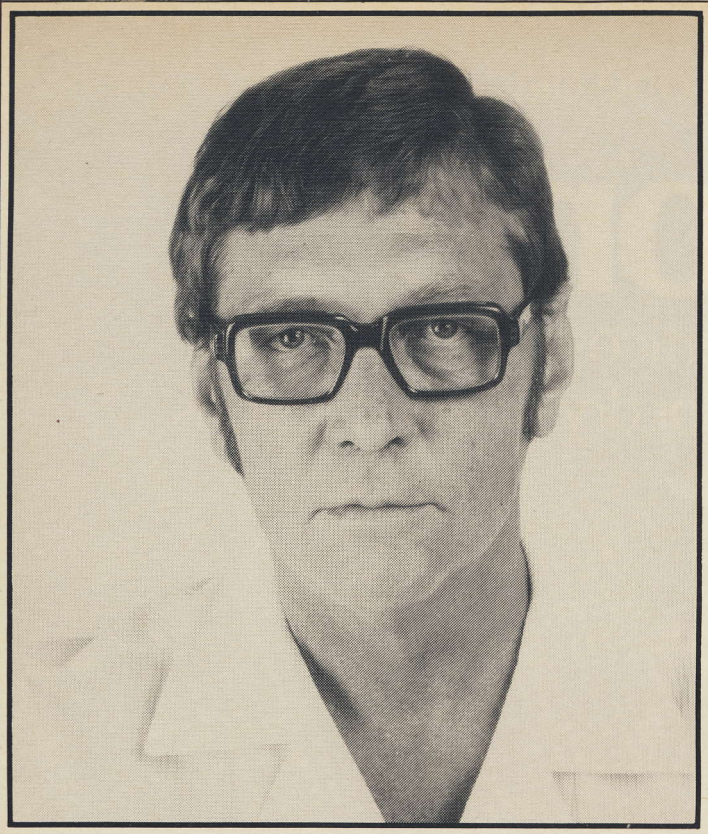


Acute Asthma in the Casualty Department, Emergency room assessment and management at H.F. Verwoerd Hospital Pretoria

Dr C.J. van der Merwe and Dr J.A. Ker.

OPSOMMING:
Die belangrike kliniese tekens van akute asma word bespreek. Insiggewende inligting in verband met die waarde van eenvoudige kliniese waarnemings soos byvoorbeeld die graad van pulsus paradoxus word verduidelik en die betekenisvolle gebruik van spesiale ondersoeke word uiteengesit. Noodbehandeling geniet dan aandag op 'n redelik didaktiese maar gebalanseerde manier.

Dr. C.J. van der Merwe graduated with MBChb at the University of Pretoria in 1955. After completion of the Intership at the Krugersdorp General Hospital he returned to the then Pretoria General Hospital as Senior House Officer to the Department of Surgery.

During 1957 he travelled to the United Kingdom and after six months as Senior House Officer he was appointed Surgical Registrar to the Birmingham Regional Hospital Board. A post he held for 2½ years.

On return to South Africa he worked as Surgical Registrar at King Edward VIII Hospital Durban and after 2 years was appointed Senior Surgical Registrar to the Department of Plastic and Reconstructive Surgery, Wentworth Hospital, Durban.

After a second visit to the United Kingdom where he was appointed Senior Surgical Registrar he returned to the R.S.A. and practised as a family practitioner in Pretoria.

During 1970 he was appointed to the staff of the H.F. Verwoerd Hospital. In 1973 he was appointed Senior Medical Officer and in 1974 Principle Medical Officer in charge of the Casualty Department of the H.F. Verwoerd Hospital, a position he is still holding at present.

In 1977 he gained the qualification M. Prax Med and was appointed Senior Lecturer to the Department of Family Medicine, University of Pretoria.

Recognising acute asthma presents few problems. It has nevertheless become evident that "sudden unexpected" deaths frequently occur. The reason for these catastrophies is usually misassessment and undertreatment.

It is important to emphasise that the clinical features of asthma do not necessarily reflect the physiological alterations present. It is for the same reason that the degree of response to treatment may be poorly correlated with existing physiological changes associated with inadequate treatment.

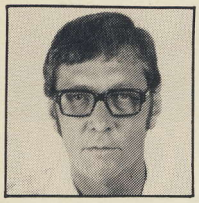
It can thus be fallacious to accept that an improvement in the clinical picture is necessarily an indication for cessation of therapy. It is therefore crucial to utilize available laboratory assessment to prevent undertreatment and too early discharge.

Pathophysiology of acut asthma:
The pathophysiologic hallmark of asthma is a reduction in airway diameter. This is brought about by: (1) Contraction of smooth muscle; (2) Oedema of bronchial wall; (3) Thick secretions.

