STATUS ASTHMATICUS IN A MEDICAL INTENSIVE CARE

T K Lim

ABSTRACT

This paper describes 19 consecutive episodes (18 patients) of status asthmaticus managed in a medical intensive care. Eleven patients required mechanical ventilatory support [MV] and 2 (11%) died. Most patients had acute respiratory acidosis although arterial blood gases alone could not predict the need for MV. The peak inspiratory airway pressure measured during MV was a useful index of the severity of underlying lung disease. The most serious complication of MV was barotrauma. On follow up studies, two distinctive patterns of chronic asthma could be identified. One group of patients had highly labile pulmonary function with unpredictable and wide changes in peak flow rates while another group had poorly reversible obstruction.

Key Words: Acute bronchial asthma, respiratory failure, mechanical ventilation

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INTRODUCTION

Status asthmaticus (SA) may be defined as severe life-threatening airflow obstruction which does not respond promptly to bronchodilator treatment (1). This failure to respond initially to therapy with bronchodilator drugs identifies a population of asthmatic patients with severe clinical illness who are at risk of experiencing ventilatory failure and death. The management of SA involves multiple modalities which may include endotracheal intubation and mechanical support of ventilation (MV). These acutely ill patients are usually managed in an intensive care setting with close monitoring of their clinical state and ready availability of facilities for MV should the need arise. The use of tracheal intubation and MV may predispose patients to further complications (2).

This paper describes the management and clinical outcome of a group of patients with SA in a medical intensive care (MIC). Review of clinical experience in this sub-group of asthmatics with the most severe illness may help to identify high risk patients and improve on their management.

PATIENTS AND METHODS

Eighteen consecutive patients with bronchial asthma (19 admissions) were managed in the MIC of the National University Hospital over a 3 year period. All patients had severe acute asthma which did not respond to bronchodilator therapy with inhaled salbutamol, ipratropium bromide and intravenous theophylline and hydrocortisone. Patients with chronic obstructive lung disease and who had fibrotic or malignant lung disease were excluded from this analysis. There were 10 men and 8 women with a mean (SEM) age of 56 (4)yrs. They had bronchial asthma for a mean (SEM) duration of 11 (3)yrs. The pH and pCO_2 values were measured from arterial blood. In patients who required MV, the pH and pCO_2 was obtained immediately before tracheal intubation, and for the patients who did not require MV, the highest pCO_2 and concurrent pH were charted in Table 1. Mechanical support of ventilation was instituted when either the patients appeared fatigued in the presence of severe airways obstruction which was unresponsive to intensive pharmacotherapy or when respiratory arrest occurred requiring emergency tracheal intubation. A volume-cycle ventilator was used in all cases (Ohmeda CPU 1). During MV the peak end-inspiratory airway pressure [Paw(peak), cmH_2O] was recorded and charted hourly. This measurement was used as an index of the respiratory system impedance to tidal ventilation.

The case records were reviewed for complications which occurred during treatment in the MIC and for clues in the clinical history or subsequent follow up which might help to identify a predisposition to SA.

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RESULTS

Table 1 shows the clinical data for 19 consecutive admissions to the MIC for SA. The first 8 admissions were managed without MV. Only the most severely ill patients were admitted to the MIC. This was reflected in the development of ventilatory failure (acute CO_2 retention > 45 mmHg) in all but one and in the large proportion of patients who eventually needed MV [11/18 (61%)]. One patient (Table 1, No. 8) was admitted twice to the MIC but needed MV only once. Two patients died in the MIC (No. 17 & 18), the other 9 patients required MV for an average of 2.5 days before they were successfully extubated.

Figure 1 plots the values of pH (semi-log scale) against the pCO_2 (mmHg) from Table 1. Note the close correlation between pH and pCO_2 which was within the zone of "acute respiratory acidosis". The dotted lines delineate the zone of acute respiratory acidosis on the acid-base map (3). There was no significant difference in either pH or pCO_2 between "MV" and "no MV" groups.

