Acute severe asthma: Part 1. Pathophysiology and clinical assessment

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Asthma manifests itself in a two-phase reaction. An early phase, causing bronchial muscle contraction, occurs within minutes after an antigenic stimulus. Within 6 to 18 hours, a late asthmatic response begins. This response is associated with cellular infiltration of the bronchial submucosa by eosinophils, neutrophils, and lymphocytes. The late asthmatic response is most effectively reversed by the use of corticosteroids. Predicting patient outcome solely on the basis of either physical findings, or pulmonary function tests, or historical events is frequently inadequate. Historical events, clinical assessments, and laboratory and pulmonary function measurements are best used in a complementary and integrated fashion to determine appropriate disposition of the acutely asthmatic patient.

(Key words: Bronchial asthma, bronchospasm, status asthmaticus, pulmonary function tests)

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Mortality from bronchial asthma has increased in children and adults in the United States and other western nations during the past 10 years, despite the availability and use of effective pharmacologic agents for the control of the disease. Most of the increased deaths attributable to asthma have occurred in patients 45 years of age or older.¹ The mortality rate among black children and adults has been at least twice as high as that for whites.² During much of the period of increasing deaths caused by asthma, US hospitalizations for treatment of asthma also increased by as much as 50% among adults and 200% among children.³ Approximately 50% of deaths from asthma in this country occur in hospitals. These trends in deaths caused by asthma come at a time when deaths from respiratory diseases other than asthma are gradually decreasing.⁴

The Asthma Mortality Task Force⁵ concluded in 1986 that "fatalities in patients with asthma are related to withholding corticosteroid or to inadequate steroid dosage in patients with severe asthma." The task force recommended an educational campaign among physicians and patients, emphasizing the rationale for using corticosteroids. This article reviews the pathophysiologic aspects of the response to asthma triggers. The assessment of the severity of acute asthma is discussed.

Pathophysiology of asthma

The increased bronchial responsiveness in asthma manifests itself in a two-phase reac-

tion.⁶ The early asthmatic reaction characteristically involves the release of mediators, resulting in bronchial smooth muscle contraction and peribronchial vascular leakage with edema. When limited to a single brief stimulus, this early response begins within minutes, causing a significant decrease in expiratory airflow as measured by reduction of forced expiratory volume in 1 second (FEV₁). The early asthmatic response resolves spontaneously within 1 or 2 hours, or it may be aborted by administration of an aerosol β -adrenergic receptor agonist. Within 6 to 18 hours after an inhaled antigen stimulus, a late asthmatic response begins and a second decrease in FEV₁ occurs. This late response corresponds with cellular infiltration of the bronchial submucosa and mucosa by eosinophils, neutrophils, and lymphocytes. Mucosal edema is increased, and submucosal basement membrane swelling also occurs.^{7,8}

The early asthmatic response, bronchospasm, is relieved most quickly by inhaled β -adrenergic receptor agonist bronchodilators, whereas the late asthmatic response, that is, eosinophilic inflammation, is most powerfully relieved by systemic corticosteroids; somewhat less potently by aerosol corticosteroids.⁹⁻¹² Pretreatment of the patient with β -agonists or theophylline can block the immediate or early response. Pretreatment with cromolyn sodium or sustained treatment with aerosol corticosteroids can prevent or attenuate both the early and late asthmatic responses.⁹ Use of these medications does not cure asthma, but in some situations may help to make the asthmatic patient's airways less sensitive to irritants or other stimuli.

With persistent, effective therapy of several months' duration, the patchily denuded basement membrane of the airway, with its exposed vagal nerve endings, may be covered by a new and intact bronchial epithelium. The intact epithelium of ciliated columnar cells then acts as a protective barrier for the underlying basement membrane. In a significant number of adolescent and young adult asthmatic patients, when this sequence of healing events occurs, the asthma may become asymptomatic. At this point, if results of pulmonary

function studies remain normal for a month or two, the asthma may be said to be in remission. Pharmacologic treatment may be substantially reduced or discontinued with little risk of recurrence for months or occasionally years in some instances.

