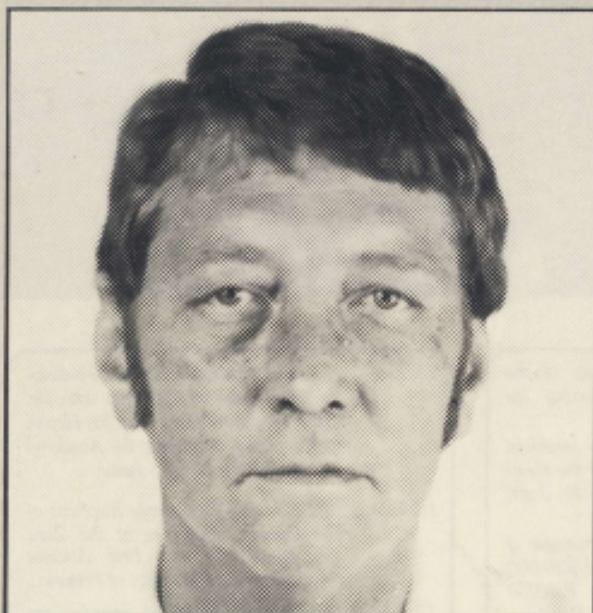


Acute asthma

Approach to initial management and results evaluated

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Summary

Pathophysiology and symptoms of acute asthma are described. Signs indicating pulmonary and cardiovascular system involvement respectively and the use of special and laboratory tests are discussed. Management and outcome of acute asthma attacks in 475 patients using the standard programme of the Emergency Unit of the H.F. Verwoerd Hospital are described.

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KEYWORDS: Asthma; Airway Obstruction; Respiratory Insufficiency; Respiratory Function Tests; Emergency Service, Hospital; Diagnosis, Laboratory; Bronchial Diseases.

Asthma is characterised by an increased responsiveness of the tracheobronchial tree to a multiplicity of stimuli resulting in recurrent episodes of generalised airway obstruction that abate spontaneously or with treatment¹. It is physiologically manifested by narrowing of the air passages and may be relieved spontaneously or as a result of therapy. The disease is episodic with acute exacerbations interspersed with symptom-free periods.

Clinically acute asthma is manifested by paroxysms of dyspnea, cough and wheezing. The obstruction can vary in severity and can occur for a short period, or persist for hours, days or even weeks. The latter is referred to as status asthmaticus.

The diagnosis usually presents few problems but unexpected deaths occur all too frequently.

The usual reason for the mortality is misassessment of the severity of the pathophysiological changes followed by under-treatment.

Acute asthma

It is, therefore, important to realise that the clinical features of the patient do not reflect the actual physiological alterations nor the degree of response to treatment.

It is recommended that acute asthma be treated until the FEV₁ is greater than 2,1 litres (more than 60 percent predicted) or PEF_R is over 300 litres per minute (more than 60 percent predicted) irrespective of apparent clinical improvement.

PATHOPHYSIOLOGY

The hallmark of acute asthma is narrowing of the airways which is due to:

- (1) bronchial smooth muscle spasm
- (2) airway inflammation with mucosal oedema and
- (3) hypersecretion of thick, tenacious mucus and decreased clearance.

The smooth muscle spasm accounts for the rapidly reversible types of airway obstruction, whereas the inflammatory oedema and mucous plugging of the airways account for the nonresponsive forms of the disease². The mechanical effect is air trapping which results in deterioration of lung functions which has invariably occurred over a period of days or weeks prior to the acute attack. The net result of this deterioration is resistance to breathing due to increase in airway resistance which in turn is responsible for:

- (1) increased work of breathing
- (2) decreased forced expiratory volumes
- (3) decreased airflow rates
- (4) decreased elastic recoil of the lungs
- (5) hyperinflation and
- (6) ventilation-perfusion ratio mismatching.

An abnormal distribution of ventilation (due to uneven airway constriction) and pulmonary bloodflow (due to hypoxic vaso-constriction) exists. This results in carbon dioxide build-up which is responsible for an increased ventilatory drive. The latter explains the finding that hypercapnia is seen only in the minority of patients and indicates a lack of physiological compensation which should be viewed as most serious. The ventilation-perfusion mismatching is furthermore responsible for hypoxia (hypoventilation). These changes, of which the first two are the most important, can be assessed with a blood gas analyses. Anything which is inclined to decrease muscle ability such as fatigue or lack of calorie intake would be predisposing to respiratory failure. For the same reason the administration of any agent (medication) which might depress muscular action should be avoided.