Figure 2 compares the mean Paw(peak) measurements during acute illness within 24 hours of tracheal intubation with that measured just before extubation. Note the marked decrease in peak airway pressures following clinical resolution of SA before successful extubation.

TABLE 1
CLINICAL DATA FROM 19 [18 PATIENTS] ADMISSIONS TO THE M.I.C. FOR ASTHMA

Mechanical Ventilation [MV] was not needed in the first 8 admissions

No.	Sex	Age [y]	Years with asthma	pH	PCO_2 mmHg.	Days on MV	P_{aw}(peak-cmH_2O) Acute – Remit
1.	M	35	15	7.29	55		
2.	M	43	2	7.29	70		
3.	M	46	20	7.29	55		
4.	M	47	10	7.36	43		
5.	M	36	15	7.03	104		
6.	F	72	15	7.17	78		
7.	M	50	10	7.11	96		
8. [a]	M	65	3	7.10	73		
mean		49	11	7.22	68		
(SE)		(5)	(2.5)	(0.05)	(8)		
8. [b]	M	65	3	7.16	62	2	54 – 10
9.	M	72	30	7.15	72	3	30 – 10
10.	F	51	20	7.10	80	4	80 – 8
11.	F	64	25	7.29	72	3	32 – 18
12.	F	51	4	7.22	68	3	44 – 8
13.	F	73	10	–	–	3	50 – 14
14.	M	33	1	7.09	117	1	47 – 10
15.	F	33	2	7.34	50	1	7 – 12
16.	F	34	10	–	–	1	22 – 8
17.	M	62	2	–	–	–	45 – 8
18.	F	52	3	–	–	–	40 – 16
mean		56	11	7.17	79	2.5	44 11
(SE)		(4)	(3)	(0.03)	(8)	(0.4)	(5) (1)

P_{aw}(peak-cmH_2O): peak airway pressure during MV.
No. 8 [a] & [b] – admitted twice but needed MV only once [b].
No. 17 – died from cerebral anoxia
No. 18 – cardiorespiratory arrest in the casualty, died in the MIC.