The relative contribution of each of these to the patients' overall ventilatory impairment vary. The net result however is: (1) An increase in airway resistance; (2) decreased forced expiratory volumes and flow rates; (3) hyperinflation of the lungs; (4) increased work load of breathing; (5) abnormal ventilation and perfusion; (6) abnormal blood gasses.

Assessment of the acute asthmatic episode:
Assessment is based on questioning and careful clinical examination assisted by laboratory and Röntgen examination. It is important to realize that a given episode of asthma may be of greater severity than is superficially indicated by the clinical picture.

Symptoms:
Dyspnoea is the major complaint. This is a reflection of the increased workload of breathing. An acute asthma attack can be a serious problem in patients with a



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history of previous hospital admissions for a similar attack, in those who had been on steroid therapy and in patients on whom the usual therapy has now become ineffective.

SIGNS:

PULMONARY SYSTEM

Dyspnoea

Dyspnoea is obvious in these patients and expiration is prolonged.

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 compliment the action of the accessory muscles which may become necessary for breathing.

Retraction of the sternomastoid muscle during inspiration is obvious in the patient with a serious asthma attack.

Cyanosis:

The development of central cyanosis is a serious sign because the asthmatic patient normally hyperventilates even in the presence of moderately severe obstructive airways disease. Because hyperventilation continues during sleep it can not be explained on emotional grounds. The increased ventilatory drive is due to afferent vagal stimulation. Asthma is a condition which causes stimulation of the respiratory centre via the vagus and results in hyperventilation. The net result is an elevation of PO_2 and a reduced PCO_2 during the early phase of this disease.

It must be emphasised that the presence of central cyanosis indicates serious respiratory disease.

Auscultation:

The characteristic ausculatory findings are of prolongation of expiration and the presence of expiratory ronchi.

Equal inspiratory and expiratory phases indicates severe obstruction. It is important to realise that a "silent" chest can indicate a serious degree of obstructive airways disease.

Hyperinflation:

Thoracic overinflation is present during episodes of acute asthma(1) and alters with the degree of airway obstruction. Even when bronchospasm has been relieved, some degree of temporary hyperinflation may still persist.

CARDIOVASCULAR SYSTEM:

Tachycardia:

A sinus tachycardia greater than 110/min. can be an indication of a severe attack.(2) The degree of tachycardia may be related to the level of arterial hypoxaemia.(3) A drug induced tachycardia must be born in mind in patients receiving treatment.

Pulsus paradoxus:

This is an exaggeration of the normal reduction in arterial pressure (usually 5mm of mercury) during inspiration and is associated with a combination of high lung volume and increased intra thoracic-pressure changes.(4) 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 66% patients with FEV_1 of less than 40%. An inspiratory fall in systolic pressure(5) of more than 10 mgm mercury is significant proof of airway obstruction with severe hyperinflation of the lungs. The degree of paradox is directly related to the degree of airways obstruction. Pulsus paradoxus usually disappears rapidly, usually within a few hours of the initiation of therapy. (The degree of paradox is measured with a blood pressure manometer).

SPECIAL INVESTIGATIONS PERFORMED IN THIS PROGRAMME

Bloodgas analysis:

This investigation is performed routinely on admission and during treatment. Hypoxaemia with hypocapnia(6) is the usual finding. Carbon dioxide retention appears to be less common in patients suffering from severe asthma than in those with chronic obstructive disease with superimposed acute infections. A reduced PO_2 to 60mm Hg (with a reduced PCO_2) is the common bloodgas picture. An elevated PCO_2 (and possibly even a normal PCO_2) indicates serious impairment of ventilation and vigorous therapy is called for.

Electrocardiogram:(7)

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, S-T segment or T wave abnormalities.

Sinus tachycardia diminishes progressively as the asthma diminishes and improvement of the severe electrocardiographic abnormalities usually follows rapidly after the initiation of therapy and all these changes revert to normal after relief of the asthmatic episode.

Chest Röntgenogram:

The most important use of the X ray is to exclude complications. The most common is pneumonia or atelectasis due to mucus obstruction(8) especially in the right middle lobe. The changes of a pneumothorax and a pneumomediastinum can be demonstrated. The degree of hyperinflation is difficult to evaluate.

MANAGEMENT OF ACUTE ASTHMA:

Oxygen:

Either a concentration mask (Venturi type) is used or long nasal catheters.

Fluid administration:

An infusion of 5% Dextrose and water is preferred to enhance rehydration of the interstitial tissues and thus reduce the viscosity of the bronchial secretions. As much as 1 litre of the solution is administered during the first hour (provided no contra-indication for fluid loading exists) and an additional 1 litre during the following 3 hours.