Protracted asthma unresponsive to outpatient therapy, that is, status asthmaticus, signifies severe airway inflammation with mucous plugging in addition to bronchospasm. The mucous plugging is the major cause of maldistribution of ventilation and hypoxemia that sometimes result in death. The inflammatory process with edema and airway plugging also causes severe air trapping within the lungs with ensuing rupture of alveoli and wicking of air down the bronchial sheath through the hilum into the mediastinum. From the mediastinum, air may be forced into the subcutaneous tissues of the neck and chest wall to produce subcutaneous emphysema, or the air may dissect into the pleural space to result in a pneumothorax. Usually, tension is generated from such air trapping because of mucous plugging and bronchial edema and can lead to catastrophic deterioration. In a recent retrospective review, more than one third of asthmatic patients seen in cardiac arrest had a pneumothorax.¹³

Assessment of severity

The extreme inflammatory changes involved in an acute asthmatic attack usually do not develop in a few hours or even a few days. Commonly, the symptoms, most notably cough, begin several days to several weeks before the emergent episode. Since cough without associated wheezing usually is not ascribed to asthma by most patients and by many physicians, the bronchial inflammation producing the cough continues to increase while the patient—and sometimes the physician—inappropriately medicates the symptom with expectorants, decongestants, or sometimes even cough suppressants. It must be emphasized that a cough persisting more than a few hours in a previously diagnosed asthmatic patient should be viewed as a harbinger of a potentially severe or life-threatening attack. In one study, 12% of patients coming to the emergency room

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BRIEF SUMMARY

Contraindications: Severe LV dysfunction (see **Warnings**), hypotension (systolic pressure < 90 mm Hg) or cardiogenic shock, sick sinus syndrome (if no pacemaker is present), 2nd- or 3rd-degree AV block (if no pacemaker is present), atrial flutter/fibrillation with an accessory bypass tract (eg, WPW or LGL syndromes), hypersensitivity to verapamil.

Warnings: Verapamil should be avoided in patients with severe LV dysfunction (eg, ejection fraction < 30%) or moderate to severe symptoms of cardiac failure and in patients with any degree of ventricular dysfunction if they are receiving a beta-blocker. Control milder heart failure with optimum digitalization and/or diuretics before Calan SR is used. Verapamil may occasionally produce hypotension. Elevations of liver enzymes have been reported. Several cases have been demonstrated to be produced by verapamil. Periodic monitoring of liver function in patients on verapamil is prudent. Some patients with paroxysmal and/or chronic atrial flutter/fibrillation and an accessory AV pathway (eg, WPW or LGL syndromes) have developed an increased antegrade conduction across the accessory pathway bypassing the AV node, producing a very rapid ventricular response or ventricular fibrillation after receiving LV verapamil (or digitalis). Because of this risk, oral verapamil is contraindicated in such patients. AV block may occur (2nd- and 3rd-degree, 0.8%). Development of marked 1st-degree block or progression to 2nd- or 3rd-degree block requires reduction in dosage or, rarely, discontinuation and institution of appropriate therapy. Sinus bradycardia, 2nd-degree AV block, sinus arrest, pulmonary edema and/or severe hypotension were seen in some critically ill patients with hypertrophic cardiomyopathy who were treated with verapamil.

Precautions: Verapamil should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of overdosage. Verapamil may decrease neuromuscular transmission in patients with Duchenne's muscular dystrophy and may prolong recovery from the neuromuscular blocking agent vecuronium. It may be necessary to decrease verapamil dosage in patients with attenuated neuromuscular transmission. Combined therapy with beta-adrenergic blockers and verapamil may result in additive negative effects on heart rate, atrioventricular conduction and/or cardiac contractility; there have been reports of excessive bradycardia and AV block, including complete heart block. The risks of such combined therapy may outweigh the benefits. The combination should be used only with caution and close monitoring. Decreased metoprolol and propranolol clearance may occur when either drug is administered concomitantly with verapamil. A variable effect has been seen with combined use of atenolol. Chronic verapamil treatment can increase serum digoxin levels by 50% to 75% during the first week of therapy, which can result in digitalis toxicity. In patients with hepatic cirrhosis, verapamil may reduce total body clearance and extrarenal clearance of digitoxin. The digoxin dose should be reduced when verapamil is given, and the patient carefully monitored. Verapamil will usually have an additive effect in patients