SYMPTOMS

Symptoms are:

- (1) **dyspnoea** is the major complaint. This is a reflection of the increased workload of breathing
- (2) **cough** which usually produces stringy, yellow-green sputum due to eosinophiles. Coughing is also inclined to exacerbate airway obstruction. It is as well to remember that accumulation in the sputum of both polymorphs and eosinophiles can produce yellow-green sputum. Sputum of this colour does therefore not always indicate infection and microscopic examination should be performed to determine the reason for the colour of the sputum.
- (3) **wheezing** increases as the attack progresses but can disappear on account of improvement or as a result of the "silent chest" of respiratory failure.

The subjective assessment of wheezing does not indicate the severity or duration of the asthmatic attack³.

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SIGNS OF PULMONARY SYSTEM INVOLVEMENT

- (1) **Dyspnoea** which is obvious: the patients insist on sitting in an upright position. (This causes a reduced venous return and lessens the effect of intra-abdominal pressure on the thoracic cage.) The shoulder girdle is thrust forward so as to complement the action of the accessory muscles which may become necessary for breathing.
- (2) **Cyanosis** is a later sign and is rarely seen because the usual respiratory alkalosis produced (as a result of hyperventilation) causes a leftward shift of the oxy-hemoglobin dissociation curve^{4,5}. The development of central cyanosis is a serious sign because the asthmatic patient normally hyperventilates even in the presence of moderately severe obstructive airway disease. Because hyperventilation continues during sleep it cannot be explained on emotional grounds. The respiratory centre is stimulated via the vagus and this results in hyperventilation. The net result is an elevation of PO₂ and a reduced PCO₂ during the early phase of the disease. It must, therefore, be emphasised that the presence of central cyanosis indicates serious respiratory involvement.
- (3) **Alteration in mentation** or consciousness is indicative of severe airway obstruction. Agitation and restlessness, too, indicate hypercapnoea although non-specific⁶.

SIGNS OF CARDIOVASCULAR SYSTEM INVOLVEMENT:

- (1) *tachycardia* greater than 110 per min indicates a serious attack. A heart rate of greater than 130 per minute indicates severe airway obstruction⁸. (FEV_1 less than or equal to 1 litre), but many patients with equally severe asthma have a normal pulse rate (less than 100/minute)⁹. As the asthma is successfully treated the heart rate will drop. However the heart rate will also drop with relatively small improvements in airflow and thus still persistently severe asthma⁹.
- (2) *tachypnoea* is a frequent finding in moderate as well as in severe asthma and is, therefore, also not a reliable predictor of severity of an attack.
- (3) *pulsus paradoxus* of more than 10 mmHg indicates severe asthma. Pulsus paradoxus is caused by arterial pressure reduction on inspiration. It is dependent on hyperinflation and is directly related to the degree of airway obstruction. The relation to FEV_1 has been proved in that pulsus paradoxus is not present in patients with a FEV_1 higher than 60% of normal but is present in all patients with a FEV_1 less than 20% and a significant arterial paradoxus is found in two thirds of patients with FEV_1 of less than 40%. An inspiratory fall in systolic pressure of more than 10 mm mercury is significant proof of airway obstruction¹⁰. It is thus an exaggeration of the normal reduction in arterial pressure (usually 5 mm of mercury) during inspiration and is associated with a combination of high lung volume and intrathoracic pressure changes¹¹. (The degree of paradox is measured with a blood pressure manometer pumped up well above systolic pressure, releasing the air slowly and assessing the effect of inspiration on the blood pressure reading). Pulsus paradoxus usually disappears rapidly, usually within a few hours of the initiation of therapy.
- (4) *the use of accessory muscles* indicates the presence of marked hyperinflation and a FEV_1 of less than 1 litre. It must again be emphasized that the absence of these signs does not rule out severe airway obstruction and that they disappear with only minimal improvement in airflow but still persistent asthma present^{12,9,13,14}.
- (5) *with auscultation the characteristic findings are* prolongation of the expiration phase and the presence of expiratory bronchi. Equal inspiratory and expiratory phases indicate severe obstruction. A "silent" chest can indicate a serious degree of obstructive airways disease.
- (6) *hyperinflation or thoracic overinflation* is present during episodes of acute asthma and alters with the degree of airway obstruction. A degree of hyperinflation remains temporarily present even after bronchospasm has been relieved.

SPECIAL AND LABORATORY INVESTIGATIONS

- (1) *Chest Radiographs*. Chest roentgenograms should be obtained to exclude underlying pathology or to demonstrate complications. These include pneumonia, mucous obstruction with atelectasis, pneumothorax and pneumomediastinum. The degree of hyperinflation can also be assessed to some extent.
- (2) *Arterial blood gas analysis* should be done on admission, during treatment and prior to final decision on the fate of the patient regarding discharge or admission to the ward or to the intensive care ward^{15,16}.