Bronchodilators:

A piggy back infusion (150ml Dextrose and water) is chosen for the administration of drugs with bronchodilating properties. For the patient with a heart rate of 115/min. or less aminophylin 500mgm and 10mgm hexoprenoline (B-adrenergic stimulant) is added to the infusion. This solution is titrated for the administration of aminophyllin at a rate of 6mgm/kg/hour during the first 20 minutes followed by a maintenance dosage of 0,9mgm/kg/hour. In the event of the heart rate being more than 115/min., the hexoprenaline is omitted and the aminophylin dosage is adjusted accordingly.

Corticosteroid therapy:

The administration of steroids during an acute attack of asthma is mandatory. Hydrocortisone 200mgm I.V. is administered with increased dosage to 400mgm in patients already receiving steroids. It must be emphasised that the optimum dosage of steroids is not equivocal.

The intravenous therapy is followed by oral administration of prednisolone (on a sliding scale).

Steroids in very large doses (measured in grams of initial doses of hydrocortisone) are suggested by some to avoid steroid resistance(9). The administration of very large doses of I V methylprednisolone have been reported to be without side effects.(10).

Adrenaline:

This agent is not included in our programme because of the effectiveness of BB-adrenergic stimulants.

Sedation:

Sedation may be related to the failure of effective treatment and is not routinely administered.

Antibiotics:

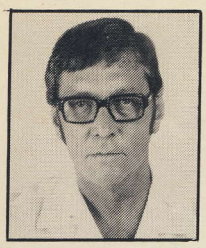
Prophylactic antibiotic therapy is not indicated.

This regime is directed towards the emergency care of the acute asthmatic patient, and does not cover other forms of therapy for less urgent cases.

SURVEILLANCE IN THE EMERGENCY ROOM:

Our patients remain in the emergency room for approximately four to six hours except in the event of a life threatening attack in which case admission to the pulmonary intensive care is arranged, after initial primary critical care has been administered.

An assessment of the degree of improvement is made on clinical grounds, repeat bloodgas analysis and measurement of peak airflow.



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On completion of treatment the peak airflow is measured. Patients with a 50% flow or less will be admitted and those with a reading of 50-60% will be observed in the emergency room for an extended period.

IDENTIFICATION OF THE HIGH RISK ASTHMATIC PATIENT:

A patients examined in our emergency room, presenting with any one or more of the undermentioned, will be admitted:

1. A history suggestive of a previous life threatening acute asthma attack.
2. Central cyanosis
3. Pulsus paradoxus in excess of 10mm Hg
4. Diminution of consciousness
5. Exhaustion
6. Pneumothorax or pneumomediastinum
7. Gross hyperinflation on chest röntgenogram
8. PO_2 less than 60mm Hg
9. Significant elevation of PCO_2
10. Electro cardiographic changes suggestive of pulmonary hypertension in the presence of an acute attack
11. All patients presenting in a first attack and all young patients.

CONCLUSION:

Patients with an acute asthmatic attack should be carefully evaluated before and after emergency room therapy. This will ensure the admission of all high risk asthmatic patients after primary critical care measures had been taken. This will obviate the discharge of patients who have not yet adequately recovered.

The management for acute asthma is based on adequate fluid therapy, administration of bronchodilators and corticosteroids therapy.

References:

1. Case, J.F., et al. Lung mechanics during provocation of asthma. Clinical Science 40 381-391 (1971).
2. Rees, H.A., Millar, J.S. and Donald, K.W. a Study of the clinical course and arterial bloodgas tensions of patients in status asthmaticus: Quarterly Journal of Medicine 37: 541-561 (1968).
3. Slutsky, A.S. and Rebuck A.S. Heartrate response to isocapnic hypoxia in conscious man: American Journal Physiology (1978).
4. 6, 7 Rebuck, A.S. and Read, J. Assessment and management of severe Asthma: The American Journal of Medicine. Vol. 51, Dec. 1971.
5. Rebuck, A.S. and Pengally, L.D. Development of pulsus paradoxus in the presence of airway obstruction. New England Journal of Medicine, 288: 66-9 (1973).
8. Rebuck, A.S. Radiological aspects of severe asthma. Australian Radiology 14: 264-268 (1970).
9. Schwartz, H.J. Lowell, F.C., Melby, J.C. Ann. Intern. Med. 1968, 69, 493.
10. Novak, E. et al. Clinical Pharmacol. Therap. 1970, 11, 711.
11. Clark, T.J.H. and Godfrey., Asthma Chap. 16. 1970 Publishers W.B. Saunders Company Philadelphi, Toronto.