Semilog Plot of pH vs PCO2 in Arterial Blood

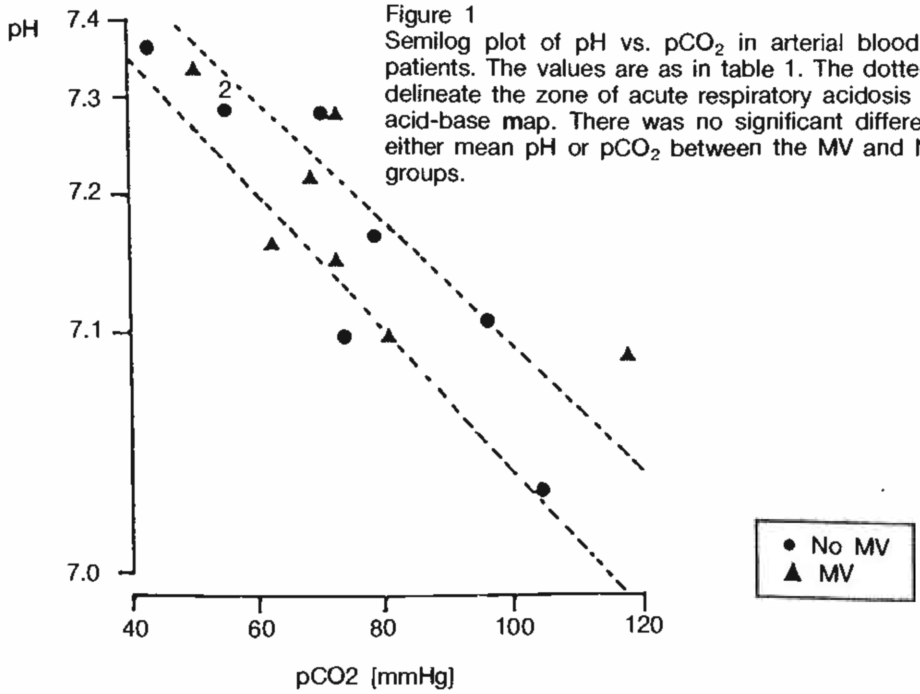
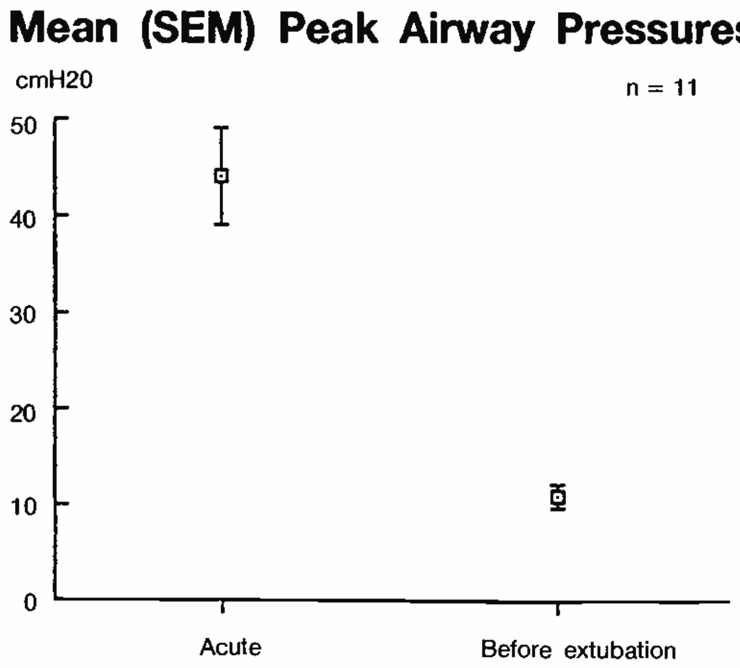


Figure 1
Semilog plot of pH vs. pCO_2 in arterial blood in 15 patients. The values are as in table 1. The dotted lines delineate the zone of acute respiratory acidosis on the acid-base map. There was no significant difference in either mean pH or pCO_2 between the MV and No MV groups.

Figure 2
This figure compares the mean(SEM) peak end-inspiratory airway pressures (cmH_2O) in 11 patients measured during acute illness within 24 hours of tracheal intubation and during clinical remission just before extubation.



Complications were encountered only by the patients who needed MV. The major complications included rebound metabolic alkalosis (3 cases), pneumonia, premature extubation, hypotension with coronary insufficiency and barotrauma (1 case each). The barotrauma in one patient followed resuscitation in the casualty department and resulted in extensive subcutaneous and mediastinal emphysema which contributed to her death. All the other complications were effectively managed and did not result in any serious morbidity.

On chart review and follow-up studies of the 16 patients who survived, two patterns of bronchial asthma could be identified – chronic persistent or "irreversible" asthma with baseline airflow obstruction and labile or"brittle" asthma with wide swings in lung function over short time intervals (4). Examples of these two patterns during in-patient charting of peak expiratory flow rates are shown in figure 3.

DISCUSSION

While SA occurs only in a small group of patients with bronchial asthma, it is a life-threatening illness which deserves careful examination. The recent reports of an increase in both the prevalence and mortality rates of bronchial asthma further highlight the importance of this condition (5, 6).

