudden and profound side effects in the form of cardiac arrhythmias. New drugs have been synthesized which are, at least in name, beta2 specific or preferential. These are terbutaline sulfate and isoetharine. Though terbutaline in controlled studies has not exhibited the preferential beta2 adrenergic effect (according to product literature), isoetharine, however, has proved to be useful (based on a nebulized metered dose) in providing bron-
chodilatation without cardiac side effects for anesthetized patients. 24

The asthmatic patient presenting for emergency surgery requiring general anesthesia cannot be exposed to a prolonged induction period as recommended previously for scheduled surgical intervention. A rapid sequence induction-intubation is necessary to avoid the possibility of regurgitation-aspiration. The patient should be preoxygenated and given an anticholinergic and a defasciculatory dose of gallamine or pancuronium bromide. Induction should be accomplished with ketamine 2 mg/kg followed by succinylcholine 1.5 mg/kg. Cricoid pressure must be maintained throughout induction-intubation. Once intubation has been accomplished, inhalation of halothane should commence.

Conclusion

Keeping in mind that the practitioner's skill and experience may be more important than the choice of agent or technique, it is suggested that regional anesthesia be administered when possible to the asthmatic patient presenting for surgery. If general anesthesia and endotracheal intubation is the method of choice, the patient should be induced with either halothane and nitrous oxide by mask or intravenous ketamine, Valium®, or Brevital® (not associated with the parasympathomimetic effects of thiopental) or a combination of the above. Instrumentation of the airway should not be attempted until the patient's reflexes have been obtunded as indicated by a 20% reduction of the preinduction blood pressure. However, it should be noted that hypotension may be produced before reflexes are obtunded in some patients.

As stated earlier, maintenance of an adequate plane of anesthesia throughout the surgical procedure and “deep” extubation are equally as important as during the induction period. Preoperative preparation and postoperative care and followup also are not to be neglected.

REFERENCES

(2) Ibid. 463.
(21) Lowry, C. and Fielden, B. 1976. Broncho-
spasm Associated with Enflurane Exposure—Three Case Reports. *Anaesth Intens Care*. 4:257.


**AUTHOR’S NOTE**

The author wishes to state that the opinions in this article are his own and are not to be construed as those of the Department of Anesthesia-Ft. Carson Army Hospital, the U.S. Army Nurse Corps, the Department of the Army, or the Department of Defense.

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