modestly elevated in both patients, yet the arteriovenous differences in oxygen content implied that the cardiac output was normal in one case and considerably reduced in the other. Many more such studies will be needed to clarify the problem of oxygen transport in the asthmatic attack.

Cerebral oxygenation may be particularly vulnerable in the asthmatic, exposed as he is to episodes of relatively sudden hypoxia, often combined with hypocapnia, which is known to decrease cerebral blood flow. From measurements made during exposure of normal men to high altitude (Severinghaus *et al.* 1966), where arterial P_{O_2} and P_{CO_2} values similar to those in the asthmatic attack may be encountered (P_{O_2} 44 mmHg, P_{CO_2} 35 mmHg), the hypoxia induced cerebral vasodilatation, thereby protecting cerebral oxygenation to some extent. However, the mean P_{O_2} of the jugular venous blood in these studies was 27 mmHg, as compared to the normal value of 32 mmHg, but as consciousness is lost when this falls below 20 mmHg, the margin of safety is not great.

Hypoxia is undoubtedly common in the severe attack of asthma, and its relief by oxygen therapy appears entirely rational. If bronchodilators are employed hypoxæmia may be potentiated and again concomitant oxygen appears desirable. Carbon dioxide retention may be aggravated by uncontrolled oxygen, at least in children (Simpson *et al.* 1968), and although high concentrations of oxygen are probably safe for most asthmatics, the recognition of potentially lethal CO_2 retention necessitates arterial puncture and blood gas analysis. In the asthmatic, as opposed to the bronchitic, CO_2 retention is most often acute. It is widely accepted that intermittent positive pressure ventilation is then indicated.

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The Intensive Therapy of Asthma

Death in an attack of asthma is due to acute respiratory failure, sometimes associated with the misuse of isoprenaline given as an aerosol (Fraser *et al.* 1971). In 1962 we set out to prevent adult patients dying from severe asthma. This was achieved in 1969 and was made possible by using the services of a general intensive therapy unit (ITU) (Jones 1967). Most of the patients were admitted from the medical beds of the hospital and a few were referred from hospitals outside our district. Patients are transferred to the intensive therapy unit when an attack of asthma is judged to be severe. An attack is classified according to the disability of the patient (Table 1); all patients with severe asthma (Grade III) and those with moderate asthma (Grade II) of more than eight hours' duration are treated in the intensive therapy unit rather than in an intermediate stay ward.

The care of the adult asthmatic in a general ITU can be grouped under three headings:

(1) Intensive nursing care: There is a high nurse-to-patient ratio of one-to-one, and the nurses are both competent and confident. The patient can therefore be efficiently nursed day and night and feels 'safe'.

(2) Intensive observation: Most of the clinical observations required to assess the severity of the asthma and the progress of the attack are made by experienced nurses who have been specifically trained in the care of the seriously ill. The asthmatic is therefore under continuous observation by nurses who can detect signs of deterioration at an early stage and report these signs directly to the physician. Repeated assessments are also made by readily available doctors, who are experienced in the treatment of severe asthma. The important clinical observations are: the

Table 1
Method for grading the severity of asthma (Dr P M Gett)

Grade I: Patient able to carry out housework or job with difficulty (IA), or great difficulty (IB)
Grade II: Patient confined to a chair or bed but able to get up with moderate difficulty (IIA), or with great difficulty (IIB)
Grade III: Patient confined to a chair or bed
Grade IV: Moribund

Table 2
The Stage II therapy of asthma

Time	*Procedure*	*Anæsthetic drugs*
Few minutes	Preoxygenation, induction of anæsthesia, manual IPPV with oxygen	Methohexitone, suxamethonium
One hour	Inhalation anæsthesia and bronchial lavage with 1% sodium bicarbonate, manual IPPV	Ether
20–40 hours	Mechanical IPPV (Cape-Waine ventilator) controlled by muscle relaxant or sedative	Pancuronium, phenoperidine
8–24 hours	Assisted mechanical I PPV (Bird ventilator)	None

Table 3
Results of Stage II therapy of asthma

Date	*No. of cases*	*Lived*	*Reference*
1962–6	22	18	Riding & Ambiavagar (1967) Ambiavagar & Jones (1968)
1967	18	16	Williams (1968)
1968	7	6●	
1969	1	1	
1970	1	1	

●One patient died due to cardiac arrest during transfer

mental state, the heart rate, the degree of fatigue or exhaustion and the ability to raise sputum. The pH, $Paco_2$ and Pao_2 can readily be measured day or night in the laboratory of the ITU.

(3) Intensive therapy of severe asthma can be divided into two stages; when the first fails then the second is invariably indicated and is usually successful.

Stage I therapy consists of steroids, oxygen therapy, intravenous aminophylline, rehydration and intermittent positive pressure breathing (IPPB). Our practice is to give prednisolone (15 mg) or intravenous hydrocortisone (100 mg) six-hourly. The choice is made after deciding whether fluid therapy is to be by mouth or vein. This decision is made by seeing whether breathlessness prevents the patient from drinking a glass of water. Oxygen in concentrations of 28–35% is given by means of a Ventimask or by the Edinburgh mask. The oxygen therapy is interrupted only to enable the patient to cough, drink, swallow tablets or use IPPB. Experience, without supporting scientific evidence, has convinced me that IPPB definitely helps to stop an attack by enabling the patient to raise the tenacious sputum. We use the Bird ventilator fitted with a mouthpiece. Patience and firm handling are required to start the treatment, which is given for five to ten minutes each hour. Stage I therapy rarely fails to stop a severe attack. Failure is nearly always due to poor quality of the treatment, for example, inadequate steroid, too little or excess oxygen or sedation. However, experience has shown that the best treatment fails when given too late.

Stage II therapy consists of intermittent positive pressure ventilation (IPPV), bronchial lavage, intravenous hydrocortisone and fluid therapy. The indications for Stage II treatment are: cardiac arrest, coma and when the experienced physician feels that Stage I treatment is failing. The danger signs which indicate the probable need for Stage II therapy are: confusion, increasing breathlessness, increasing heart rate, pulsus paradoxus, ineffective cough, exhaustion, hypercapnia or increasing hypoxæmia. The methods of Stage II treatment are given in papers from this unit (Riding & Ambiavagar 1967, Ambiavagar & Jones 1967, Williams & Crooke 1968). Our current therapy is summarized in Table 2. Artificial respiration quickly oxygenates the blood and relieves mechanical stresses on the circulation (Ambiavagar *et al.* 1967); total alveolar ventilation improves slowly. The method of bronchial lavage is safe and simple, causes no morbidity and invariably produces a harvest of mucous plugs (aptly termed 'little worms' by Dr C M Fletcher). The bronchoscope is unnecessary and potentially dangerous in this condition. The method of bronchial lavage of Ramarez-R *et al.* (1965) was devised to obtain alveolar material in cases of mucoviscidosis and is not advised in the treatment of asthma.

The direct results of Stage II treatment are summarized in Table 3. The indirect results of our experiences in the resuscitation of the moribund asthmatic led us to improve the standards of care of all asthmatic patients. Education of the patient, detailed supervision at a clinic, good treatment of an attack and early admission to the hospital have reduced the incidence of severe asthma and the need for Stage II treatment.

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