The mainstay of treatment for SA is intensive pharmacotherapy with bronchodilator and glucocorticoid drugs (7). Mechanical ventilation should be avoided as far as possible unless respiratory arrest or rapid deterioration in consciousness occurs. The decision to intubate and mechanically support ventilation in SA is a difficult one. In this series of patients arterial blood gases alone did not identify those patients who needed MV. While young men may retain CO_2 acutely, they tolerate it better and may recover rapidly with intensive therapy while the older women may require mechanical support more frequently. This observation is similar to that reported by Mountain et al. and reminds us that arterial gas results (pH and pCO_2) are poorly related to the severity of airways obstruction (8,9). The mortality rate of 11% (2/18) is comparable to that reported in other studies (10, 11, 12). Both patients died from hypoxic brain damage following severe acute airways obstruction and respiratory arrest.

The high end-inspiratory airway pressures [Paw-(peak)] measured during the "acute" period (Table 1 and figure 2) were due to a combination of active expiratory effort by the patient such as during attempted coughing and increased inspiratory resistance from airways obstruction. High Paw(peak) and low lung compliance are major risk factors which predispose patients to ventilator associated barotrauma (13). Pulmonary barotrauma was seen in only one patient in this series but the widespread distribution of extra-alveolar air under pressure contributed to her death. The high Paw may be reduced by adjusting the ventilator to deliver a lower inspiratory flow rate and tidal volume at the expense of some carbon dioxide retention (10). Muscle relaxant drugs might have to be used in order to reduce airway pressures and improve patient-machine coordination in the immediate post-intubation period. This will reduce the risk of barotrauma and may allow the respiratory muscles a period of rest. A neuromuscular relaxant drug was needed in all patients who received mechanical ventilatory support in this series. While the dangers of circulatory depression and histamine release are well described, we encountered no major side effects with pancuronium which was the most commonly used muscle relaxant and should be administered together with a sedative drug such as diazepam (14).

As shown in figure 2 the Paw may be used as an index of the severity of underlying airway disease. The end inspiratory peak airway pressure is the sum of the