receiving blood-pressure-lowering agents. Disopyramide should not be given within 48 hours before or 24 hours after verapamil administration. Concomitant use of flecainide and verapamil may have additive effects on myocardial contractility, AV conduction, and repolarization. Combined verapamil and quinidine therapy in patients with hypertrophic cardiomyopathy should be avoided, since significant hypotension may result. Concomitant use of lithium and verapamil may result in a lowering of serum lithium levels or increased sensitivity to lithium. Patients receiving both drugs must be monitored carefully. Verapamil may increase carbamazepine concentrations during combined use. Rifampin may reduce verapamil bioavailability. Phenobarbital may increase verapamil clearance. Verapamil may increase serum levels of cyclosporin. Verapamil may inhibit the clearance and increase the plasma levels of theophylline. Concomitant use of inhalation anesthetics and calcium antagonists needs careful titration to avoid excessive cardiovascular depression. Verapamil may potentiate the activity of neuromuscular blocking agents (curare-like and depolarizing); dosage reduction may be required. There was no evidence of a carcinogenic potential of verapamil administered to rats for 2 years. A study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. Pregnancy Category C. There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor, and delivery only if clearly needed. Verapamil is excreted in breast milk; therefore, nursing should be discontinued during verapamil use.

Adverse Reactions: Constipation (7.3%), dizziness (3.3%), nausea (2.7%), hypotension (2.5%), headache (2.2%), edema (1.9%), CHF, pulmonary edema (1.8%), fatigue (1.7%), dyspnea (1.4%), bradycardia: HR < 50/min (1.4%), AV block: total 1°-2°-3° (1.2%), 2° and 3° (0.8%), rash (1.2%), flushing (0.6%), elevated liver enzymes, reversible non-obstructive paralytic ileus. The following reactions, reported in 1.0% or less of patients, occurred under conditions where a causal relationship is uncertain: angina pectoris, atrioventricular dissociation, chest pain, claudication, myocardial infarction, palpitations, purpura (vasculitis), syncope, diarrhea, dry mouth, gastrointestinal distress, gingival hyperplasia, ecchymosis or bruising, cerebrovascular accident, confusion, equilibrium disorders, insomnia, muscle cramps, paresthesia, psychotic symptoms, shakiness, somnolence, arthralgia and rash, exanthema, hair loss, hyperkeratosis, macules, sweating, urticaria, Stevens-Johnson syndrome, erythema multiforme, blurred vision, gynecostasia, galactorrhea/hyperprolactinemia, increased urination, spotty menstruation, impotence.

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with an acute asthmatic episode denied having dyspnea; however, all reported coughing.¹⁴ Wheezing or dyspnea (or both) usually indicates a more advanced stage of asthma with severe inflammation, edema, and mucous plugging. This stage may be reached by gradual progression over several days or weeks from the cough stage, or it may occur over a few hours after a concentrated exposure to antigens or irritants. However, systemic anaphylaxis should be considered in patients having a sudden onset of difficulty breathing or wheezing.

Assessment of the degree of ventilatory impairment begins with simple observations. Speech broken into two- or three-word phrases because of dyspnea generally indicates significant alteration in pulmonary mechanics. Respiratory rates of more than 25 breaths per minute that do not slow to between 12 to 20 breaths per minute shortly after treatment are reason for concern and closer monitoring of pulmonary function. A heart rate of more than 110 beats per minute or systolic and diastolic hypertension are signs of disturbed pulmonary mechanics and hypoxemia.

Diaphoresis, sternocleidomastoid muscle contraction, and paradoxical pulses are associated with severe airway obstruction.¹⁵⁻¹⁷ Diaphoresis may be more specific for severe airflow obstruction than accessory muscle use or paradoxical pulse. The accessory muscle use and paradoxical pulse tend to occur together.¹⁵ Sterncleidomastoid muscle contraction and paradoxical pulse have been noted to be absent in 52% of patients with an $FEV_1 < 1$ L.¹⁶ Inappropriate or paradoxical inward motion of the lower anterior chest wall and upper abdomen during inspiration is an indication of diaphragmatic overload.¹⁸ The efficiency of diaphragmatic contraction is reduced because it is displaced downward by the overdistended lungs, and the contractile force required during inhalation is increased because of inspiratory airflow obstruction and air trapping. Particularly ominous are signs of confusion, agitation, or stupor. Mechanically assisted ventilation is often needed at this point to prevent bradypnea, apnea, and death.