This investigation is still recommended in spite of apparently poor correlations between gas tensions and severity of asthma as measured by pulmonary function testing¹³ which is (according to some workers) not useful in predicting patient outcome¹⁷. According to these workers arterial blood gases are not sensitive in the assessment of asthma severity, and severe airway obstruction as measured by pulmonary function testing may be associated with relatively normal blood gas analysis¹⁷.

In our setting we find blood gas analysis most useful and the investigation is performed routinely on admission and during treatment. Hypoxaemia with hypocapnia is the usual finding. Carbon dioxide retention is less common in patients suffering from severe asthma than in those with chronic obstructive lung disease with superimposed acute infections. The common blood gas picture is: reduced PO_2 to 60 mmHg with a reduced PCO_2 . Bearing in mind the existing hyperventilation, an elevated PCO_2 and possibly even a normal PCO_2 would indicate serious impairment of ventilation and vigorous therapy is called for.

- (3) *Electrocardiogram*. The changes are non-specific but can be of value¹¹. The possible changes are the following: Sinus tachycardia is always present frequently accompanied by other changes. The most common changes are right axis deviation, clockwise rotation of the heart, P-pulmonale, various S-T segment and T wave abnormalities. Sinus tachycardia diminishes as airways obstruction is relieved by therapy and improvement of any severe electrocardiographic abnormalities usually follows rapidly after the initiation of therapy. All the changes revert to normal after relief of the asthmatic episode.
- (4) *Pulmonary function testing* is the most accurate assessment of airways obstruction. The FEV_1 is the best single spirometric test with which to assess airway obstruction and responsiveness to treatment¹⁹. The PEFR (peak flowrate) as measured by the Mini-Wright peak flow meter correlates well with the FEV_1 and the Standard-Wright peak flow meter and should be utilized in the Emergency Department setting^{20,21}. A valid measurement for PEFR requires a brief maximal exhalation from total lung capacity, since it is effort and volume dependent²².

Cessation of therapy should preferably not be based on clinical improvement only but rather on all available laboratory assessments.

It is recommended that acute asthma be treated until the FEV₁ is greater than 2.1ℓ (more than 60% predicted) or PEFR is over 300 litres per minute (more than 60% predicted) irrespective of apparent clinical improvement²¹.

MANAGEMENT OF THE ACUTE ASTHMA ATTACK

Oxygen administration

A Venturi-type mask or long nasal catheters are recommended for 100% oxygen administration.

Intravenous fluid administration

An infusion of 5% dextrose in water is preferred to enhance rehydration of interstitial tissues by which the viscosity of secretion is reduced. Provided there are no contra-indications for fluid administration, one litre is administered during the first hour and a second litre during the following three hours.

Bronchodilator therapy

Bronchodilators intravenous are routinely administered in a piggy-back infusion (200mℓ dextrose in water) and not in the infusion for rehydration. Provided the heart rate of the patient is less than 115 per minute 500 mg aminophyllin and 10mcg hexoprenaline (beta-adrenergic stimulant) is added to the piggy-back infusion. In the event of a heart rate above 115 per minute the hexoprenaline must be omitted. The solution is titrated to ensure a loading dose of aminophyllin at a rate of 6 mg/Kg per hour during the first 20 minutes. The maintenance dose for aminophyllin is 0.9 mg/Kg/hour for the average patient. However, a decreased clearance of aminophyllin is found in the aged and in patients with congestive cardiac failure, pulmonary oedema, pneumonia, congestion of the liver due to right heart failure and in liver disease (smokers have an improved clearance). Surveillance for side effects is indicated. These are dysrhythmias, convulsions, cardio-respiratory arrest, nausea, vomiting with abdominal discomfort, headache and anxiety. To summarise — for practical administration the maintenance dosage is 0.9 mg/Kg/hour except for patients

- over 50 years
- with chronic obstructive disease
- with congestive cardiac failure and
- with advanced liver disease.

For this group of patients the dosage is halved.

Corticosteroid therapy

As per usual with corticosteroids, there is an ongoing debate concerning the usefulness of these agents in acute asthma. Some investigators find no evidence of improvement after steroid usage²³, whereas others note significant improvement in pulmonary functioning testing as early as one hour after intravenous administration, with peak effects in four to eight hours^{24,25}. In our setting it is mandatory to administer steroids to all patients with acute asthma because mucosal oedema is part of the primary problem and the anti-inflammatory action of steroids may well be of value^{26,27}. The dosage for hydrocortisone is 200 mg intravenously as a bolus (2 mg/Kg²⁸) repeated every four to eight hours until acute state has subsided, then followed by oral administration. In the event of patients already receiving steroids the initial dose is 400 mg (4 mg/Kg). Others believe higher doses (500 mg methylprednisolone) are required initially to overcome steroid resistance²⁹. However the initial dose for methylprednisolone is mostly a slow bolus intravenous administration of 100 mg.