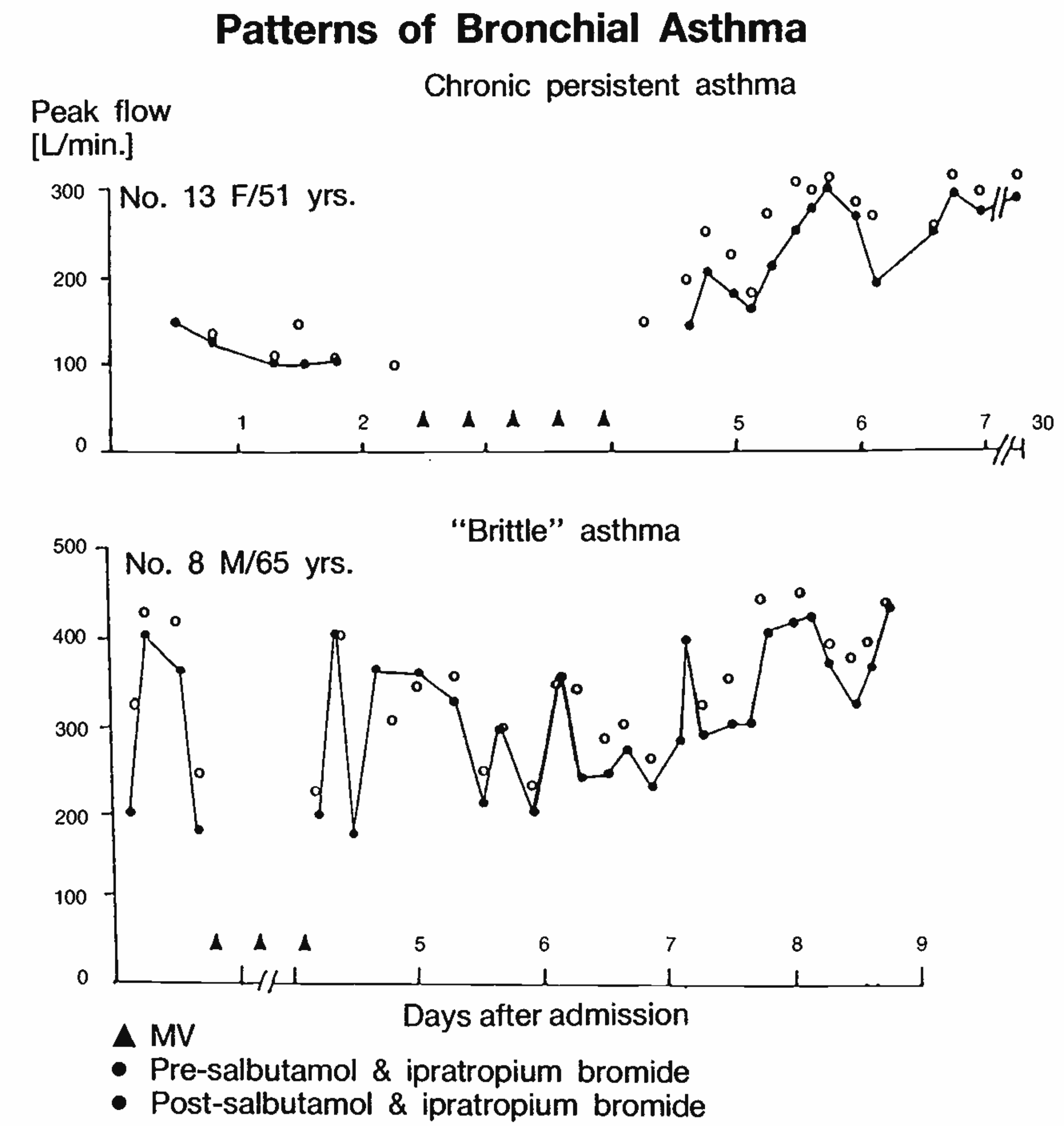


Figure 3
These two figures chart the peak expiratory flow rates (L/min) before (•) and after (o) inhalation of bronchodilator drugs. They show the different patterns of bronchial asthma in these two patients with SA.

pressures dissipated against frictional and elastic forces in the lung and chest wall. It also incorporates the intrinsic-PEEP associated with hyper-inflation (15). While various sophisticated measurements of lung function may be derived from analysis of the airway pressure tracing during volume-cycled ventilation at constant inspiratory flow, nevertheless, we have found the Paw a crude but useful index of overall respiratory system impedance in guiding therapeutic decisions and during the weaning process (16).

Turner-Warwick in a classic paper described the different patterns of bronchial asthma which may be identified by long-term charting of lung function results (4). Subsequent reports have confirmed the suggestion that some of these patterns may identify patients which are at high risk of dying suddenly form bronchial asthma (12). Among the patients described in this report two distinctive patterns could be identified on review of their peak flow charts as shown in figure 3. The individual patterns tended to persist during out-patient and home recording of the flow rates. The highly "labile" pattern with wide and unpredictable variation in airways obstruction was more common among younger patients. All the three young patients, aged 33-34 years, who needed MV had normal spirometry measured within 24 hours of severe airways obstruction which required emergency intubation and mechanical ventilatory support. This may be a variation of the well known and more common "morning dipping" pattern where predictable falls in lung function occur in the early hours of the morning. The "irreversible" pattern of obstruction which improves very little with either bronchodilator drugs or corticosteroids was observed in older patients and especially among women. These subjects fail to perceive any discomfort despite severe underlying airways obstruction and when at home, tend to self administer bronchodilator drugs much less than prescribed. They only seek help in hospital during severe life-threatening airways obstruction. Subjects with "labile" asthma may have highly reactive airway smooth muscle which undergo spasm with little of no provocation whereas the "irreversible" group may have predominantly airway inflammatory changes which result in chronic airway obstruction. We suggest that patients who exhibit either of these two pattems on charting of their pulmonary function should be carefully monitored and should receive prompt, intensive pharmacotherapy even during apparently mild exacerbations.

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