Objective measurements of pulmonary me-

Figure 1. Nomograms for prediction of normal forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV_1) in adult men and women. (Reprinted by permission from Morris JF: Spirometry in the evaluation of pulmonary function. West J Med 1976;125:110.)

chanics and arterial blood gases are often used to assess the severity of symptomatic asthma and to facilitate decisions regarding need for hospitalization or timeliness of discharge or both. Arterial blood gases generally reveal hypoxemia and hypocapnia. In severe episodes of asthma, eucapnia or hypercapnia is often found. When arterial PCO_2 measures 40 mm Hg or more, the FEV_1 is less than 25% of

predicted normal value in more than 90% of cases.^{19,20} Essentially all such patients will require admission to the hospital or intensive, prolonged therapy to restore ventilatory function adequate to allow discharge.

Concern has been raised about relying on patient signs and symptoms to determine patient disposition.^{15,16,21,22} McFadden and associates²¹ found that when patients were relieved of dyspnea and subjective wheezing, they only produced an FEV₁ of 49% and peak expiratory flow rate (PEFR) of 43% of predicted normal. When objective findings of retractions and wheezing were reversed, patients had an FEV₁ of 63% and PEFR of 54% of predicted normal. Pulmonary function testing has been advocated as an important tool for assessing response to therapy and determining disposition of the patient.^{21,23-25} Measurements of the PEFR or FEV₁ or both may be obtained in patients who are able to cooperate and perform the forced expiratory maneuvers. The PEFR or FEV₁ or both may be measured initially and after subsequent aerosol bronchodilator treatments until a satisfactory ventilatory response is achieved. The measured value of FEV₁ may be compared with the predicted normal FEV₁ for the patient based on sex, height, and age (Fig 1).

Pretreatment FEV₁ or PEFR values have not been shown to be accurate in predicting patient response to therapy.^{16,23,26-28} Also, proposed clinical index scoring systems^{14,29} based on presenting signs and symptoms have not been found accurate in predicting patient outcome.^{16,30,31} Several authors have suggested that decisions based on the degree of change in pulmonary function tests, after treatment, are useful^{16,23,32}; however, their predictive accuracy has not been validated in prospective studies. Martin and coauthors²³ suggested that patients having a 20% improvement in PEFR after initial therapy were unlikely to require

Figure 2. Estimated frequency distribution of the posttreatment forced expiratory volume in 1 second (FEV₁) in three groups of patients. (Adapted by permission from Nowak and colleagues.¹⁹) The vertical bar represents an FEV₁ of 2.1 L. The shaded area represents those patients successfully discharged and having FEV₁ < 2.1 L. If an FEV₁ ≤ 2.1 L had been used as a recommendation for admission, one third of patients would have been admitted unnecessarily.²⁴

hospitalization. An absolute improvement of < 400 mL in FEV₁ was associated with a high relapse rate in the study by Kelsen and associates.¹⁶ Nowak and coworkers³² found that patients with a PEFR < 100 L/min who did not demonstrate an improvement of 60 L/min after initial therapy often required hospitalization. An FEV₁ > 2.1 L or PEFR > 300 L/min has been advocated as a criterion for discharge.^{19,24,32} These values correspond to approximately 60% of predicted normal values for the average adult man. However, clinical decisions based solely on pulmonary function values at disposition will not accurately identify all patients who can be discharged successfully (Fig 2).

Worthington and Ahuja²⁶ found that 72% of the admissions would have been unnecessary if all patients unable to achieve a FEV₁ ≥ 2.1 L/min were admitted. They propose that patients with a final FEV₁ > 2.4 L/min can be discharged successfully with a high degree of confidence; however, this value would have identified only 41% of their patients who were discharged without relapse. Another complicating factor is that older asthmatic patients may have chronic obstructive airway disease

affecting their ventilation status. Use of FEV₁ or PEFR values for these patients necessitates knowledge of their normal asymptomatic values if the physician is to correctly evaluate the patient's responsiveness to therapy.

Pulmonary function testing is especially useful in the evaluation of high risk patients. Those who are currently taking or who have recently received corticosteroids, patients treated on an outpatient basis within 10 days, those with repeated previous hospitalizations, or patients with a history of mechanically assisted ventilation for status asthmaticus should demonstrate adequate FEV₁ or PEFR function before discharge.

This article has discussed the pathophysiologic aspects of asthma and reviewed various recommendations for assessment of the acute attack. Part 2 will provide an overview of current therapy for this serious health problem.

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