Inhalation therapy

Inhalation therapy is started with a Hudson mask using the following solution: 1 part hexoprenaline, 2 parts saline. A high oxygen flow of approximately six litres is used to achieve a ventury effect with a very fine spray. The physiotherapist would use a Devulbis with the following solution: hexoprenaline 10 mℓ and normal saline 20 mℓ and bronkese 10 mℓ added.

Physiotherapy

In our setting physiotherapy in combination with inhalation therapy is used extensively. Physiotherapy is instituted after the initial infusion of dextrose and water and the loading dose of aminophyllin.

EMERGENCY DEPARTMENT SURVEILLANCE

Patients should be kept in the Emergency Department for four to six hours surveillance except in the event of the high-risk patient or life-threatening disease in which case admission to a pulmonary intensive-care unit should be ensured immediately after stabilization.

Assessment for discharge

Assessment is not made on clinical grounds in this unit but the parameters used are repeated blood gas analysis at least on admission, during the treatment period and prior to discharge and on measurement of peak air flow rate (PEFR). In the event of the peak flow being less than 50% after treatment the patient is admitted. If the peak flow is 50 to 60% treatment is continued and the peak flow is reassessed.

Although the clinical status is of lesser importance it is still necessary to evaluate for recovery before discharging the patient. The expected order of recovery is: the disappearance of the pulsus paradoxus followed by disappearance of accessory muscle use; then disappearance of electrocardiogram abnormalities, loss of the sense of dyspnoea and lastly a decrease in the intensity of wheezing.

IDENTIFICATION OF THE HIGH RISK PATIENT

This all-important decision is based on the observation that the following entail a high risk:

- (1) a history of previous life-threatening attack, an attack regardless of regular high doses of steroids and cases where previously optimal therapy is now failing
- (2) the use of accessory muscles
- (3) central cyanosis
- (4) pulsus paradoxus in excess of 10 mm
- (5) presenting with exhaustion
- (6) those with disturbance of level of consciousness
- (7) a "silent" or quiet chest on auscultation in the patient with tachypnoea
- (8) a PO₂ less than 60 mm mercury
- (9) a PCO₂ above 55 mm mercury
- (10) chest radiograms revealing serious underlying pathology
- (11) a peak flow (PERF) that is unresponsive to treatment
- (12) all first attack patients
- (13) all young patients
- (14) electrocardiogram indicative of P-pulmonale and right ventricular hypertrophy.

Deterioration of a patient with acute asthma despite the above described management program warrants intubation and mechanical ventilation (volume ventilation).

In this unit sedation is ensured with diazepam for intubating purposes and pancorium bromide (pavulon) 4 mg is afterwards administered to achieve relaxation of bronchial constriction (to allow trapped air to escape) and of respiratory muscles (to enable mechanical ventilation.) Pancorium bromide is a neuroblocking agent with a relatively short action.

RESULTS OF 475 PATIENTS TREATED FOR ACUTE ASTHMA IN THE EMERGENCY UNIT OF THE HF VERWOERD HOSPITAL

The above described management programme is the standard regimen in this unit. During the period 1 August 1982 to 31 March 1983 a total of 475 patients presenting with acute asthma were treated following the described regimen.

Age incidence:

0-12	—	14 percent
13-20	—	7 percent
21-30	—	10 percent
31-40	—	14 percent
41-50	—	6 percent
51-plus	—	49 percent

Males = Females ($\pm 5\%$)

Investigations

1. Chest radiograms	2. Peak flow estimation
Negative — 27%	PEFR less 50% — 18%
Abnormality — 73%	PEFR 50 — 60% — 22%
	PEFR more 60% — 60%
3. Blood gas analysis	
Normal	— 15%
Respiratory alkalosis	— 6%
Respiratory acidosis	— 46%
Mixed respiratory and metabolic acidosis	— 33%
PO ₂ less 60 mmHg after treatment	— 18%
PCO ₂ above 55 mmHg after treatment	— 10%

Final outcome

Admission to ICU: 10%
 Admission to general ward: 8%
 Discharge after treatment: 72%
 Re-admission during the first 48 hours following discharge:
 0,6% of those discharged (2 patients)

CONCLUSION

This management regimen appears to be effective. Patients with acute asthma require:

- (1) In-depth evaluation.
- (2) Surveillance period of no less than 4 hours.
- (3) Intensive therapy.
- (4) Re-evaluation after a period of intensive therapy to decide final actions to be taken on available laboratory assessments.

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